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TO THE ISSUE OF ACUTE MYOCARDITIS

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К ВОПРОСУ ОБ ОСТРОМ МИОКАРДИТЕ

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Abstract. According to statistics, pathology of the cardiovascular system can occur in 80% of patients who have undergone various acute viral infections, including influenza. All these manifestations are compensatory in nature and are completed independently without additional therapy. Children have respiratory viral infections, arising heart failure and various rhythm disturbances, quite often with a protracted course. Perhaps the emergence of an acute process, as well as the presence of viruses that can cause difficulties in terms of diagnosis and diagnosis, and other controversial issues. This attempt to highlight some controversial points. The detailed historical aspect of the formation of this nosological unit.

Аннотация. По статистике патология сердечно-сосудистой системы может встречаться у 80% пациентов, перенесших различные острые вирусные инфекции, включая грипп. Большинство этих проявлений носит компенсаторный характер и завершается самостоятельно без дополнительной терапии. Но есть дети, у которых на фоне респираторных вирусных инфекций возникает сердечная недостаточность и различные нарушения ритма, довольно часто с затяжным течением, а в некоторых случаях с риском летального исхода. Поражения миокарда возможно, как вследствие острого процесса, так и при персистенции вируса, в связи с чем возникают некоторые трудности в плане диагностики и постановки диагноза и др. спорные моменты. Авторами данного обзора предпринята попытка осветить некоторые спорные моменты. Детально рассматривается исторический аспект формирования данной нозологической единицы, а также ключевые вопросы этиопатогенеза данной патологии.

Keywords: myocarditis, children.

Ключевые слова: миокардит, дети.

One of the urgent problems of modern cardiology is diseases of the heart muscle, among which a special place belongs to myocarditis [1, p. 1890].

Myocarditis mainly affects young people and children. G. R. Somers (2015), in his study, noted that myocarditis was verified in 8.6–21% of cases, out of the total number of sudden deceased children [2, p. 143]. Myocardial damage can occur in the midst of an infectious disease, and during the recovery period. With high confidence, it can be argued that from 1 to 5% of all patients with respiratory viral infections as well as the flu may have signs of infectious myocarditis.

So, in one of the studies, which included a retrospective analysis of 1000 case histories of patients who died before the age of 10 months, inflammatory changes in the myocardium were identified in 9.78% of children [3, p. 224]. Mortality, depending on the severity of the underlying disease in children with myocarditis, varies from 0.3 to 26% [4, p. 1130].

It should be noted that persons with a history of acute myocarditis, for 12 years after the disease, have a significantly greater risk of death or surgical interventions associated with heart transplantation [5, p.49].

Today, the frequency of occurrence and prevalence of myocarditis in children, according to various researchers, varies widely. According to Belozerov (2014), the prevalence of myocarditis in the population is 10: 10,000 [5, p. 50]. Recent studies (2015) show that today the incidence and prevalence of myocarditis in children tend to decrease, most likely due to pathomorphosis and hypo diagnosis of the disease [6, p. 555]. This is due to the fact that myocarditis is characterized by a large variety of clinical symptoms (from asymptomatic and low-symptomatic, «erased» forms to severe diffuse myocarditis and cardiogenic shock), and the lack of generally accepted informative diagnostic criteria [7, p. 551].

The term ‘myocarditis’ first used I. F. Sobernheim in 1837 [3]. In 1887, the Russian doctor Abramov SS presented a description of a kind of heart disease, which was combined with severe heart failure, and was characterized by severe progressive course with a further fatal outcome. The macroscopic picture revealed by him reflected a complete dilation of the cavities, thrombotic masses inside the chambers, areas of thinned myocardium with areas of cardiosclerosis. The direct relationship of infection and the identified «finds» it has not been established. A. Fidler in 1899, during microscopic examination of preparations, determined inflammatory infiltration and isolated this disease into a separate form: idiopathic myocarditis. In subsequent studies conducted in the 20th century, it was shown that changes in this process usually correspond to viral myocardial damage, sometimes with the development of autoimmune reactions

The aetiology of myocarditis in children is extremely diverse, but most often myocarditis is caused by an infectious agent, in particular viruses. So in 6-8% of cases, myocarditis develops during or shortly after sporadic and epidemic viral infections [7, p. 555]. It was found that in acute viral infections, myocardial involvement in the pathological process occurs in 10% of cases [8, p. 906]. Coxsacks A and B enteroviruses, ECHO, rubella virus, adenovirus, herpes simplex virus, Epstein–Barr virus, cytomegalovirus, influenza virus, and others have a particular cardiotropic nature [9, p. 203] TORCH-complex. This is due to the cardiotropic nature of viruses and the imperfection of the immunological protection of newborns and young children predisposed to this disease [10, p. 304]. If a woman is infected, then during pregnancy there is a real possibility of transmission of the virus from the mother to the fetus either in utero or in the neonatal period [11, p. 1670]. Carriage of cytomegalovirus infection and herpes simplex virus can cause intrauterine myocarditis [12, p. 1698]. According to the results of some studies, myocarditis in infants was one of the manifestations of generalized IUI, wherein 50% of cases the infection was caused by cytomegalovirus or herpes simplex virus [13, p. 2616]. The fact is that the viruses of the herpes family have the whole set of properties allowing them to become the cause of chronic

cardiovascular pathology with alteration of the vascular endothelium, proliferation of smooth muscle cells, various immunopathological changes, including polyclonal humoral activation, morphological changes of cardiomyocytes [14, p. 500].

A common cause of myocarditis in infants is the Coxsackie B virus, which accounts for about 50% of cases [2, p. 143; 4, p. 1130]. Coxsackie viruses have an affinity for specific receptors located on the surface of the cardiomyocyte. It is important to emphasize that the infection caused by the Coxsackie virus is erased in half of the cases, which significantly complicates diagnosis [3, p. 221]. Among children who died of myocarditis, Coxsackie B-antigen is released in 41% of cases [4, p. 1131]. Often myocarditis occurs in infants with congenital heart defects, which themselves are accompanied by cardiomegaly and heart failure, while the identification of myocarditis causes difficulties, and their prognosis for patients is most serious [12, p. 1699]. Unfortunately, the aetiology of non-rheumatic myocarditis is diagnosed only in 25% of cases, while in 75% the aetiology is represented by an undifferentiated viral infection [13, p. 2616].

Several mechanisms play a role in the pathogenesis of viral myocarditis — the direct cytotoxic effect of the virus on cardiomyocytes (CMC), activation of apoptosis processes, reactions of the primary and secondary immune response, remodelling of the contractile apparatus of the heart muscle [6, p. 555] These processes take place in three successive stages with different clinical symptoms. In the initial phase of the disease, the virus enters the CMC, endothelial cells and fibroblasts by endocytosis. Myocardial damage in the initial stages of the disease can be realized by direct virus-mediated lysis of CMC or through activation of the primary immune response [7, p. 551]. In the case of fulminant forms of myocarditis, the massive death of CMC can lead to a serious violation of the contractile function of the heart and the rapid progression of heart failure. Macrophages and natural killers exacerbate the damage to the heart muscle, destroying infected CMC using perforins and granzymes, and also support active inflammation in the myocardium, producing pro-inflammatory cytokines [8, p. 905]. The initial phase of myocarditis in the case of an adequate immune response can end with the complete elimination of the virus from the myocardium with subsequent recovery, but it can go into the second phase - autoimmune [12, p. 1699].

The second phase of viral myocarditis begins, as a rule, 10–14 days after the virus enters the myocardium and is characterized by activation of secondary (specific immunity) reactions with the production of specific anti-myocardial immunoglobulins of classes G, M and A by plasma cells and proliferation of antigen-specific T-lymphocyte clones [3, p. 221]. Leukocyte chemotaxis is stimulated, which is accompanied by their migration to the inflammatory focus and adhesion to endotheliocytes, impaired microcirculation, and severe damage to the contractile apparatus of the heart. The main proinflammatory cytokines that are produced by immune cells in the inflammation in this phase of the disease are γ -interferon, α tumour necrosis factor (TNF- α), IL-1 β , IL-2, IL-6, IL-17A, IL-23 [7, p. 557]. The cytokine imbalance plays a great role in the further progression of the process and the formation of heart failure [10, p. 305]. In the case of a prolonged inflammatory process in the heart muscle, the disease transitions to a third, chronic phase, in which the main pathological process is remodelling of the heart muscle with progressive dilatation and the development of chronic heart failure [9, p. 203]. Signs of inflammation in the myocardium during the histological examination may not be detected, but profound structural and functional changes in the contractile apparatus of the heart with the development of fibrosis are usually irreversible. Subsequently, the transformation of the disease into dilated cardiomyopathy (DCMP) is possible [20]. In 20% of cases, acute myocarditis can turn into DCM as a result of the persistence of the viral genome and the constant maintenance of immune inflammation in CMC [2, p. 144].

Thus, at present, there is an urgent need to create clear criteria for the diagnosis of myocarditis in children, which would allow detecting the disease at early stages of its development,

using available laboratory and instrumental methods of research and without resorting to invasive technologies [1, p.1899].

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