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SIGNIFICANCE OF CHANGES IN MYOCARDIAL ENZYMES IN CHILDREN WITH CONGENITAL HEART DEFECTS

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ABSTRACT

Introduction. In young children, CHD can occur with disruption of the energy supply of the myocardium and tissue homeostasis. To assess its condition, biochemical indicators are used that reflect various links of the pathological process, both in the whole body and in the myocardium.

Purpose of the study: This paper presents diagnostically significant indicators of myocardial damage in young children with congenital heart defects.

Material and methods. 150 children with congenital heart disease aged 2 months to 3 years were hospitalized in the pediatric cardio-rheumatology department of the State Institution "National Medical Center" (Tajikistan). They were under observation for the period from 2008 to 2018, and 30 children comprised comparison groups. The structure of defects was represented by: defects of the interventricular (VSD) (50) and interatrial septal (ASD) (50), tetralogy of Fallot (TF) (50). All children underwent clinical-instrumental, laboratory and biochemical research methods: ECG, echocardiography, radiography, troponin-T, MB fraction (MB-CPK), LDH, ALT, AST.

Results. Clinical manifestations of cardiac dysfunction in children depended on the type of defect, severity, presence or absence of heart failure. Elevated enzymes AST, LDH, MB-CK and troponins in children with congenital heart disease show the state of the cardiovascular system in congenital heart defects, being markers of myocardial dysfunction. The consequence of chronic heart failure is an increase in the level of AST in children with circulatory disorders of 2 A-B degrees. The degree of myocardial damage in children with CHD allows us to assess the activity of CPK-MB and troponin I in a comprehensive assessment of clinical and functional research methods

Conclusions. In children with CHD, there is an increase in the enzymes CF-CF, LDH and troponin I, which are the most significant in the diagnosis of markers of myocardial dysfunction that determine myocardial damage. The consequence of chronic heart failure is an increase in the level of AST in children with circulatory disorders of 2 A-B degrees

Keywords: congenital heart defects, young children, myocardial enzymes

RELEVANCE.

Diseases of the cardiovascular system occupy one of the leading places in the pathology of childhood, among which the most common are congenital heart defects (CHD).

More than half of the children with congenital heart disease, born with malformations incompatible with life, thanks to the achievements of modern cardiac surgery in recent years, can receive treatment. The use of new methods of diagnosis and radical correction of defects, timely therapy, improve the outcomes of operations, reduce the level of disability, infant and child mortality. D12.

The clinical assessment of the cardiovascular system in young children is determined by various factors related to the health of the child, the state of cardiac activity, the presence of concomitant pathology, etc. Their effects determine the nature of the course of the defect, both before and after surgery. The occurrence of concomitant pathology in congenital heart defects is associated with the peculiarities of the course of the antenatal period and the impact of perinatal factors. The severity of CHD also depends on the influence of concomitant diseases. In turn, the severe course of the defect can contribute to the development of intercurrent pathology.

In young children, CHD can occur with a violation of the energy supply of the myocardium and tissue homeostasis. To assess its condition, biochemical indicators are used that reflect various links of the pathological process, both in the whole body and in the myocardium. The leading value belongs to the levels of troponin - T (Tr-T), activity of MB - the fraction of creatine phosphokinase (MB-CPK), lactate dehydrogenase (LDH), aspartate aminotransferase (AcAt) in the blood serum. Changes in the levels of biochemical parameters may indicate energy disorders, myocardial damage, increased permeability of cell membranes of cardiomyocytes, the severity of the pathological process in various heart pathologies.

This paper presents diagnostically significant indicators of myocardial damage in young children with congenital heart defects.

METHODS AND MATERIALS

The study is based on the analysis of the results of a study of 150 children with CHD aged 2 months to 3 years, who were hospitalized in the children's cardio-rheumatology department of the State Institution "National Medical Center" of the Ministry of Health and Social Protection of the Republic of Tajikistan, for the period from 2008 to 2018, and 30 children - a comparison group (relatively healthy children, without CHD), of which 15 were boys and 15 were girls.

The structure of defects was represented by: defects of the interventricular (VSD) (50) and interatrial septal (ASD) (50), tetralogy of Fallot (TF) (50). All children were in a serious condition and had signs of NK 1-2 A-B degrees. The diagnosis of congenital heart disease was established postnatally, in the first months of life, based on a comprehensive clinical and instrumental study. The degree of NC was verified according to the Strazhesko-Vasilenko classification modified by N.A. Belokon. The research methods included: the study of anamnesis, clinical features of the course of congenital heart disease, data from instrumental research methods: ECG, echocardiography, radiography. To assess the biochemical state of cardiac activity, the level of troponin - T in the blood was determined using a test system, the activity of creatine phosphokinase of the MB fraction (MB-CPK) by enzyme immunoassay, the activity of LDH, AST.

RESULTS

We analyzed the clinical and biochemical features of the CHD course in the three examined groups. It was revealed that in the first group of examination (children with ASD), patients complain of shortness of breath during physical exertion (minor), fatigue (35). With an objective examination, their condition is more often regarded as relatively satisfactory; there is a normal color of the skin and visible mucous membranes. The leading signs were perioral cyanosis of a transient nature (42), moderate enlargement of the borders of the heart (38), mainly to the right side, systolic murmur with maximum localization in the III-IV intercostal space to the left of the sternum (48). A total of 15 children showed clinical signs of heart failure of the 1st degree. Certain features were revealed during the examination of the heart area: on palpation, the apex beat was found to the right of the sternum, mainly in the right half of the chest there are borders of relative and absolute cardiac dullness.

In the second group of examination (with VSD), clinical signs of heart failure of 1-2 B degree were revealed. 30 children were often disturbed by palpitations at rest. Shortness of breath occurred in 28 patients. The color of the skin was changed in 20 patients with circulatory disorders. In more than half of the patients, the main complaint was frequent acute respiratory infections (more than 4-5 times a year), which were characterized by a protracted course and frequent complications. Pneumonia was the most common (38). With small defects, there were no signs of circulatory disorders (NK). The children developed normally and did not require treatment; a scraping systolic murmur of moderate intensity was clinically heard in the 4th-5th intercostal space to the left of the sternum (20). Pulmonary hypertension did not develop. With moderate and large defects, symptoms more often manifested from 1.5-2 months.

life. Shortness of breath (46), fatigue during feeding (25), lag in weight and physical development (32), sweating (25) were noted. Most children have a history of recurrent pneumonia (30). On examination, the heart hump was pronounced, in the 4th intercostal space on the left (15) systolic trembling was noted (10), the 2nd tone was split and amplified in the area of the pulmonary artery (30), in some patients there was a mezadiastolic murmur in the apex area (15), associated with a relatively mitral stenosis due to large shunt, or diastolic murmur associated with relative pulmonary valve insufficiency (30). In 12 children with a large defect, there were signs of total heart failure: enlarged liver, spleen, shortness of breath, tachycardia, edema, congestive rales in the lungs.

An analysis of the case histories of patients in the 3rd group of children (with Fallot's tetrad) showed that the clinical signs, severity and course of congenital heart disease in the blue group differ significantly from those in the pale forms. On the basis of such clinical signs as shortness of breath, cyanosis, shortness of breath and cyanotic signs, noise, the diagnosis of Fallot's tetrad can be suspected already in the first weeks and months of life of children. In almost half of sick children, the diagnosis of Fallot's tetrad occurs already in the maternity hospital. Severe course with cyanosis in the neonatal period occurs in 1/3 of patients. Shortness of breath and cyanotic attacks usually occurred in children in the 2nd, 3rd years of life. Clinical signs of dyspnea-cyanotic attacks were manifested by severe weakness (50), deep rapid breathing (50), loss of consciousness (45).

The forced position of the child (the desire to take various facilitating positions) was found in all children with Fallot's tetrad and from an early age.

The physical development of children with Fallot's tetrad differed markedly from healthy children. Sick children have already been born with low body weight and height in 40 children. In the first year of life, malnutrition progressed rapidly, and by the end of 1 year of life, 45 children had malnutrition of varying degrees.

A typical manifestation of the blue forms of congenital heart disease were shortness of breath and cyanotic seizures observed in 46 out of 50 children, usually at an early age. Shortness of breath-cyanotic attacks from birth appeared very rarely. By the age of one month, they appeared already in 18 examined children. At the age of 6 months, almost all patients had the main clinical symptoms of Fallot's tetrad. And at the age of one year, the leading signs were in all children.

Thus, the clinical manifestations of cardiac dysfunction in children depended on the variant of the defect, the severity, the presence or absence of heart failure.

The analysis of the conducted biochemical studies showed that the indicators of the enzymes being determined in all study groups were increased compared to the control group. ALT levels were within normal limits. The level of AST in the main group was normal, but higher than ALT. CPK values were within the normal range, and LDH and CK-MB levels were increased in the main group.

Troponins T and I did not exceed normal values. But in the main group, the level of cardiac troponins was higher compared to the comparison group. Their numerical values were significantly higher (1.5-2 times) in the main group with VSD and Fallot's tetrad, in relation to the comparison group and to group 1 (with ASD). The increase in troponin I between groups was statistically significant.

Chart 1. Indicators of levels of enzymes and troponins in children with CHD

Indicator	Children with ASD	Children with VSD	Children with TF	Conditionally healthy.	Reliability
ALT, U/l (norm 5-40 U/l)	27 (22;35)	24 (17;32)	22 (16;30)	15 (12;23)	p<0,05
AST, U/l (norm 5-40)	34 (29;40)	42 (33;56)	44 (36;57)	17 (15;21)	p<0,005
LDH, U/l (norm 200-400)	512 (422;556)	545 (446;625)	556 (465;630)	250 (200-310)	p<0,005

CPK, U/l (norm 30-170)	92 (61;140)	110 (75;140)	115 (78;138)	85 (55;130)	p<0,1
KFK-MV, U/l (norm 1.8-24)	44 (32;65)	48 (35;78)	51 (38;80)	10,8 (0,9;16)	p<0,05
Troponin T, ng/ml Norm 0-0.1	0,13 (0;0,14)	0,15 (0;0,16)	3,15 (0,18;4,47)	0,10 (0;0,11)	p<0,05
Troponin I, ng/ml (normal 0-0.5)	0,02 (0,01;0,05)	0,03 (0,01;0,04)	0,04 (0,01;0,07)	0,01 (0-0,005)	p<0,05

As can be seen from the chart , in children with VSD and Fallot's tetralogy, the levels of troponin - T, CPK-MB were elevated. In children with VSD and TF, there was a statistically significant increase in LDH, CPK-MB and AST. From troponins, the highest values(on average 0.03 ng/ml) were found in children with VSD and TF, while in other groups they were practically unchanged. The levels of ALT and CPK practically did not change and were within the normal range. In children with CHD, AST and LDH levels were elevated. The level of LDH was increased by an average of 50 units.

In the main study group, there was an increase in LDH, CPK-MB and AST in the main group, more often in children with VSD and TF. Before the operation, the level of AST was elevated, which was associated with the presence of circulatory disorders.

Analysis of changes in troponin T in the study groups revealed the highest values in the group in children with VSD and TF (0,15 и 3,15). In the first group of children with ASD and in the comparison group, there were no significant changes in the troponin T index. The level of troponin I in children with VSD and TF (0,03 и 0,04) was much higher than in children with ASD and the control group.

An elevated level of troponin I was detected in children whose condition during hospitalization was regarded as severe.

The change in troponin I values is associated with a deeper lesion and changes in the trophism of the ventricular myocardium, which was confirmed by the data of instrumental research methods.

Elevated enzymes AST, LDH, MB-CK and troponins in children with congenital heart disease show the state of the cardiovascular system in congenital heart defects, being markers of myocardial dysfunction. The consequence of chronic heart failure is an increase in the level of AST in children with circulatory disorders of 2 A-B degrees. The degree of myocardial damage in children with congenital heart disease makes it possible to assess the activity of CPK-MB and troponin I in a comprehensive assessment of clinical and functional research methods.

CONCLUSIONS.

In children with CHD, there is an increase in the enzymes CF-CF, LDH and troponin I, which are the most significant in the diagnosis of markers of myocardial dysfunction that determine myocardial damage. The consequence of chronic heart failure is an increase in the level of AST in children with circulatory disorders of 2 A-B degrees

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