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Case of acute recurrent myocardial infarction in a child

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Information on the essential aspects of prevalence, etiology and pathogenesis of coronary heart disease, which is the leading cause of death in the world, is given in this article. It is emphasized that the incidence of ischemic coronary events is the result of a complex sequence of pathogenetic links, which involves the formation of atherosclerotic lesions and destabilization as the primary provoking factor.

Purpose — the attraction of the medical community attention to the problem of ischemic heart disease in children.

A **clinical case** of recurrent myocardial infarction in a child who was examined and treated at the MNPE «Ivano-Frankivsk Regional Children's Clinical Hospital of the Ivano-Frankivsk Regional Council», is described here. The key points of the patient's complaints, anamnesis of life and this disease are described, the data of the objective condition at the hospitalization and during dynamic observation of the child, which showed the signs of left ventricular overload, coronary blood flow disorder, dilatation of the left heart ventricles, decreased left ventricular contractility. The data of the main indices of laboratory and instrumental methods of research, including X-ray diagnostic methods, performed not only on the basis of the department, but also in the Scientific-Practical Medical Center of Pediatric Cardiology and Cardiac Surgery of the Ministry of Health of Ukraine (Kyiv). The treatment of this clinical case in the child is described in this article. The data of dynamic observation are given. The discussion and conclusions emphasize the problem of coronary insufficiency in pediatrics, which requires clinical understanding, the choice of the correct diagnostic algorithm and qualified medical care.

The study was performed in accordance with the principles of the Declaration of Helsinki. Informed consent of the child's parents was obtained for the research conduction.

No conflict of interests was declared by the authors.

Keywords: children, coronary heart disease, myocardial infarction, clinic, diagnosis.

Випадок гострого повторного інфаркту міокарда в дитини

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Наведено відомості про основні моменти поширеності, етіології та патогенезу ішемічної хвороби серця, яка є провідною причиною смерті у світі. Наголошено, що інцидент ішемічних коронарних подій є наслідком складної послідовності патогенетичних ланок, що передбачає формування атеросклеротичного ураження та дестабілізацію як первинний провокативний фактор.

Мета — привернути увагу медичної спільноти до проблеми ішемічної хвороби серця в дітей.

Описано **клінічний випадок** повторного інфаркту міокарда в дитини, яка перебувала на обстеженні та лікуванні в КНП «Івано-Франківська обласна дитяча клінічна лікарня» Івано-Франківської обласної ради. Описано основні моменти скарг пацієнтки, анамнезу життя й цього захворювання, наведено дані об'єктивного стану на момент госпіталізації та отримані під час динамічного спостереження за дитиною, які свідчили про ознаки перевантаження лівого шлуночка, порушення коронарного кровоплину, дилатацію лівих камер серця, зниження скоротливої спроможності лівого шлуночка. Представлено основні показники лабораторних та інструментальних методів дослідження, у тому числі рентгенологічних методів діагностики, виконаних не тільки на базі відділення, але й у Науково-практичному медичному центрі дитячої кардіології та кардіохірургії МОЗ України (м. Київ). Детально описано лікування дитини. Наведено дані динамічного спостереження. В обговоренні та висновках наголошено, що проблема коронарної недостатності в педіатрії існує, а це потребує клінічного розуміння, вибору коректного діагностичного алгоритму та якісного медичного супроводу. Дослідження виконано відповідно до принципів Гельсінської декларації. На проведення досліджень отримано інформовану згоду батьків дитини.

Автори заявляють про відсутність конфлікту інтересів.

Ключові слова: діти, інфаркт міокарда.

Cardiovascular disease is the leading cause of death in the world — 17.3 million people die for a cause of them every year. Coronary heart disease (CHD) has been characterized as «the most common epidemic of mankind, threatening to spread to the Earth's entire population in the near future if its prevention would not be possible to develop by studying the pre-disease and disease onset» [3]. Although the successes of preventive lifestyle and medical

interventions in CHD is almost unprecedented, the achievable risk reduction remains below 40%. Some acute ischemic events in adults have their origin in childhood [9]. In addition, most episodes of ischemia occur in persons not determined by risk profiling, it has been proven that CHD is more often stochastic than deterministic [7].

The incident of ischemic coronary events is the result of a complex sequence of pathogenetic links involving the formation of atherosclerotic lesions

and destabilization as the primary provoking factor. The actual development (or absence) of clinical manifestations is associated with the occurrence or absence of thromboembolic complications. The development of the latter ones and their clinical consequences reflect the complex interaction of pro- and antithrombotic factors, fibrinolytic system, ischemia time, residual size of the coronary lumen, the necessity for oxygen supply to tissues, existing collateral vascular networks, etc. [8].

In recent decades, the incidence of circulatory diseases among children under 14 years of age has increased 2.1-fold, and mortality from CHD in people under 30 years of age has increased at 15–20%. In 2014 alone, 11 children died of CHD in Ukraine, or 0.015 per 10,000 children [10]. The main risk factors for cardiovascular disease, particularly in adolescents, are: high blood pressure and cholesterol and blood glucose levels; smoking; insufficient consumption of vegetables and fruits; overweight and obesity; hypodynamia, etc. Recent studies have shown that 17% of children under 5 years of age have atherosclerotic changes in the coronary arteries, and an increase in basal triglycerides in children aged 5–17 years with paternal early coronary heart disease (CHD) (especially maternal line) is a predictor of metabolic syndrome development [6].

Acute coronary syndrome (ACS) in childhood is a relatively rare pathology, due to the partial difficulties in diagnosis and lack of general practitioners' vigilance. Among the reasons of ACS in children, the most common ones are congenital anomalies of the coronary arteries and acquired pathology (coronaritis, carditis, tumors, traumas, etc.) [1].

Purpose of the study – the attraction of the medical community attention to the problem of ischemic heart disease in children.

Clinical case

A clinical case of recurrent myocardial infarction in a child is represented in this article. The girl aged 21 months, was admitted to the Nephrology Department (cardiorheumatology bed) of the Regional Children's Clinical Hospital in Ivano-Frankivsk with a diagnosis of «Acute bilateral lower lobe focal pneumonia complicated by toxic syndrome. Dilated cardiomyopathy?».

The study was performed in accordance with the principles of the Declaration of Helsinki. Informed consent of the child's parents was obtained for the research conduction.

Complaints of the mother on admission: the child has a wet cough, shortness of breath, pale skin, increased fatigue and lethargy of the child, poor weight gain.

It was established from the anamnesis that during the last 6 months before the actual hospitalization the child had an inpatient treatment for pneumonia and acute respiratory diseases three times. There is a history of acute pyelonephritis, aphthous stomatitis, acute enterocolitis (rotaviral infection).

From the anamnesis of life it is known that the child was born from the second full-term pregnancy, the second urgent birth, with a body weight of 2800 g. The marriage is not related. Mother is 28 years old, father is 33 years old. The pregnancy proceeded without complications. The child was vaccinated out of schedule due to frequent illnesses. Family tuberculosis is denied by parents.

Objectively, the child's condition on admission is severe due to heart and respiratory failure. Shortness of breath at rest, respiratory rate – 46/min. The child has proper physique, low nutrition (weight deficit – 1.8 σ). The skin is clean, pale. Visible mucous membranes are clean, pale pink. The tongue is wet, uncoated. Peripheral lymph nodes are not enlarged. Above the lungs at the percussion – shortening of the percussion sound paravertebrally and in the lower parts, auscultatory – harsh breathing, wet conductive rales diffusely. The boundaries of cardiac dullness are extended to the left. Heart tones are rhythmic, of low sonority, tachycardia, heart rate – 170 beats/min, systolic murmur at the apex, non-conductive. The abdomen is soft and painless. The liver protrudes from the edge of the costal arch at 3.5 cm. The spleen is not palpable. Evacuation is 1 time/day, formed. Urination is free. There is some swelling of the feet.

Paraclinically: hemogram – neutrocytosis, shift of leukocyte formula to the left (rod nuclear cells – 11%, segmented cells – 45%), biochemical analysis of blood – hypoproteinemia (44.2 g/l), hypoglycemia (2.6 mmol/l), coagulogram – without peculiarities, general analysis of urine – proteinuria (0.165 g/l), ketonuria (+), procalcitonin – 0.139 ng/ml. Immunogram: IgG – 4.9 g/l, IgM – 0.78 g/l, IgA – 0.27 g/l.

Electrocardiography (ECG): heart rate – 182–188 for 1 min. There are: paroxysmal supraventricular tachycardia, coronary blood flow disorders. Bioelectric systole is normal. Deviation

of electrical cardiac axis (ECA) to the left. Increased ECG voltage. Left ventricular overload.

Echocardiography: dilatation of the left parts of the heart: Left ventricular end-diastolic diameter (LVEDD) – 3.8 cm (norm 2.4–3.2 cm), pulmonary artery (PA) – 2.9 cm (norm 1.5–2.1 cm). Ejection fraction – 22%. Mitral valve – regurgitation (+). Tricuspid valve – regurgitation (+). In the abdominal aorta, the blood flow is pulsating. Leaves of pericardium: no abnormalities. The walls of the myocardium are not thickened. Total contractility is reduced. Pleural cavities – there is a small amount of effusion.

Ultrasound of the abdominal cavity: hepatomegaly, parenchyma of normal echogenicity. Hepatic veins are without peculiarities. The spleen is not enlarged, structurally heterogeneous. Echogenicity of the renal parenchyma is increased. There are calcinates in the thickness of the parenchyma (1–2 mm). The hollow system is not broadened.

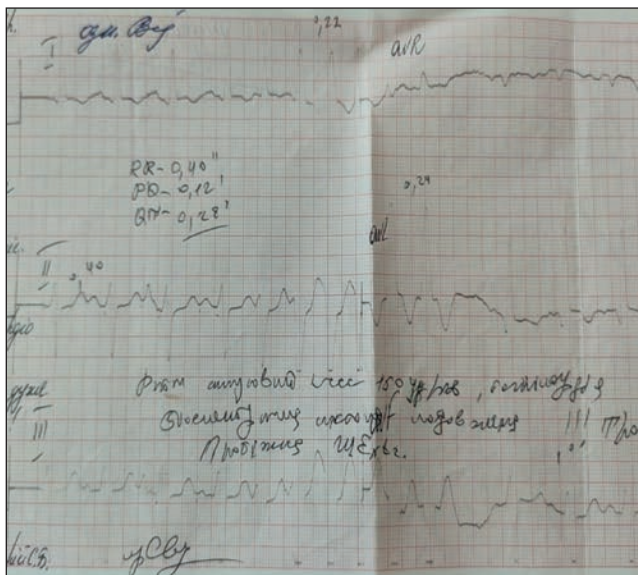


Fig. 1. ECG: heart rate – 150 per 1 min, ischemic changes in the myocardium

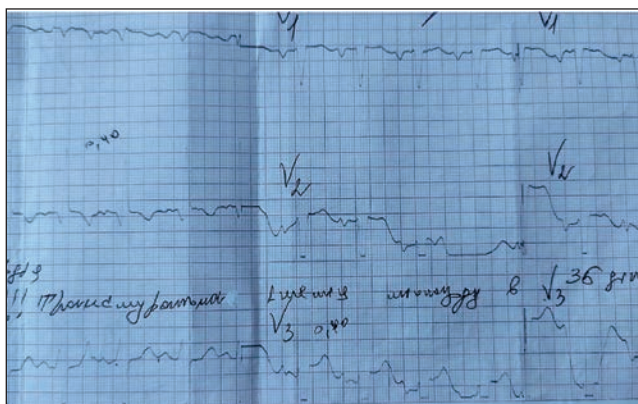


Fig. 2. ECG: heart rate – 150 per 1 min, ischemic changes in the myocardium

Thoracic organs radiography: lung markings are enriched, strengthened and indistinct in both lungs. The roots are dilatated. The sinuses are free. The boundaries of the heart are significantly extended to the left. Conclusion: cardiomegaly, hypervolemia in the small circle of blood circulation.

The dynamics contained signs of left ventricular overload on the ECG, coronary blood flow disorders, dilatation of the left ventricles of the heart, decreased contractility of the left ventricle (EF – 30%).

Diagnosis: «Dilated cardiomyopathy. CHF of the IIA–IIB degree. Anomaly in the development of coronary arteries: ALCAPA syndrome? Coronaritis? Bilateral focal pneumonia, of stagnant-bacterial genesis, acute course, respiratory depression (RD) of the I degree. Moderate protein-energy deficiency. Micronephrolithiasis».

In order to perform the additional examination and verification of the diagnosis, the child was sent to the Scientific-Practical Medical Center of Pediatric Cardiology and Cardiac Surgery of the Ministry of Health of Ukraine (Kyiv).

Echocardiography: LV EDD – 38 mm, end-diastolic index of the left ventricle (LV EDI) – 142.18 ml/m², the cavity is significantly expanded, ejection fraction (LVEF) – 30%, contractility is significantly reduced, interventricular septum thickness – 9 mm, left ventricular posterior wall thickness – 8 mm, left ventricular myocardial mass – 9337 g, left ventricle LV wall hypertrophy, interventricular septum is thickened up to 10 mm in the middle third, increased trabecularity, abnormal coronary blood flow in the walls of the left ventricle. Coronary arteries depart from the aorta, left – 3 mm, right – 2 mm, tiny coronary-left ventricular fistulas are not excluded. Conclusion: myocardial pathology. Moderate left ventricular hypertrophy with moderate dilatation and systolic dysfunction.

Magnetic resonance therapy (MRI) of the heart: the left ventricular cavity is moderately dilated. Coronary arteries branch out without peculiarities. With the introduction of contrast – there is the hypoperfusion of the interventricular septum in the middle and apical parts, subendocardial parts of the myocardium, along the lateral wall of the left ventricle in the middle and apical parts, in the apex. There is a transmural accumulation of contrast agent in the apical-anterior septal segment with myocardial damage more than 75%, interventricular septum – in the middle parts,

anterior LV wall in the middle and apical parts with myocardial damage up to 50%, anterolateral papillary muscle, subendocardially of lateral wall in the middle and apical areas. There are no blood clots in the heart cavities. There is some free fluid in the pericardial cavity up to 0.4 cm in the apex. Free fluid in the pleural cavities is absent.

X-ray surgical examination of the heart and major vessels: the right coronary artery has multiple stenoses of peripheral branches, the distal part is filled retrogradely, through the flows from the interventricular branch. The diagonal branch has numerous stenoses. There is the left type of coronary blood flow, circumflex artery (Cx) is not changed. Blood flow in the apex of the heart is sharply reduced.

Based on the performed examinations (MRI of the heart with contrast medium, coronary angiography, probing of the heart cavities), the diagnosis was made: «Condition after myocardial infarction. Multiple peripheral stenoses of the coronary arteries. Pronounced systolic dysfunction of the left ventricle. Coronaritis? Circulatory inefficiency of the IIA degree. Primary immunodeficiency, unspecified». Treatment is prescribed: enalapril, verospirone, aspirin-cardio, trimetazidine.

One week after discharge from the Cardiac Surgery Center, the mother noted that the child had increased shortness of breath, fatigue and lethargy. The child was re-hospitalized into the Regional Hospital. An objective examination revealed that the general condition of the child was severe due to the manifestations of chronic cardiovascular insufficiency. She is sluggish, inactive, responds to the examination by crying. Shortness of breath at rest, respiratory rate 35–40 per 1 minute. The child is of proper physique, reduced nutrition. The skin is clean, pale, acrocyanosis is present. Above the lungs at the percussion — there is some pulmonary sound, auscultatory — harsh breathing, weakened in the lower parts. The limits of relative cardiac dullness are extended to the left. Heart tones are rhythmic, of low sonority, there is a systolic murmur at the apex and base of the heart, non-conductive, tachycardia, heart rate = 140–160 beats/min. The abdomen is soft and painless. The liver protrudes at 1.5 cm from the costal arch. The spleen is not palpable. Evacuation is daily, formed. Urination is free. Peripheral edema is absent.

Paraclinically: in the hemogram — there is the deficient anemia of mild degree (hemoglobin —

98 g/l), deviations from norm are not revealed in the general analysis of urine. In the biochemical blood analysis — there is a decrease in serum iron (4.8 $\mu\text{mol/l}$), dyslipidemia: a decrease in high density lipoproteins (0.53 mmol/l) and low density lipoproteins (2.33 mmol/l), an increase in the atherogenicity index (8.32), markers of myocardial damage (CPK, troponin-I) are above normal: creatine phosphokinase (CPK-MB) — 28 units/ml (norm up to 24 units/ml), troponin I — 0.423 ng/ml (maximum positive concentration 0.32–0.50 ng/ml). Coagulogram shows an increase in international normalized ratio (INR) (1.53), platelet hypoaggregation (8.6%). Glycemic profile — without abnormalities. Immunological blood test — hypogammaglobulinemia due to the decreased IgG levels (3.8 g/l).

Examination for markers of Toxoplasma, Rubella, Cytomegalovirus, Herpes infections (TORCH infections) (mycoplasma, ureaplasma, chlamydia, herpes virus type I and II, cytomegalovirus, Epstein–Barr virus) did not reveal data on the activity of these infections.

ECG: heart rate — 150 per 1 min, tachycardia. There are ischemic changes in the myocardium. Bioelectric systole is normal. LV hypertrophy (Fig. 1, Fig. 2, Fig. 3).

On the basis of a typical ECG picture (elevation of the ST segment >2 mm in leads V₃-V₆, negative wave «T» in the I standard lead, tachycardia, deviation of the ECA to the left).

Ultrasound examination of the heart: the left parts are sharply extended: the left ventricle — 3.8 cm (N 2.8+0.4 cm), EDI — 137 ml/m², the left atrium — 2.9 cm (norm 1.8–2.2 cm). The total contractility of the left ventricle is reduced: the ejection fraction is 30%. Mitral valve - regurgitation (+). Tricuspid valve — regurgitation (+).

Thoracic organs radiography: lung markings in the medial parts of both lungs are enriched, indistinct. The roots are dilated. The sinuses are free. The borders of the heart are extended to the left. *Conclusion:* X-ray picture is characteristic in dilatation of the left ventricle, hypervolemia in the small circle of blood circulation.

Ultrasound examination of the abdominal cavity, neurosonography, ultrasound examination of the thymus — are without pathological changes: echocardiographic data (sharply reduced LV myocardial contractility (EF — 22–30%), dilatation of the left ventricle and atrium), X-ray data of the thoracic organs (enriched, indistinct lung mark-

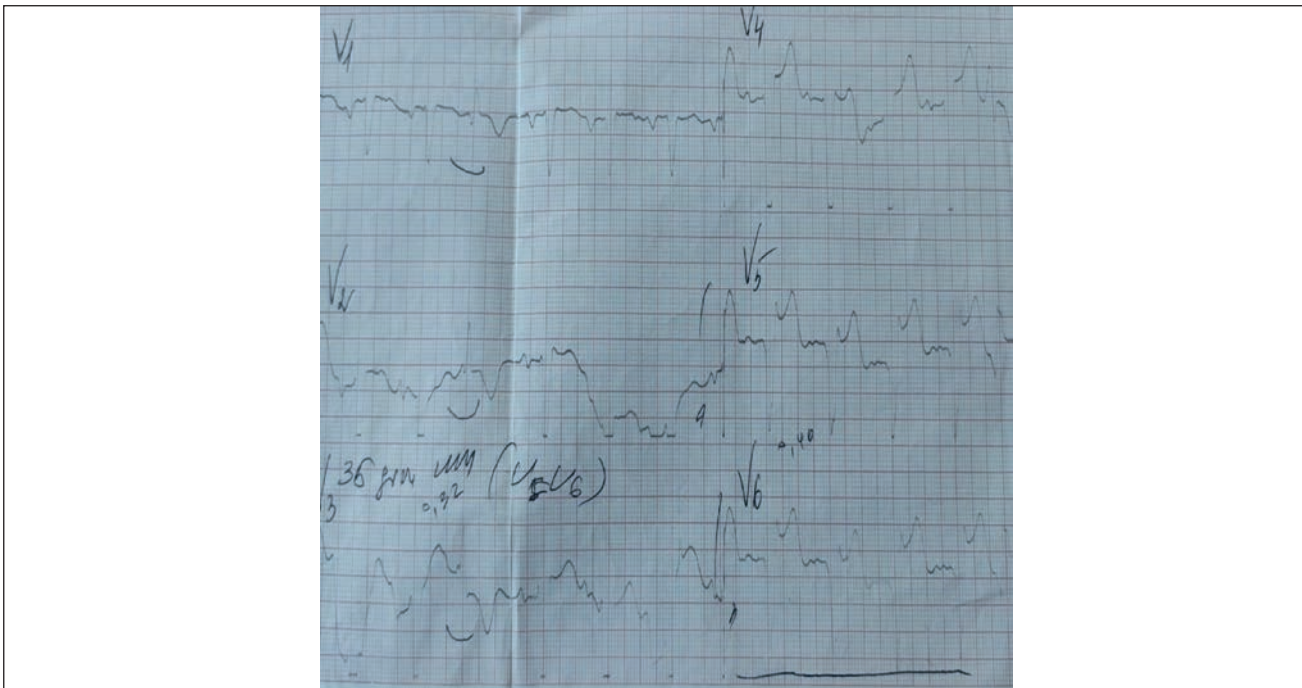


Fig. 3. ECG: heart rate — 150 per 1 min, ischemic changes in the myocardium

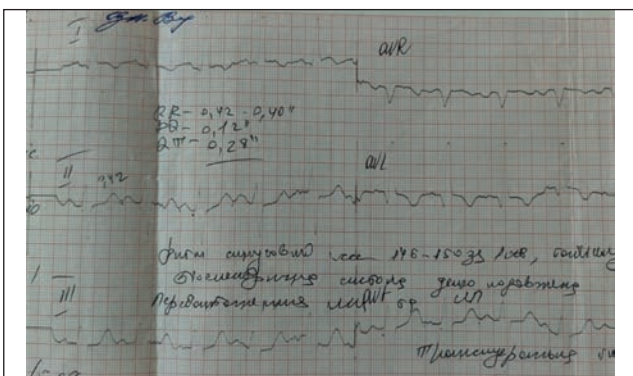


Fig. 4. ECG: sinus rhythm, heart rate — 146–150 per 1 min. Transmural myocardial ischemia in the posterobasal region of the left ventricle

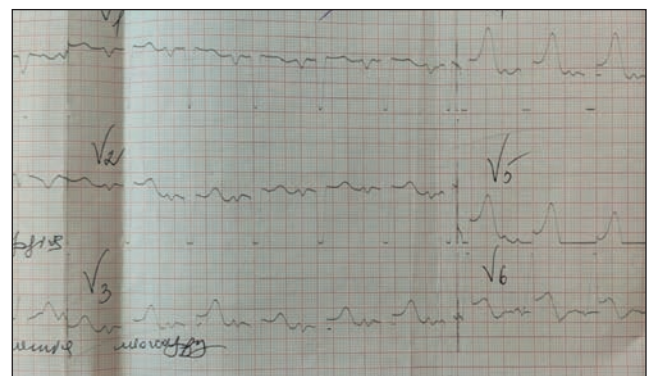


Fig. 5. ECG: sinus rhythm, heart rate — 146–150 per 1 min. Transmural myocardial ischemia in the posterobasal region of the left ventricle

ings, dilated roots, heart borders are extended to the left), laboratory data (increased level of troponin I, CPK-MB, decreased concentrations of IgG, serum iron and hemoglobin), as well as examinations performed at the Center for Pediatric Cardiology and Cardiac Surgery (Kyiv), the diagnosis was made: «Congenital heart disease: abnormal development of coronary arteries with multiple stenoses of peripheral branches. Condition after myocardial infarction. Severe systolic dysfunction of the left ventricle. CHF of the IIA degree. Primary immunodeficiency, unspecified. Moderate protein-energy deficiency. Mild iron deficiency anemia».

The child was prescribed such a treatment: enalapril, verospirone, eplerenone, furosemide, torasemide, metoprolol, asparkam, cardonate, trimetazidine, aspirin-cardio.

In the dynamics the child's condition deteriorated, the child became more irritable, restless.

ECG: sinus rhythm, heart rate — 146–150 per 1 min, tachycardia. Bioelectric systole — prolonged. Transmural myocardial ischemia in the posterobasal region of the left ventricle. There is the run of supraventricular tachycardia (Fig. 4, Fig. 5).

Echocardiography: preliminary data.

In the dynamics there was a steady increase in the concentration of troponin-I: 1.51 ng/ml, which corresponds to a critical value (>0.50 ng/ml), 1.05 ng/ml, 1.47 ng/ml.

Addition to the diagnosis: «Acute recurrent myocardial infarction with localization in the posterolateral LV area. Severe systolic dysfunction of the left ventricle. CHF of the IIA – IIB degree».

Nitroglycerin patch on the chest, morphine, noophene were added to the treatment.

In dynamics, the condition remained severe, clinically stable. ECG: sinus rhythm, heart rate —

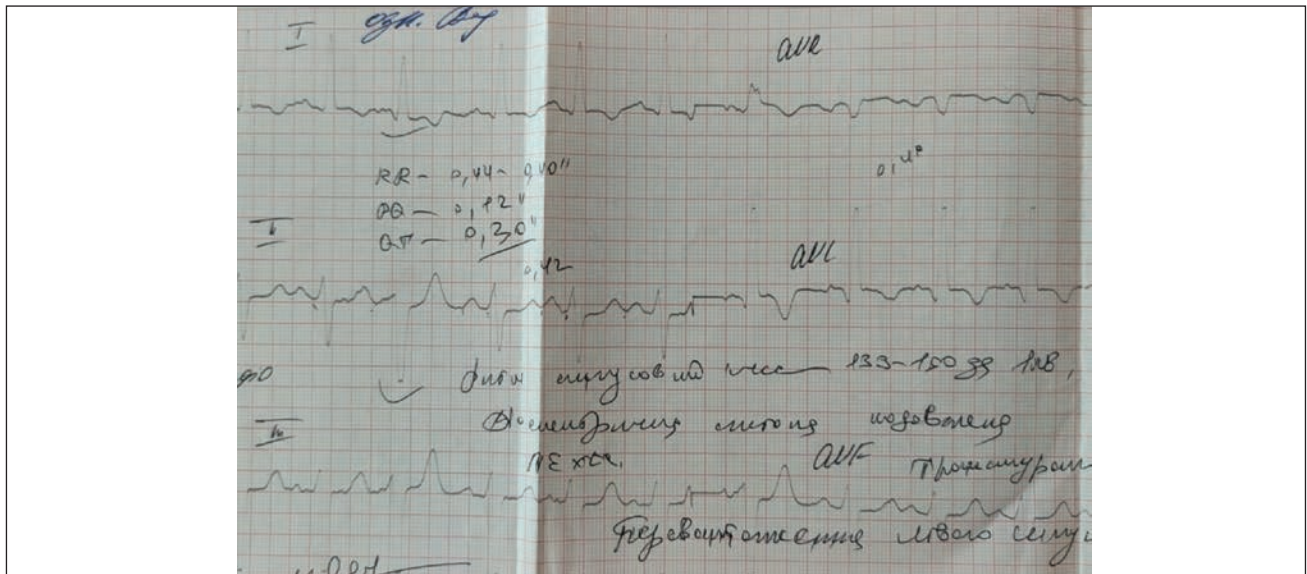


Fig. 6. ECG: sinus rhythm, heart rate — 133–150 per 1 min, tachycardia, bioelectric systole slightly prolonged, LV overload, transmural myocardial ischemia

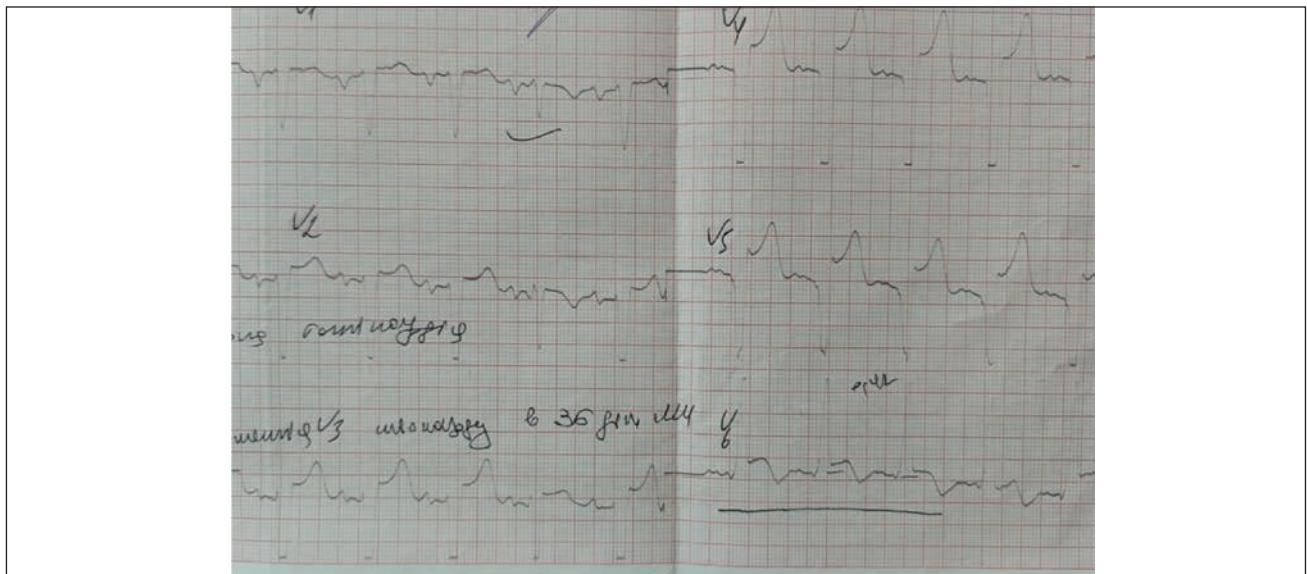


Fig. 7. ECG: sinus rhythm, heart rate — 133–150 per 1 min, tachycardia, bioelectric systole slightly prolonged, LV overload, transmural myocardial ischemia

133–150 per 1 min, tachycardia, bioelectric systole slightly prolonged, LV overload, transmural myocardial ischemia (Fig. 6, Fig. 7).

After 14 days the child was discharged. The condition after discharge remained serious. According to the telephone message, the child died 10 days after discharge from the hospital. The parents refused to perform post-mortem examination.

Discussion

Diagnosis of coronary syndrome in pediatric practice is difficult due to insufficient vigilance of medical staff due to low prevalence of the disease, often blurred clinical picture (children, especially at an early age, can not clearly locate and describe

pain; such pain equivalents as anxiety, moodiness, disturbed sleep, tachypnea or tachycardia, quite often clinically misinterpreted by health professionals), masking coronary heart disease with other inflammatory or non-inflammatory cardiomyopathies [4]. However, the problem of coronary insufficiency in pediatrics exists and requires clinical understanding, the choice of the correct diagnostic algorithm and qualified medical care [5]. At the same time, it should be understood that the risk of death from various causes or cardiovascular complications (recurrent myocardial infarction, cardiac death) is at least 30% higher in both early (1–3 years) and in long-term (3–5 years) period after myocardial infarction. Vigilance of medical

workers, careful monitoring allow to diagnose children's coronary insufficiency in time, to stop the progression of the disease and to prevent mortality [2].

Conclusions

Thus, coronary insufficiency is an important problem in pediatric cardiology and pediatrics despite its relative rarity in childhood. Primary care physicians should be alert to children with an unclear etiology of pain syndrome, a clinic of heart failure, and recurrent broncho-pulmonary diseases. It would also be optimal to have an annual EKG screening of

children before the start of the school year, which would make it possible to diagnose clinically «silent» forms of coronary insufficiency in a time.

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Declaration of financial and other relationships. The work did not have sponsorship. The authors are solely responsible for submitting the final version of the manuscript for publication. All authors participated in the development of the concept of the article and the writing of the manuscript. The final version of the manuscript is approved by all authors.

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