BRITISH MEDICAL JOURNAL

British Medical Journal

Volume 3, No.1, January 2023

Internet address: http://ejournals.id/index.php/bmj
E-mail: info@ejournals.id
Published by British Medical Journal
Issued Bimonthly
3 knoll drive. London. N14 5LU United Kingdom
+44 7542 987055

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PROGNOSTIC COURSE OF DISEASE IN PATIENTS WITH DILATED CARDIOMYOPATHY

Abdurakhmanova Dilnoza Furkatovna
Ibragimov Nodir Shobotirovich
Tashkent pediatric medical institute
Heard of department of Pediatrics 1,
Pediatric cardiac surgery

Abstract: Dilated cardiomyopathy (DCMP) is a severe disease characterized by the development of cardiomegaly as a result of a decrease in myocardial contractile function, a primary internal defect of damaged cardiomyocytes, as a result of a strong expansion of the heart chambers, especially the left ventricle. The disease is characterized by a severe course, often leading to disability and a high risk of death . In many children, it is difficult to determine the onset of the disease, because it passes for a long time, almost without symptoms. In a number of patients, increased weakness, weight gain and/or physical retardation, fainting, syncopal state, tendency to recurrent pneumonia are noted. Sometimes, the only sign of the disease is the disturbance of intraventricular and atrioventricular conduction, changes in extrasystolic appearance on the ECG. Their occurrence is much lower in children than in adults. About 10 percent of people with dilated cardiomyopathy are people over age 65. In the United States, the condition is 3 times more common in men than women and 3 times more common in African Americans than whites. In one year, 5-8 people out of 100,000 people are affected (10). Despite significant clinical changes in the course of the disease, its prognosis in children is usually not good .

Keywords: dilated cardiomyopathy, hypertrophic cardiomyopathy, heart, open arterial passage, prognosis.

Dilated cardiomyopathy (DCMP) is the most common clinical form of cardiomyopathy in children [1]. According to information in the literature, dilated cardiomyopathy (DCMP) (in 60% of cases) and hypertrophic cardiomyopathy (GCMP) (in 40%) cases are observed to be widespread among the population (2.3.). DCMP is characterized by an expansion of the left ventricular barrier, a decrease in myocardial contractility under hemodynamic load, and a violation of the systolic function of the left ventricle. In this form of cardiomyopathy, the indicators of disability and death prevail in children, and it is considered the main cause of chronic heart failure [4.5.]. The prevalence of DCMP varies from 3.8 per 100,000 in Europe to 40 per 100,000 in the United States [5]. The disease is more common in boys than in girls. The prognosis of the disease is very serious, and its onset depends on the age of the patient and the severity of heart failure. According to the results of observation of children aged 5 months to 15 years for 10 years, a positive result was observed in 14% of cases, clinical stabilization in early age children - 21.1%, lethal result was observed in 64.8% of cases [6]. However, in recent years, due to the widespread introduction of heart transplantation into practice, there is an increasing trend in the survival rate of children [7]. Patient Abdukaimov A. He was born on January 8, 2010 in the Mirabad district of Tashkent city (12 years old at the time). 3rd pregnancy 2nd child. The first fetus died at birth. The pregnancy passed with the risk of miscarriage up to the 20th week. Late on the background of anemia. Had a quick cold. The fetus was born asphyxiated at 38 weeks in a bruised condition. Body weight 2980 g, height 52 cm, Apgar score - 7/7. He was discharged home from the maternity hospital on the 5th day. In the 3 months of the patient's life, a heart murmur

was detected by the resident pediatrician during a medical examination. The patient was referred to a pediatric cardiologist, an echocardiography (ExoCG) was recommended. An ultrasound examination revealed a septal defect, moderate dilatation of the left ventricular surface, and sent him to a specialized center for cardiac surgery.

By the end of the fourth month of life, the patient began to show signs of natural mother's milk, wheezing during breast feeding and rapid fatigue, profuse sweating. Transferred to artificial feeding. The patient was under the supervision of a pediatrician and a cardiologist until the age of 2.5 years. Autoplasty operation was performed due to congenital heart defect (CHD) due to septal septal defect (SSD). It is recommended to be under the supervision of a cardiologist and a pediatrician. The patient was not subjected to heavy physical work, and was not scheduled to see a cardiologist. By 2017, the child went to see a cardiologist due to increased symptoms of wheezing and shortness of breath, as well as a couple of short-term fainting spells, and Holter monitoring was performed during the day. Extrasystole was observed. Picture 1.

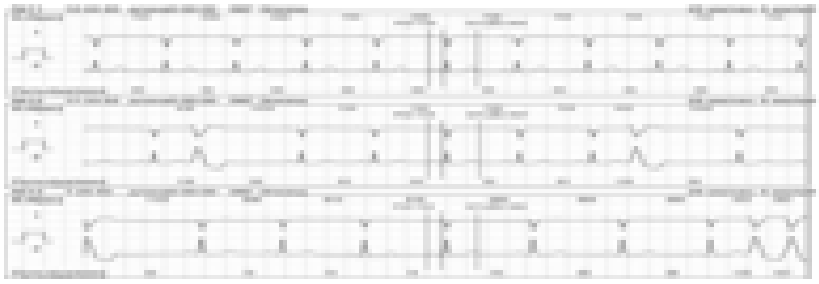
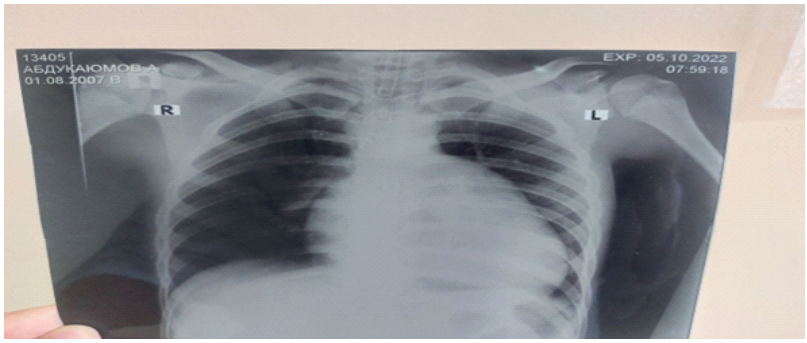


Figure 1. Holter monitoring: Extrasystole.

The patient was diagnosed with Cardiomyopathy, Intercompartmental barrier defect. Because of the general condition of moderate severity, conservative treatment was recommended. The following checks were made. General blood analysis: Hb-149 g/l, Ht- 41, RK- 0.8, leukocyte- 13, neutrophil count - 50%, eosinophil - 2%, monocyte - 6%, EChT - 9 mm.cm.ust. Hepatitis markers "V" and "C" are negative. RW is negative. Biochemical blood analysis: Total protein - 66.4 g/l, glucose - 8, urea - 4, creatinine - 59.6, SRO (-). ECG analysis: Sinus rhythm. The number of heart contractions (HC) is 74 beats/min. The electrical axis of the heart is shifted to the left. PQ interval extended to 0.24. Incorrect blockade of the anterior horn of the bundle of Hiss of the left leg. Left ventricular extrasystole. ExoKG examination revealed some profound organic changes in the heart: end-diastolic dimension (DD) - 6.1 cm, end-systolic dimension (SD) - 4.9 cm, left ventricle (LV) - 4.1 cm, ventricular septal defect (SD). - 2.4 cm, ejection fraction (EF) - 50%, foci of hypokinesia, mitral regurgitation 2nd degree. The above examination of the patient

after the results, he was assigned to be under constant cardiologist supervision and to receive conservative treatment procedures regularly on time. According to his mother, he did not have the opportunity to receive medical treatment in full order. Cases of loss of consciousness were observed several times. On 27.09.2022, the body temperature rose to 38.5-39.0 C and was treated as an outpatient. The fever subsided, but on the second day of the illness, the patient's symptoms of shortness of breath and lack of air increased and he lost his appetite. He was brought to the city clinical children's hospital No. 1 by ambulance and admitted to the intensive care unit. His general condition on arrival is serious. Severe acute circulatory failure due to cerebral insufficiency. The clinical signs typical of the decompensation stage of dilated cardiomyopathy were observed three times, and suddenly due to the dilatation of the left ventricle, blood did not reach the

brain from the aorta due to the ventricle contracting enough and not being able to supply blood to the aorta. Because of this, cases of euphoria have been observed. At this time, the patient had asystole, apnea, dilated pupils, freezing of hands and feet, hypothermia. Immediate resuscitation procedures were carried out with hyperventilation, intubation and IVF twice. After some time, Khushi regained consciousness and was transferred to independent breathing. Normal body temperature. His attitude is passive, he is not able to adequately answer questions by himself. The skin is dry, the subcutaneous fat layer is poorly developed. The skin is pale, marble-like. The bases of the eyes are dark, the triangle of the lips and nose is blue.Peripheral lymph nodes are not enlarged, the submandibular lymph node is slightly enlarged. Breathing through the nose is independent. On auscultation, there are dry rales in the lungs with a background of rough breathing.All borders of the percussive heart are widened, the impulse of the heart tip is shifted to the left-down. On auscultation, heart sounds are muffled, systolic noise is heard at all points. At the peak, a "quail rhythm" is heard in III-IV -tones, a double tone over the pulmonary artery. The tongue is covered with a wet white coating. Swollen abdomen is painless on palpation. The liver protruded from the right rib cage 3.5 x 3.0 x3.0 cm.The spleen is not enlarged.Diarrheais prone to constipation.Urinary excretion is reduced compared to the fluid intake. The following laboratory instrumental tests were conducted. General blood analysis: Hb-156 g/l, erythrocyte - 5.39, Ht- 42.2%, thrombocyte - 240, RK- 0.8, leukocyte- 12.5, neutrophil t/ya - 0, s/ya - 69.7, lymphocyte - 24, eosinophil - 2%, monocyte - 6%, EChT - 9 mm.cm.ust. RW is negative. Biochemical blood analysis: Total protein - 62.3 g/l, albumin - 41.3, glucose - 13.5, ALT-26, urea - 4.1, creatinine - 63.8, SRO (-). X-ray: Increased lung imaging. Hypovalemia in the lungs. The roots are asymmetrical. The sinuses are free. The borders of the heart are enlarged to the right and left. The heart is flattened. Cardio thoracic index (CTI) is 74%. Cardiomyopathy. Picture 2.



Picture 2.

In ultrasound examination of internal organs (UE): diffuse changes of the pancreas, hepatomegaly, conglomerate salts in the kidney.

ECG analysis: Sinus rhythm. HIGH - 75 units/min. The electrical axis of the heart - shifted to the left. The PQ interval has widened to 0.30. Violation of repolarization processes in the myocardium of the left ventricular wall. Left ventricular hypertrophy. Incorrect blockade of the anterior horn of the bundle of Hiss of the left leg. Picture 3.

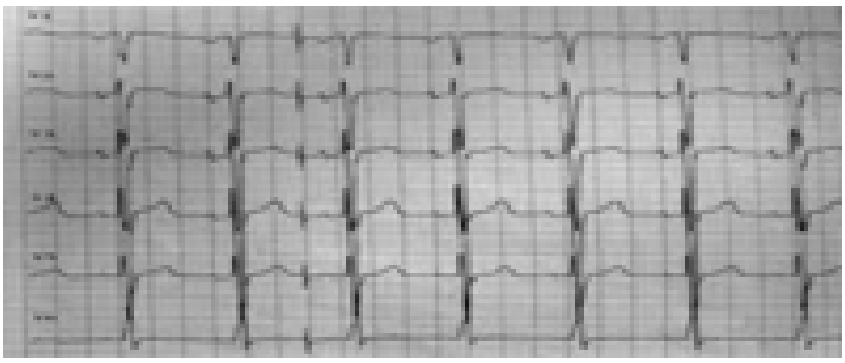


Figure 3. Electrocardiography.

ExoCG examination analysis: mitral valve regurgitation grade 3, tricuspid valve regurgitation grade 2. End-diastolic size (DS) - 6.8 cm, end-systolic size (SS) - 6.0 cm, left ventricle (LV) - 3.9 cm, ventricular septal barrier (SB) - 2.6 cm, ejection fraction (EF) - 40%, myocardial diffuse hypokinesia. Figure 4.

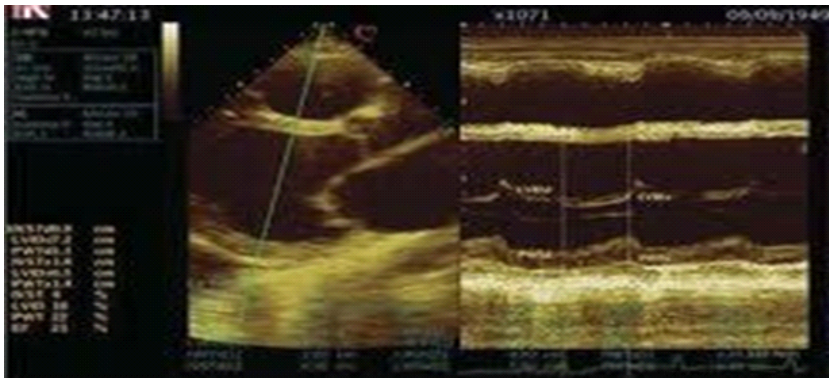


Figure 4. Echocardiography.

A number of narrow specialist reviews were conducted. Neuropathologist: Symptomatic epilepsy. ENT: chronic rhinosinusitis.

Based on the above clinical, anamnestic, instrumental and narrow specialist conclusions and examinations, the diagnosis was made: Main: Dilated cardiomyopathy decompensation stage. Xamrox: The condition after autoplasty of secondary compartment septal defect. Complication: Chronic heart failure stage II A. Functional class III according to Nun.

In order to alleviate the patient's heart failure, fluid and salt control, APF inhibitors, as well as beta-blockers, and diuretics were recommended while controlling daily fluid intake and excretion. Anticoagulant and antiarrhythmic drugs were recommended to be used under laboratory control during cardiological monitoring. It was emphasized to the patient's relatives that heart transplantation was the last indication.

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