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Dilated Cardiomyopathy in The Practice of a Pediatric Cardiologist

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Abstract: The thesis highlights literature data on the prevalence, possible causes, pathomorphology, and diagnosis of dilated cardiomyopathy in children and adolescents. A description of the clinical observation of dilated cardiomyopathy as an outcome of carditis of unspecified origin in a 3-year-old child is provided. Carrying out complex therapy for 8 weeks had a positive effect on the course of the disease.

Keywords: dilated cardiomyopathy, children, method, treatment.

Introduction

It is known that cardiomyopathies are a special group of heart diseases, which are based on primary predominant damage to the myocardium of unknown or unclear etiology ("primary chronic myocardial disease"), united according to certain clinical and anatomical features: the presence of cardiomegaly, cardiac failure, heart failure (HF), tendency to cardiac arrhythmias, thromboembolic complications and frequent fatal outcome in the form of sudden cardiac death [1]. Dilated cardiomyopathy (DCM) is characterized by significant cardiomegaly due to pronounced dilatation of the cavities of the heart, especially the left ventricle, and pronounced contractile failure of the myocardium, caused by a primary internal defect of damaged cardiomyocytes. This is accompanied by progressive chronic heart failure, often refractory to drug therapy, and a poor prognosis.

MAIN PART

Dilated cardiomyopathy is the most common form of cardiomyopathy in both adults and children.

Despite the unproven causative factors of DCM, the following are most often identified:

1. Hereditary predisposition - is proven by the high frequency of the presence of familial forms of the disease (familial cardiomyopathy), when DCM is diagnosed in blood relatives of probands who have clinical manifestations of the disease [1, 3]. Familial forms of the disease are detected in 20–34% of cases [4], and with targeted electrocardiographic and echocardiographic examination of practically healthy relatives, signs of the initial (preclinical) stage of DCM are found in 9–21% of cases [4]. The most common is the autosomal dominant mode of inheritance of DCM with a penetrance of about 60% [1]. Mutant genes are localized on chromosome 1-locus q 32 and on chromosome 9-q 13 q 22, but the exact identification of mutant genes has not been completed. It is assumed that the disease is associated with a congenital genetic defect of cardiac muscle tissue, a disorder of myocardial metabolism at the cellular level, a defect in the structure of mitochondria, deficiency of lactate dehydrogenase, and a disorder of myofilament synthesis [1, 2].

2. The role of viral infection in the occurrence of DCM is justified by cases of the development of the disease after a viral infection, the detection of viral ribonucleic acid (RNA) in the myocardium of patients with DCM and the creation of an experimental model of DCM as an outcome of viral myocarditis [1, 2, 5]. Clinical manifestations of a virus-like disease before the development of DCM

symptoms in patients are detected, according to various authors, with a frequency of 10 to 52% of cases [2]. In addition, 1–5 years after endomyocardial biopsy-confirmed viral myocarditis develops DCM in 8–52% of patients. In patients with DCM, in 42–56% of cases, antibodies to cardiotropic viruses are detected, especially to group B Coxsackie enteroviruses, as well as enteroviral RNA in the myocardium (50% of cases), which is also an indirect indicator of the connection between DCM and viral infection.

3. A prolonged toxic cardiodepressive effect of xenobiotics is assumed - metal compounds (copper, cadmium, cobalt, zinc, lead, etc.), lubricants, pesticides and other substances, as well as large doses of antitumor drugs (anthracycline antibiotics, cytotoxic drugs), ethyl alcohol and its metabolites, which have a damaging effect on the membranes and mitochondria of cardiomyocytes. This is accompanied by a violation of redox and energy processes in the myocardium, myocardial fibrosis, and a violation of contractile processes in the myocardium [1, 4].

4. Certain importance is attached to metabolic disorders associated with a deficiency in the body of amino acids, especially tryptophan, vitamin B1, selenium, taurine, carnitine, in which cardiomyopathy develops, resembling dilated cardiomyopathy.

5. In the pathogenesis of DCM, the role of autoimmune mechanisms triggered by cardiotropic viral infection in patients with a genetically determined tendency of the immune system to autoimmune reactions is assumed. This contributes to the development of viral myocarditis with a chronic course, cytolysis, myocardiofibrosis and myocardiosclerosis. Thus, myocarditis and DCM represent two successive stages of autoimmune myocardial disease.

6. Apoptosis, caused by the disinhibition of apoptosis genes under the influence of angiotensin II, b-agonists and other factors, can play a certain role in the progressive nature and programmed death of cardiomyocytes.

The diagnostic criteria for dilated cardiomyopathy are as follows:

1. History: presence of cardiomyopathies in the family and in close relatives, cases of sudden death or diseases accompanied by refractory congestive heart failure among relatives, especially at a young age; onset of clinical manifestations at 9–10 years of age, predominance of boys and adolescents.

2. Clinical data: manifestation of the disease with congestive (FC III–IV) HF of the left ventricular type, refractory to therapy, occurring in the absence of severity of acute-phase indicators of process activity; combination of congestive heart failure with severe cardiac arrhythmias and a tendency to thrombus formation (in the left cavities of the heart) and thromboembolic complications;

pronounced early expansion of the borders of the heart to the left upward, a three-part rhythm of the protodiastolic gallop and regurgitant noise of relative mitral or mitral-tricuspid insufficiency, increasing with increasing cardiac decompensation.

3. X-ray examination: signs of pronounced venous congestion in the pulmonary circulation, spherical, mitral or trapezoidal heart shape, cardiothoracic index more than 0.6–0.65.

4. Electrocardiography (ECG): moderate signs of left ventricular and left atrium hypertrophy, increased RV6/Rmax index > 3; blockade of the left bundle branch or its anterosuperior branch

in and; various heart rhythm disturbances, especially atrial fibrillation and ventricular arrhythmias.

5. Echocardiography (EchoCG): pronounced dilatation of the cavities of the heart, especially the left ventricle and left atrium, with a slight increase in the thickness of their walls; a significant increase in the end-diastolic volume of the left ventricle; pronounced hypokinesia of the posterior wall of the left ventricle and interventricular septum, significant (below 30–40%) decrease in ejection fraction.

CONCLUSION

Thus, in the above clinical observation, the development of dilated cardiomyopathy in a child was probably the result of carditis of unspecified origin. Dilated cardiomyopathy is indicated by: family history - a case of sudden cardiac death in a relative at a young age, clinical data - the course of the disease with heart failure in the absence of acute phase indicators of process activity, expansion of the borders of the heart to the left upward, systolic murmur relative mitral-tricuspid insufficiency, EchoCG data - dilatation of the left ventricular cavity, an increase in the end-diastolic volume of the left ventricle, a decrease in ejection fraction, severe mitral regurgitation, increased pressure in the pulmonary artery. After 8 weeks of complex treatment, positive dynamics in the course of the disease were noted: tachycardia and shortness of breath decreased, ejection fraction increased, end-diastolic volume of the left ventricle decreased, mitral insufficiency decreased with continued dilatation of the left ventricular cavity.

REFERENCES

1. Amosova E.N. Cardiomyopathies. — K.: Book Plus, 2019. — 421 p.
2. Belokon N.A., Kuberger M.B. Diseases of the heart and blood vessels in children. - M.: Medicine, 2017. - T. 1. - P. 436-437.
3. Yuldashevna, K. N. (2023). Features of Viral Conjunctivitis in Children. *Research Journal of Trauma and Disability Studies*, *2*(5), 17-26.
4. Udyeva, N. (2023). DIAGNOSIS AND TREATMENT OF ADENEVIRUS CONJUNCTIVITIS IN AMBULATORY CONDITIONS. *International Bulletin of Medical Sciences and Clinical Research*, *3*(5), 220-223.
5. Худдиева, Н. (2023). ДИАГНОСТИКА И ЛЕЧЕНИЯ АДЕНОВИРУСНОГО КОНЪЮНКТИВИТА В АМБУЛАТОРНЫХ УСЛОВИЯХ. *Евразийский журнал медицинских и естественных наук*, *3*(7), 23-27.
6. Худдиева, Н. Ю. (2023). Особенности Вирусного Конъюнктивита У Детей. *Research Journal of Trauma and Disability Studies*, *2*(5), 27-36.
7. Odilova, G., & Xuddiyeva, N. (2023). ADENOVIRUSLI OFTALMOINFEKSIYA NATIJASIDA RIVOJLANGAN «QURUQ KO ‘Z» SINDROMINING FUNKSIONAL DIAGNOSTIKASI. *Естественные науки в современном мире: теоретические и практические исследования*, *2*(7), 17-19.
8. Limarenko M.P., Smirnova T.V., Tkachenko Yu.Yu. A case of hypertrophic cardiomyopathy in a 16-year-old patient // Child’s Health. — 2018. — No. 3(12). — P. 96-99.
9. Якубов, И. Ю. (2023). МОДИФИКАЦИЯ ПОЛИТЕТРАФТОРЭТИЛЕНА СОПОЛИМЕРИЗАЦИЕЙ ТЕТРАФТОРЭТИЛЕНА В ПРИСУТСТВИИ НИЗКОТЕМПЕРАТУРНОГО ИНИЦИАТОРА. *IJODKOR O'QITUVCHI*, *3*(29), 367-374.
10. Якубов, И. (2023). СИНТЕЗ И ИССЛЕДОВАНИЕ СВОЙСТВ ПОЛИМЕРОВ И СОПОЛИМЕРОВ ТЕТРАФТОРЭТИЛЕНА С ГЕКСАФТОРПРОПИЛЕНОМ. *International Bulletin of Medical Sciences and Clinical Research*, *3*(6), 222-229.
11. Nagornaya N.V., Kartashova O.S. Dilated cardiomyopathy in children: modern approaches to diagnosis, treatment and prognosis // Child’s Health. — 2017. — No. 2(5). — P. 26-32.
12. BobonazarovGapporYadgorovich, B., Rahimovna, O. N., & RabimovaZilolaSHuhratqizi, R. Study on the Distribution of Bee Trachea Acarapis Woodi Ticks in Bee Yard of

Kashkadarya Region. *International Journal of Innovations in Engineering Research and Technology*, *8*(04), 27-30.

13. Babonazarov, G. Y., Omonova, N. R., Orziyeva, Y. M., & Khosilova, G. A. (2022). Economic Damage Caused by Scabies Itch Mite, Sarcoptes Scabiei (Acariformes: Sarcoptidae) to the Wool Production of Sheep. *Journal of Pharmaceutical Negative Results*, 2433-2436.
14. Moiseev V.S., Sumarokov A.V. Heart disease: A guide for doctors. - M.: Universum Publishing, 2011. - 463 p.