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Myocardial Infarction in A Teenager (Clinical Case)

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ABSTRACT

Myocardial infarction in childhood is rare, so identifying its cause requires a wide range of studies. The combination of pain syndrome with the characteristic clinical features of MI requires urgent hospitalization of a child of any age and an examination, including an assessment of repolarization on the ECG, Coagulography, markers of myocardial destruction, the functional state of the left ventricle using echocardiography and visualization of the coronary arteries. This approach will ensure maximum effectiveness of therapy and positive prognosis. We present the observation of a patient with an anginal attack and Myocardial infarction documented on an electrocardiogram.

Keywords: Myocardial infarction; Teenager; Pain syndrome; Anginal attack

INTRODUCTION

Myocardial Infarction (MI) in childhood is rare, so identifying its cause requires a wide range of studies. Analysis of each clinical case is important to prevent diagnostic errors, since most cardialgias in pediatric practice are of functional origin.[1]

CLINICAL CASE

We present the observation of a patient with an anginal attack and MI documented on an electrocardiogram (ECG). Boy V., aged 14, was admitted to the department of the Krasnoyarsk Regional Clinical Center for Maternal and Childhood Protection in order to verify the diagnosis and determine the tactics of management.

In March 2021, at night he felt an intense pressing pain in the region of the heart with irradiation to the left shoulder, an increase of temperature to 38^0C. During the day, the pain syndrome persisted with an increase at night, a rise in temperature to 37.6^0C, and vomiting three times. From the anamnesis: in January 2021, he suffered from COVID-19 in a mild form - anosmia without fever and catarrhal symptoms; 5 days before attack pain he suffered an injury (falling on his back) and received non-steroidal anti-inflammatory drugs, 2 days before – sever emotional stress. In the morning, the ECG revealed ST elevation in I, V2-V5 with the formation of a negative T wave in I, avL, V_3-V_6 (Figure S1). On the 3rd day of illness he was sent to the hospital of the nearest hospital.

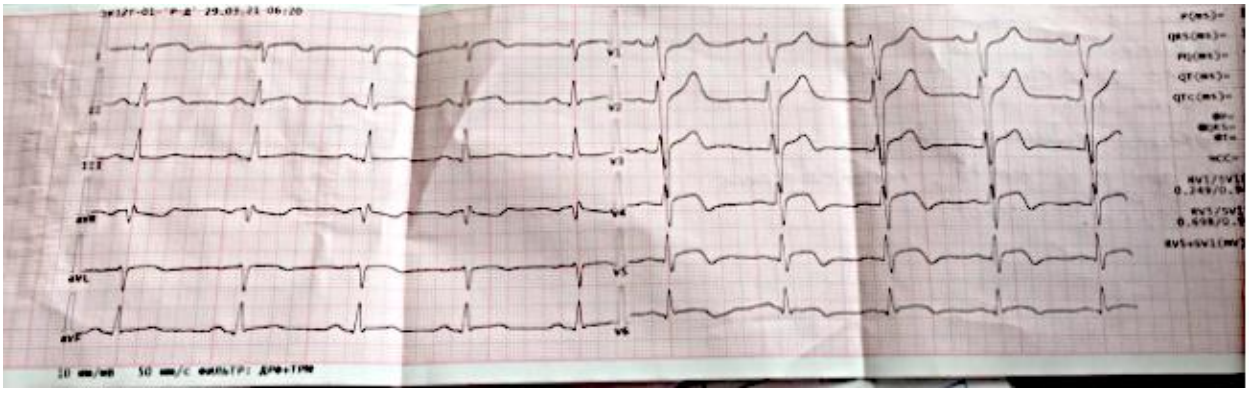

Figure S1: Myocardial infarction of the anterolateral wall of the left ventricle.

Upon admission, the patient's condition was hard: pain was pronounced, there was no shortness of breath, saturation was 99%, heart rate was 94-108 beats/min, blood pressure was 100/60 mm Hg. Therapy was prescribed: heparin 2500 IU, clopidogrel 150 mg orally, aspirin 200 mg orally, prednisolone 30 mg intravenously.

With suspicion of myocarditis, he was transferred to a level 2 hospital, where a positive troponin test was detected, C-reactive protein - 59 norms, Lactat Dehydrogenasa (LDH) 355 mmol/l (2 norms), Creatine Phosphokinase (CPK) 616 mmol/l (3 norms), creatine kinase-MB 55.3 mmol/l (2 norms), Aspartate Aminotransferase (AST) 106 mmol/l (3 norms), Alanine Aminotransferase (ALT) 34 mmol/l. D-dimer 315 (norm 250.0). According to echoCG: the heart cavities are not dilated, there are no hypokinesia zones, the valves are intact, the ejection fraction is 59%, the

Interventricular Septum (IVS) is thickened up in diastole to 10.0 mm. Within an hour, systemic thrombolysis was started - Actilyse 50 mg against the background of heparin therapy, metoprolol 25 mg × 2 times a day. Clinical state had positive dynamics - pain decreased, decreased to the norm ALT, AST, CPK, LDH.

On the 5th day of illness, he was transported to a hospital of the 3rd level. Pressing pains in the region of the heart, dizziness, heavy weakness persisted (moves in a wheelchair with the help of medical staff). Body type is normosthenic, skin is clean, respiration is vesicular, respiratory rate is 20 per minute. Rhythmic heart sounds, no murmurs, Heart Rate (HR) standing 80 beats/min, lying down - 62-68 beats/min. blood pressure (BP) 110/70 mm Hg. The liver and spleen are not enlarged.

The immunological panel parameters are negative, the level of class G antibodies to SARS-CoV-2 is 16.98, IgM is negative. Coronary angiography did not reveal anomalies in the number and development of coronary arteries, signs of coronary thrombosis (Figure S2).

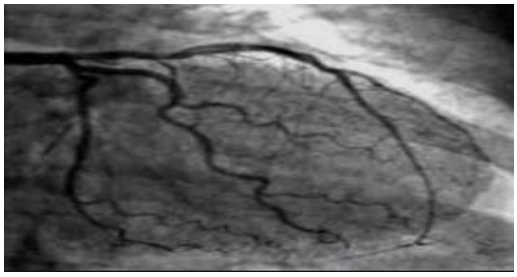

Figure S2: Normal coronary arteries.

A molecular genetic study has identified high-risk mutations C677T in the methylenetetrahydrofolate reductase gene, A2756G in the methionine synthase gene, and A66G in the methionine synthase reductase gene.
Taking into account the clinic of anginal attack and ECG, the diagnosis was made: Myocardial infarction without Q – septum, apex, side wall (with a transition to the back wall), acute phase.

On the background of therapy, the state of health improved, there are no pains, BP is 100-110/70 mm Hg. Art., HR 64-80 beats per minute. In the dynamics of the ECG - sinus rhythm with a HR of 59-83 beats in min., the electrical axis of the heart is deflected to the right, there was a blockade of the posterior branch of the left leg of the bundle of His. Complete restoration of the ST segment on the isoline. The child was discharged with clinical improvement.

DISCUSSION

The first landmark in the algorithm for diagnosing the cause of MI is the age of the patient: the maximum number of fatal outcomes has been described in newborns with anatomically normal hearts and coronary arteries. Myocardial ischemia is provoked by perinatal hypoxia, infections, vascular catheterization, erythrocytosis against the

background of feto-fetal transfusion syndrome, and thrombophilia.[2,3,4] In the group of heart defects, the basis of MI is anomalies of the origin of the left coronary artery (from the pulmonary artery).[5]

After 1st month of life, the causes of MI include Kawasaki disease,[6] acute myocarditis,[7] atherosclerosis in homozygous hypercholesterolemia,[8] and since 2020, Multisystem Inflammatory Syndrome (MIS) associated with COVID-19.[9] MIS is unlikely to be the cause of MI in this case.

Mutations in the folate cycle genes detected in a patient provide an increase in the level of homocysteine, which contributes to endothelial damage and the development of atherothrombosis in young patients.[10] We assumed that in this patient the pathology of the coagulation system became the background for the development of myocardial ischemia, Takotsubo syndrome was discussed as a possible causative factor (taking into account the influence of severe emotional stress 2-3 days before the development of the disease). This condition is accompanied by a violation of the geometry of the chamber of the left ventricle with an expansion of the middle part and hyperkinesia of the basal region, which forms a spasm of the coronary arteries.[11] This assumption is confirmed by an increase in D-dimer in the acute period and the effectiveness of thrombolytic therapy, thickening of the IVS.
A similar case of MI was described in a 15-year-old teenager with an anatomically normal heart and coronary arteries; pain and characteristic ECG changes appeared after a ball hit the chest.[12] The authors noted a rapid positive clinical dynamic and hypothesized that cause of MI was the transient coronary spasm.

CONCLUSION

The combination of pain syndrome with the characteristic clinical features of MI requires urgent hospitalization of a child of any age and an examination, including an assessment of repolarization on the ECG, coagulography, markers of myocardial destruction, the functional state of the left ventricle using echocardiography and visualization of the coronary arteries. This approach will ensure maximum effectiveness of therapy and positive prognosis.

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The authors of this article confirmed absence of financial support of this research to declare.

ETHICS STATEMENT

Written informed consent was obtained from the individual(s) for the publication of any potentially identifiable images or data included in this article.

AUTHOR CONTRIBUTIONS

All authors listed have made a substantial, direct and intellectual contribution to the work, and approved it for publication.

CONFLICT OF INTEREST STATEMENT

All the authors do not have any possible conflicts of interest

REFERENCES

1. Kundra M, Gundlach M, Nield LS, Kamat DM, Mahajan P, Kamat DM. Pediatric chest pain: keys to the diagnosis. Consultant for Pediatricians. 2006;5(8).
2. Burchert H, Lewandowski AJ. Preterm birth is a novel, independent risk factor for altered cardiac remodeling and early heart failure: is it time for a new cardiomyopathy?. Curr Treat Options Cardiovasc Med . 2019;21(2):8.
3. Poonai N, Kornecki A, Buffo I, Pepelassis D. Neonatal myocardial infarction secondary to umbilical venous catheterization: A case report and review of the literature. Paediatr Child Health. 2009;14(8):539-41.
4. Caruso E, Di Pino A, Poli D, Manuri L, Guccione P. Erythrocytosis and severe asphyxia: two different causes of neonatal myocardial infarction. Cardiol Young. 2014;24(1):178-181.
5. Naumenko EI, Grishutkina IA, Samoshkina ES. Difficulties in diagnosing congenital heart defects - abnormal origin of the left coronary artery. Russian Pediatric Journal. 2021;2(1):13-19.
6. Burns JC, El-Said H, Tremoulet AH, Friedman K, Gordon JB, Newburger JW. Management of myocardial infarction in children with giant coronary artery aneurysms after kawasaki disease. J Pediatr . 2020;221:230-234.
7. Vazemiller OA, Ponamareva NS, Chubko DM, Ushakov IYu, Makarevskaya NYu, Sharoglazov MM, et al. Difficult diagnosis: acute myocarditis or myocardial ischemia in an infant with thrombophilia? Clinical case. Vopr. modern pediatrics. 2017;16(1):54-58.
8. Leontieva IV. Familial homozygous hypercholesterolemia in children: a strategy for early diagnosis and treatment. Ros. vestn. perinatol. and Pediatrics. 2021;66(4):118-128.
9. Verdoni L, Mazza A, Gervasoni A, Martelli L, Ruggeri M, Ciuffreda M, et al. An outbreak of severe Kawasaki-like disease at the Italian epicenter of the SARS-CoV-2 epidemic: an observational cohort study. Lancet. 2020;395(10239):1771-1778.
10. Kolesnikova MA, Snigir OA, Strozenko LA, Lobanov YuF. Distribution of prothrombotic polymorphic gene variants in children with ischemic stroke. Medicine and education in Siberia. 2015;6:39.
11. Tyapkina MA, Yakovleva EV, Roshchina AA, Khalmetova GA, Basov IV. The development of Takotsubo syndrome in a young man. Archives of Internal Medicine. 2020;10(3):230-236.
12. Ivanov SN, Kukhtinova NV. A case of myocardial infarction in a fifteen-year-old adolescent without previous heart disease. Question. modern pediatrics. 2015;14(3):408-411.