MYOCARDIODYSTROPHY IN CHILDREN AND ADOLESCENTS

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Annotatsiya: "Miokardiodistrofiya" (MKD) yoki "miokard distrofiyasi" tushunchasi miokarddagi metabolizmning biokimyoviy darajada buzilishini anglatadi, ular ularni keltirib chiqargan sababni bartaraf etishda qisman yoki to‘liq qaytariladi. Miokardning uzoq vaqt davom etib boradigan va zo‘rayib boradigan distrofiyasi uning qisqarish funksiyasi susayib, yurak yetishmovchiligi boshlanishiga olib keladi. O‘tkir MKD o‘tkir yurak yetishmovchiligiga olib kelishi mumkin. Har qanday etiologiyali MKD rivojlanishining asosida, odatda, miokardning o‘tkir yoki surunkali gipoksiyasi yotadi.

Abstract: The concept of "myocardiodystrophy" (MCD) or "myocardial dystrophy" refers to metabolic disorders in the myocardium at the biochemical level, which are partially or completely reversible when the cause that caused them is eliminated. Prolonged and progressive myocardial dystrophy leads to a decrease in its contractile function and the development of heart failure. Acute MCD can cause acute heart failure. The development of CVD of any etiology is usually based on acute or chronic myocardial hypoxia.

Kalit so’z: Miokardiodistrofiya, yurak, qon tomirlar, infarct, qon aylanishining buzulishi, endokard.

Keywords:| Myocardial dystrophy, heart, blood vessels, infarction, circulatory disorders, endocardium.

Article content : Causes of MCD development

The development of myocardial dystrophy can be caused by myocardial diseases (myocarditis, cardiomyopathy), extracardiac diseases (anemia, chronic tonsillitis, various poisonings, thyrotoxicosis, hypothyroidism, chronic somatic diseases), as well as physical overstrain in athletes (hyperfunctionogenic, contributing to the development of "pathological sports heart"). These causes lead to disorders of protein, energy, especially electrolyte metabolism in cardiomyocytes, as well as the accumulation of pathological metabolites, which can cause corresponding clinical symptoms: chest pain, various rhythm and conduction disorders, heart failure. Myocardiodystrophy is always a secondary process, including vegetative, dysmetabolic, enzymatic (congenital or acquired), electrolyte, and neurohumoral disorders. As early as the last century, G.F. Lang (1960s) proposed classifying

myocardiodystrophy according to the etiological principle. This approach is still maintained today. Myocardial dystrophy can be observed in children of any age, even in newborns. The causes can be previous intrauterine infections, perinatal encephalopathy, and "deadaptation" syndromes of the CNS and cardiovascular system, especially against the background of labor stress and hypoxia. In subsequent periods of childhood, frequent colds, anemia of various origins, chronic nasopharyngeal infection, blood diseases, endocrine pathology, past myocarditis, hypodynamia, physical and sports overloads, obesity, accumulation of various xenobiotics in the body, some medications (hormonal drugs, cytostatic immunosuppressants, some antibiotics, tranquilizers, etc.) can lead to the development of myocardiodystrophy.

Diagnosis of MCD

Complaints: pain in the heart area, shortness of breath during physical exertion, palpitations, weakness, a feeling of "breaks" in the heart. However, many patients may not have any complaints.

Anamnesis. Patients usually have diseases or pathological conditions in which there is always tissue chronic hypoxic syndrome (anemia, thyroid diseases, other endocrinopathies, chronic tonsillitis, significant physical overload and sports overload, past myocarditis, various poisonings, etc.), which contributes to the formation of myocardiodystrophy. Objective examination of the heart: irregular pulse, tachycardia, bradycardia, extrasystole, muffled heart sounds, weakening of the 1st heart sound at the apex, appearance of a systolic murmur. Electrocardiographic examination has special diagnostic value, but only in combination with medical history and clinical findings. On the ECG, various arrhythmias of varying nature are detected, which do not significantly affect systemic hemodynamics (moderate sinus tachycardia or sinus bradycardia, rare, often supraventricular extrasystoles), decreased voltage of the QRS complex, incomplete blocks of the bundle of Giss's bundles. Diagnostically significant ECG signs of myocardial dystrophy are impaired repolarization processes in the myocardium, which manifest as ST-T changes: flattened or negative T wave, depression or elevation of the ST interval. These ECG manifestations are a direct reflection of disorders in the electro-physiological properties of conductive and contractile myocardial cells. In this case, the load and pharmacological tests are generally negative.

Echocardiography, as a rule, does not reveal deviations from the age norm and only in some patients, especially in the distal stages of myocardiodystrophy, slight dilation of the heart chambers and a decrease in the contractile function of the myocardium can be determined.

Nuclear magnetic resonance imaging is a highly promising method for diagnosing myocardial dystrophy. This research method allows visualization of the heart and, in combination with radioactive phosphorus spectroscopy, quantitative

assessment of the content of high-energy phosphates in cardiomyocytes, allowing for intracellular pH measurement. In broad practice, these studies are not yet being used.

Currently, one of the informative methods for diagnosing MCD in children is scintigraphy with Tl-201. Thallium is incorporated into the K+-, Na+-ATPase cellular pump, where it replaces the K+ ion and, without compromising the function of the ionic cellular pump, is distributed throughout the myocardium, allowing for the assessment of the functional integrity of cardiomyocytes, i.e., perfusion and metabolism. Using scintigraphy, it was determined that in children with myocardial dystrophy, metabolic processes predominantly suffer. Both diffuse and focal accumulation defects were identified, indicating a decrease in the number of functioning cardiomyocytes, but at the same time, sufficient myocardial contractility persisted.

In recent years, numerous works have emerged discussing mitochondrial dysfunctions in many diseases, particularly myocardiodystrophy. Mitochondrial dysfunctions lead to cell energy deficiency, which plays an important pathogenetic role in the development of MCD. Thus, a group of Russian authors conducted a study to determine the activity of mitochondrial enzymes in peripheral blood lymphocytes in children with myocardiodystrophy, revealing significant metabolic disorders. Myocardial biopsy can be considered a crucial diagnostic method for MCD. However, in MCD, there are usually no indications for it. In the initial stages of MCD, only ultrastructural changes in cardiomyocytes are detected, and gistochemical examination confirms enzymopathies.

We present the most common diseases and conditions in which there are always signs of myocardial dystrophy to one degree or another.

MCD in thyrotoxicosis. In thyrotoxicosis, two factors play a key role in the pathogenesis of MCD development:

1. Under the influence of an increased amount of thyroid hormones in the myocardium, oxidative phosphorylation is disrupted. This leads to a decrease in ATP levels, energy deficiency, and subsequently, protein deficiency.

2. Under the influence of thyroid hormones and increased sympathetic nervous system activity, significant hemodynamic disturbances occur - the minute volume increases, mainly due to increased heart rate, blood flow rate and circulating blood volume increase, peripheral resistance in the systemic circulation decreases and increases in the pulmonary circulation.

Such changes in hemodynamics require increased energy supply, which is absent. Eventually, myocardiodystrophy develops. The peculiarities of the clinical manifestations of myocardial dystrophy in thyrotoxicosis are the predominance of such arrhythmias as sinus tachycardia, extrasystole, paroxysmal tachycardia attacks, and even paroxysmal and/or atrial fibrillation can develop. Against this background, with prolonged thyrotoxicosis, chronic circulatory insufficiency develops, mainly of

the right ventricular type (swelling, hepatomegaly). Pain in the heart area is relatively rare.

In some patients, signs of myocardial dystrophy (for example, rhythm disturbances) may dominate the clinical picture of thyrotoxicosis, for which children are admitted, first of all, to a cardiologist, and then only to an endocrinologist.

Treatment of myocardial dystrophy in thyrotoxicosis necessarily involves the use of thyrostatic agents. Due to concurrent sympathicotonia, beta-blockers are also indicated for them.

We observed patients with thyroid hyperfunction, in whom various tachyarrhythmias practically did not respond to antiarrhythmic therapy. The effect occurred only with the prescription of complex therapy for thyrotoxicosis with the mandatory inclusion of thyrostatic drugs prescribed by an endocrinologist after a thorough examination of the patient.

MCD in hypothyroidism. In hypothyroidism, the basis for the development of MCD is the decrease in metabolic processes in the myocardium due to a decrease in thyroid hormones. Oxygen absorption decreases, protein synthesis decreases. At the same time, the permeability of vessels in the myocardium increases, the amount of interstitial fluid increases, which, as if pushing out myofibrils, leads to dismetabolic disorders in the cells and their swelling (sodium content increases, potassium content decreases, fluid retention occurs).

Clinically, myocardiodystrophy in hypothyroidism manifests as constant and aching pain in the heart area, arrhythmias in the form of sinus bradycardia, and various blockades (atrioventricular, atrial, ventricular). Rhythm and conduction disturbances are documented by corresponding changes in the ECG. Typical changes in the ECG, in addition to the indicated manifestations, are also the low voltage of the ECG waves, the presence of a flattened or negative T wave, especially in the chest leads.

The main method of treating MCD in hypothyroidism is the administration of thyroid hormones. The patient should be observed by an endocrinologist.

MCD in anemia. In anemia of any origin, the hemoglobin content and the number of erythrocytes decrease. Hemic hypoxia develops, which leads to energy deficiency in the myocardium. In the initial stages of anemia, moderate energy deficiency causes adaptive stimulation of blood circulation and increased cardiac function (its hyperfunction), which is aimed at preventing disorders of oxidation processes in tissues, including the myocardium.

The clinical manifestation of these disorders is a circulatory-hypoxic (hypoxemic) syndrome, characteristic of all types of anemia, manifested by shortness of breath, tachycardia, loud heart sounds, and systolic murmurs above the heart and blood vessels, which is due to increased blood flow velocity. With prolonged persistence of anemia, and therefore tissue hypoxia, an intensifying energy deficit leads to the development of dystrophic changes in the myocardium and the

suppression of its functional capacity. Against the background of increasing circulatory-hypoxic syndrome, patients develop changes in the ECG: flattened or negative T wave, incomplete ventricular blocks, moderate decrease in the ST interval in the chest leads, atrial or ventricular extrasystole, sometimes atrioventricular block of I and even II degree. With prolonged pronounced anemia, insufficient treatment can lead to heart failure.

The therapy of myocardial dystrophy against the background of anemia consists, first of all, in the treatment of anemia depending on its genesis (preparations of iron, vitamins, if indicated - glucocorticosteroids, etc.). Treatment of MCD directly has no specificity and is carried out against the background of anemia therapy according to the generally accepted schemes described above.

MCD in chronic tonsillitis. The most frequent manifestation of tonsillagen MCD is pain in the heart area, piercing, aching, prolonged, often very intense. Often, various rhythm disturbances are detected: irregular sinus rhythm, migration of the rhythm source, intraventricular and intraventricular blockades, extrasystole.

Etiotropic therapy is active complex treatment of chronic tonsillitis up to tonsillectomy (upon indications).

MCD post-myocarditic. After experiencing acute myocarditis, dystrophic changes in the myocardium can persist for 6-12 months or more. At the same time, changes are detected mainly on the ECG. Reduction of repolarization processes in the left chest leads (smoothing or inversion of the T wave), concomitant atrioventricular blocks of I-II degree, as well as various blocks of the bundle of Giss's feet, are more common. Sufficiently persistent ectopic rhythm disturbances in the form of extrasystole, parasystole, and less frequently, atrial fibrillation can be observed.

In the treatment of patients with post-myocarditic MCD, cardiotrophic and vascular drugs (**panangin**, mildronate, riboxin, neoton, magne-B6, etc.) are used.

MCD of toxic origin. This variant of MCD occurs in patients who have been receiving immunosuppressants (cytostatics, glucocorticosteroids, nonsteroidal anti-inflammatory drugs) for a long time.

MKD mainly manifests as changes in the ECG in the form of depression of the T wave, ST segment, and elongation of the QT interval. Some rhythm disturbances may be observed: sinus tachycardia or bradycardia, extrasystole, bundle branch blockades. Treatment is similar to the therapy for other variants of MCD (cardiotrophic, vascular drugs, vitamins, antioxidants).

Tactics and Methods of Treatment of MCD in Children

The success and effectiveness of MCD therapy in a child largely depends on the treatment and elimination of the cause that caused MCD, its duration, severity, and the clinical manifestations of metabolic disorders in the myocardium.

Pathogenetic therapy of MCD is carried out with cardiotrophic agents. These are medications of various pharmacological groups capable of improving metabolic

processes in the myocardium. The question of the effectiveness of these drugs has not been definitively resolved, since for most of them, the effectiveness has not been assessed in strictly controlled studies. In this regard, when prescribing cardiotrophic drugs, it is advisable to avoid polypragmasia, based on the predominance of the visible link in the pathogenesis of MCD.

To correct protein metabolism in the myocardium, vitamins and their coenzymes (folic acid, vitamins B6 and C) can be recommended, and magnesium and potassium preparations that activate protein synthesis can be used. The absorption of amino acids is enhanced by the administration of drugs with anabolic effects (retabolil, potassium orotate, riboxin, mildronate, etc.), therefore, their administration may be appropriate in combination with other medications.

Electrolyte imbalances are most actively corrected by prescribing combined medications containing potassium and magnesium salts (panangin, magnerot, etc.).

To correct energy metabolism, vitamins of the B group, cocarboxylase, ATP are used, and riboxin, mildronate, and antioxidant complexes (vitamins A + E + C) can also be used, which contribute to a decrease in lipid peroxidation and improve the antioxidant defense of the cell. Treatment courses with the drug "Neoton" (creatinine phosphate) intravenously are effective.

To eliminate the clinical manifestations of MKD, symptomatic agents are used (according to indications). Sanitation of chronic infection foci, all comorbidities that can cause or maintain MCD formation, is mandatory.

Cardialgia syndrome in MCD (often of a neurotic nature) in the patient is usually managed to be suppressed by etiotropic and pathogenetic therapy, sometimes it is necessary to prescribe multicomponent drugs such as validol, valokordin, corvalol, and sedatives (novopassit, tinctures or decoctions of arsenic, valerian, etc.).

In the presence of persistent arrhythmias, treatment is carried out with antiarrhythmic agents; in heart failure - with the appointment of cardiac glycosides, diuretics; if indicated, treatment courses can be conducted with agents from the group of angiotensin-converting enzyme inhibitors.

For extrasystole, antiarrhythmic drugs are used. It is advisable to conduct a drug test before prescribing them.

The drug of choice for supraventricular extrasystoles is calcium channel antagonists (verapamil) at 80-120 mg/day for 10-14 days. Sotalol or beta-blockers can be used.

For ventricular extrasystoles, ethacizine or etmozine is used up to 300-400 mg/day for 7 days, then at half a dose for 1-2 months.

In cases of sinus tachycardia and a positive functional test with adrenergic blockers, selective beta-blockers (atenolol, metoprolol, etc.) are indicated for 2-3 weeks at 50-100 mg/day. In cases of pronounced sinus bradycardia combined with

vagus-dependent extrasystoles, drugs that reduce vagus activity (amizil, bellataminal) are indicated. They are prescribed at 1.0 mg 3-4 times a day for 2-3 weeks.

In the complex treatment of patients with MCD, it is possible to recommend treatment courses with the daytime tranquilizer adaptol, which has a multifaceted effect (vegetotropic, has a positive effect on the cardiovascular system, relieves anxiety, improves sleep, etc.).

A special role in the treatment of all forms of CVD is given to the use of complex preparations of magnesium and potassium salts, which play an important role in the activity of the cardiovascular system.

It has been established that there is an antagonism between the action of magnesium and calcium in relation to the smooth muscles of the vessels and the myocardium. Therefore, magnesium is especially active in combination with potassium (calcium's active antagonist). Minimal disruptions in intracellular magnesium levels, especially in combination with potassium, actively alter myocardial cell function and vascular tone.

In stressful situations, the loss of magnesium by the body increases due to the influence of catecholamines on the absorption of magnesium and the metabolic processes associated with its participation. Firstly, the increased release of catecholamines (adrenal hormones) into the blood leads to the loss of cellular magnesium and its elimination with urine due to reduced reabsorption in the tubular apparatus. Secondly, catecholamines intensify lipolysis, which causes an increase in the content of free fatty acids that bind the ionized magnesium of the plasma. The release of intracellular magnesium is activated.

A decrease in magnesium content (especially in combination with a decrease in potassium) leads to a change in the magnesium/calcium ratio in the adrenal cortex cells, which causes an increase in the secretion of mineralocorticoid hormones, which further exacerbate the body's magnesium loss.

A possible role of magnesium deficiency in the pathogenesis of atherosclerosis has been established, since this microelement plays an important role in lipid metabolism and the development of hyperlipidemia with magnesium deficiency: in hypomagnesemia, the content of low-density lipoproteins increases.

In the experiment, when examining the vessels of animals with magnesium deficiency, changes characteristic of the early stages of atherosclerosis were established: muscle cell hyperplasia, fibrinoid necrosis and chronic adventitial inflammation, lipid infiltration of the endothelium, swelling and thickening of the intima, cleavage and fragmentation of the inner elastic layer, and the appearance of lipid spots.

The mechanism of the hypolipidemic effect of magnesium salts is complex and is interpreted ambiguously. Firstly, magnesium reduces lipolysis by activating the release of noradrenaline from the adrenal glands and the ends of sympathetic nerve

fibers. Secondly, magnesium salts increase the level of cAMP, which increases the activity of lipoproteins-splitting lipases.

Magnesium enhances the activity of an enzyme that increases cholesterol esterification and is a cofactor of enzymes that regulate lipid metabolism.

In arterial hypertension, significant magnesium retention was established during the load test, and a reverse correlation between systolic and diastolic blood pressure and the magnitude of magnesium excretion with urine was revealed. An inverse correlation has also been established between the concentration of magnesium in erythrocytes and blood pressure levels.

For patients with arterial hypertension who respond to magnesium therapy, higher renin activity in blood plasma is characteristic compared to those who do not respond to treatment.

Magnesium is one of the active regulators of vascular tone. It has been established that the frequency of arterial hypertension increases in biogeochemical regions where the magnesium content in drinking water is reduced.

Adverse effects associated with magnesium deficiency, manifested by increased tone, coronary artery spasm, increased sensitivity to vasoconstricting agents (angiotensin, serotonin, noradrenaline, acetylcholine), decrease quite quickly and actively when magnesium and potassium preparations are included in the complex therapy.

Currently, panangin, a complex drug, is most actively used in clinical practice.

Panangin is a combination of magnesium asparaginate and potassium asparaginate. The drug normalizes the intracellular and extracellular ratio of potassium, magnesium, calcium, and sodium ions, which contributes to the improvement of myocardial contractility. Upon entering the cells, asparaginate is incorporated into metabolic processes.

Endogenous asparaginate is a conductor of ions due to its high affinity for cells. In addition, ions in the form of complex compounds penetrate the cell due to the insignificant dissociation of its salts. Potassium and magnesium aspartates improve myocardial metabolism, improve coronary blood circulation, and enhance the effects of antiarrhythmic drugs.

Indications for panangin are: MCD, heart rhythm disturbances (ventricular extrasystole, atrial fibrillation, tachycardia), overdose and glycoside intoxication, and other conditions accompanied by symptoms of hypokalemia and hypomagnesemia.

Contraindications for prescribing the drug: hyperkalemia and hypermagnesemia, atrioventricular blockades of I-III degree, acute and chronic renal failure, increased individual sensitivity to the drug's components.

When prescribing panangin to patients, it is necessary to remember its interaction with other medications. Simultaneous administration with potassium-saving diuretics, beta-blockers, angiotensin-converting enzyme inhibitors, heparin,

and nonsteroidal anti-inflammatory drugs increases the risk of developing hyperkalemia. Prescribing potassium and magnesium drugs in conjunction with hormonal drugs (glucocorticosteroids) eliminates the hypokalemia developing during steroid administration. Panangin reduces the adverse side effects of cardiac glycosides, enhances the negative drono- and bathmotropic effects of antiarrhythmic drugs. It should also be noted that calcium preparations reduce the effect of magnesium and potassium preparations.

Panangin is available in tablet form, coated with a film film. It is recommended to prescribe 1 tablet 2-3 times a day after meals, depending on the patient's age (8-9 years old - 1 tablet, from 10 to 14 years old - 2 tablets, older than 14 years old - 3 tablets per day). The duration of the treatment course is determined individually, usually 3-4 weeks.

Dispensary observation of patients with CMD of various origins continues until all clinical symptoms disappear, as well as until the ECG normalizes (at least 6-12 months). The physical load is regulated individually. Schoolchildren are assigned physical therapy or a preparatory group for physical education (without cross-country races or competitions). Sanitation of chronic infection and comorbidity foci is mandatory. Complex cardiotrophic therapy courses are recommended 2-3 times a year (1.0-1.5 months).

Sports doctors, as well as pediatric cardiologists, rheumatologists, and pediatricians, should be engaged in the prevention of ICD development, especially in young athletes. First of all, it is necessary to carefully select children for sports. Sanitation of chronic infection sites, timely treatment of all diseases, adherence to a daily routine, and proper nutrition play an important role in the prevention of MCD.

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