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KAWASAKI-MULTISYSTEM INFLAMMATORY SYNDROME IN CHILDREN IN THE DELAYED PERIOD OF CORONAVIRUS INFECTION (COVID-19): MODERN STATE OF THE PROBLEM AND POSSIBLE NEW APPROACHES TO TREATMENT (PLASMAPHERESIS)

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COVID-19 infection usually occurs in children in a mild form, but some of them in a delayed period (one or several weeks after acute infection with COVID-19) may develop a severe inflammatory disease with clinical manifestations similar to toxic shock syndrome (Kawasaki disease), classified as multisystem inflammatory syndrome in children (MISC). It is possible that the syndrome has only a temporary connection with the COVID-19 infection. In the future, new associations of such clinical manifestations with other infectious (or non-infectious) diseases may appear. But currently, all children in the described cohorts with MISC have an association with COVID-19 infection. It is believed that the syndrome is initiated by an excessive adaptive immune response with the formation of autoantibodies. Treatment is based on anti-inflammatory, including steroid therapy, the possible use of intravenous immunoglobulin, aspirin, interleukin 1 and 6 receptor antagonists. The article analyzes current views on Kawasaki-multisystem inflammatory syndrome in children in the delayed period of COVID-19 coronavirus infection in the aspects of diagnosis, pathogenesis, clinical manifestations (with a discussion of foreign and Russian studies) and approaches to therapy and possible prevention, including the possibility of using plasmapheresis in complex therapy.

Keywords: children; COVID-19; Post-COVID-19 syndrome; Kawasaki disease; multi-systemic inflammatory syndrome; plasmapheresis; pathogenesis; diagnostics; treatment.

КАВАСАКИ-ПОДОБНЫЙ МУЛЬТИСИСТЕМНЫЙ ВОСПАЛИТЕЛЬНЫЙ СИНДРОМ У ДЕТЕЙ В ОТСРОЧЕННОМ ПЕРИОДЕ КОРОНАВИРУСНОЙ ИНФЕКЦИИ (COVID-19): СОВРЕМЕННОЕ СОСТОЯНИЕ ПРОБЛЕМЫ И ВОЗМОЖНЫЕ НОВЫЕ ПОДХОДЫ К ЛЕЧЕНИЮ (ПЛАЗМАФЕРЕЗ)

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Коронавирусная инфекция COVID-19 обычно протекает у детей в легкой форме, но у некоторых из них в отсроченном периоде (через одну или несколько недель после острой инфекции COVID-19) может развиваться тяжелое воспалительное заболевание, имеющее клинические проявления, схожие со слизисто-кожным лимфонодулярным синдромом (болезнью Кавасаки), классифицируемое как мультисистемный воспалительный синдром у детей. Возможно, синдром имеет только временную связь с инфекцией COVID-19. В будущем могут появиться новые ассоциации подобных клинических проявлений с другими инфекционными (или неинфекционными) заболеваниями. Но в настоящее время у всех детей в описываемых когортах с мультисистемным воспалительным синдромом имеется связь с инфекцией COVID-19. Считается, что синдром инициируется чрезмерным адаптивным иммунным ответом с формированием аутоантител. Лечение основано на противовоспалительной, в том числе стероидной терапии, возможном применении внутривенного иммуноглобулина, аспирина, антагонистов рецепторов интерлейкинов 1 и 6. В статье дан анализ современных взглядов на Кавасаки-подобный мультисистемный воспалительный синдром у детей в отсроченном периоде коронавирусной инфекции COVID-19 в аспектах диагноза, патогенеза, клинических проявлений (с обсуждением зарубежных и российских исследований) и подходов к терапии и возможной профилактике, в том числе к возможности применения в комплексной терапии плазмафереза.

Ключевые слова: дети; COVID-19; постковидный синдром; болезнь Кавасаки; мультисистемный воспалительный синдром; плазмаферез; патогенез; диагностика; лечение.

The COVID-19 pandemic has affected more than 100 million people worldwide, and more than 2 million of them have died. Since the beginning of the pandemic, it has been noted that children have a milder form of disease and better prognosis compared to adults [1, 18, 45]. The respiratory tract is the main target of COVID-19. It causes severe acute respiratory syndrome as well as damages the cardiovascular system, which is described as the most serious and life-threatening complication of this infection [18, 45].

Although it was initially reported that children have a mild form of COVID-19, by the summer of 2020, information regarding the severe course of this infection and its consequences in certain groups of children was published [27, 29, 39]. In the course of infection, they developed severe inflammatory disease with manifestations such as toxic shock syndrome or Kawasaki disease (KD) [29, 44, 53]. By mid-2020, more than 650 cases of monitoring pediatric patients with the abovementioned delayed symptoms after an acute phase of COVID-19 infection were published mainly in the European countries that were the most affected by the pandemic (England, Italy, France) as well as in the USA. By the end of 2020, a series of papers describing the frequency and nature of this disease course was published in the Russian Federation [2–4]. The prerequisites for the onset and severe course of this condition in children started to be registered. Despite the severity of the manifestations, mortality in the pediatric population remained

remarkable, but not high (approximately 1%). Unfavorable prognostic factors of the severe form of disease (requiring intensive therapy), which were noted in most of the publications, include age of >5 years and ferritinemia >1400 μg/L [39].

Patients with fever; systemic inflammation; and increased fatigue, pallor, shortness of breath, unstable blood pressure, hepatomegaly, and signs of gastrointestinal lesions (diarrhea, intestinal obstruction) must be treated with caution as these manifestations indicate the unfavorable course of the Kawasaki-like multisystem inflammatory syndrome in the delayed period after an acute phase of COVID-19. Increased serum ferritin and pro-brain natriuretic peptide levels, increased D-dimer levels in addition to hypoalbuminemia, thrombocytopenia, neutrophilic leukocytosis, lymphopenia, and a substantial increase in the markers of acute inflammation correspond to a cytokine storm [10, 27].

In some cases, this condition was so similar to the clinical manifestations of KD that differential diagnosis becomes highly complicated. Immediately, classification problems (whether to diagnose KD in the presence of its complete symptom complex in a child) as well as associated treatment problems arose, since KD therapy is regulated rather well.

KD causes systemic vasculitis with a polyclonal activation of B-lymphocytes and the production of autoantibodies to the cytoplasm of neutrophils and vascular endothelium. This disease (synonyms: mucocutaneous lymph node syndrome; nodule-like arteritis) was first described in Japan in 1967 and then in other countries. It represents an autoimmune, febrile, acute inflammatory disease that primarily affects young children. The disease leads to a state of immunodeficiency and inability of the immune system to counteract inflammatory pathogens. Clinical manifestations include fever, rash, mucosal lesions, conjunctival injection, pharyngeal erythema, adenopathy, and myocardial damage [5]. KD may cause macrophage activation syndrome, which is a condition wherein uncontrolled activation and proliferation of macrophages and other cell types occur and that may result in the dysfunction of various organs and systems [30]. The presence of seasonal waves of the disease, epidemiological clustering, and a very low risk of relapse suggests that infectious agents may be the main trigger for KD, although specific factors remain to be identified. There have been attempts to identify a specific microorganism, but they have not been successful yet [27]. The genetic characteristics of the host organism are probably involved in the pathophysiology of KD, which is confirmed by the excessive activation of the patient's innate immunity [12, 36].

During the period of COVID-19, viral infection was seen to aggravate the condition of patients with KD, but it was noted that children with COVID-19 were likely to develop a clinical condition like KD [43, 46, 47]. This course of COVID-19 infection is described in the literature as Kawa-COVID-19 [39], and this term is used to denote a systemic inflammatory disease associated with proven or highly suspected COVID-19 infection. These published data have led to an establishment of a new unique syndrome called **multisystem inflammatory syndrome** in children (MIS-C), which usually occurs several weeks after an acute infection of COVID-19 [6, 7], most often after 4–6 weeks [21]. The question of whether or not this syndrome in children will remain associated only with COVID-19 infection is not likely to be resolved in the future because new associations of this syndrome with other infectious (or noninfectious) diseases will appear. However, at present, most researchers are of the opinion that it is the COVID-19 infection that causes clinical manifestations similar to those of this syndrome in children and adolescents in its delayed period. The pathogenesis of this syndrome is clearly illustrated in Fig. 1. In pediatric patients, the early infection (phase I) period of COVID-19 may be asymptomatic or with mild symptoms. The pulmonary phase (phase II) is most severe in adults but mild or absent in many children. The early phase appears to trigger macrophage activation followed by helper T cell stimulation. This leads to the activation of inflammatory mediators (tumor necrosis factor, interleukins-12, -6, -1-beta, -23, -4), which promote the release of cytokines; stimulation of macrophages, neutrophils, and monocytes; and activation of B-cells and plasma cells that produce antibodies, leading to a hyperimmune response of the body in stage III. MIS occurs in the presence of a genetic predisposition. Clinical manifestations include pulmonary edema with atelectasis, meningeal manifestations, serous inflammation, heart ventricle dysfunction, and coronary aneurysm development as well as shock, acute renal failure, mesenteric lymph node inflammation, colitis, ileitis, ascites, skin changes, and gallbladder edema.

As a rule, the presence of a high titer of antibodies against COVID-19 is a characteristic feature of MIS-C. Moreover, the neutralizing ability of these antibodies, according to some authors, is not altered compared to the patients with COVID-19 without MIS-C [17], and according to others, it is

reduced due to their less specificity [57]. An increase in the concentration of inflammatory markers and the occurrence of a cytokine storm with a development of hypotension and shock (registered in 20%–100% of the patients) because of an acute myocardial dysfunction or a reaction of the systemic hyperinflammation and vasodilation are also characteristic features of MIS-C [51]. Dilatation of the coronary arteries and/or formation of aneurysms were described by some of the authors in 6%–24% of patients, and the occurrence of arrhythmias was registered in 7%–60% of cases [51]. The severity of lesions in the small blood vessels progressively worsens, suggesting that COVID-19-induced endotheliosis represents small vessel vasculitis that does not affect the main coronary arteries. The resulting inflammatory neuropathy of epicardial nerves in COVID-19 suggests a similar pathogenesis of vascular and nerve damage in this disease [32]. In the description of an autopsy wherein histological examination of a child who died due to MIS-C in the presence of COVID-19 infection was performed, it was noted that not only small but also medium-sized blood vessels are involved in the process [3]. It could be assumed that the severity of the course of this syndrome increases as bloods vessels with larger diameters are involved in the pathological process.

The fact that the survivors of Kawa-COVID-19 may be at a risk of developing permanent residual myocardial damage is particularly unfavorable, as incomplete recovery is considered to be the result of persistent inflammation of the heart muscle due to a virus-induced autoimmune response, which may extend far beyond the time frame of the disease, having a protracted course [48].

Unlike classic KD, which affects young children, systemic inflammation after COVID-19 infection is more often seen in older children and adolescents. A second interesting aspect is the fact that patients with severe MIS-C associated with COVID-19 are less likely to be Caucasians than the expected occurrence of Caucasians in the general population. A significant majority of patients with a severe course of the syndrome in the USA are African Americans and people of Latin American or Afro-Caribbean descent (in total, they make up 84% of patients with MIS-C) [23]. A second such noteworthy fact is that no case of MIS-C associated with COVID-19 have been reported in Korea and Japan, although these populations have the highest incidence of KD, and the COVID-19 pandemic has been registered in these regions; therefore, a genetic predisposition to the severe forms of the disease is possible [37]. A minor prevalence of affected males is also reported (up to 60%–66%). The question of the nature of heart damage seen in patients with MIS-C associated with COVID-19 is still under debate as some authors point out the development of isolated myocarditis without coronaritis and the formation of aneurysms [37], whereas others note the presence of coronaritis [51], which is consistent with Russian studies describing cohorts of pediatric patients with MIS-C associated with COVID-19 (n = 32) in whom signs of coronaritis and formation of aneurysms were noted (up to 16% of children) [2].

It is worth noting that the evidence of the onset of late inflammatory complications in the heart after COVID-19 infection in adult patients is also available. Among them, 58% of patients had abnormal computed tomography results (e.g., the presence of myocardial edema); impaired release of gadolinium; and decreased functional parameters of the myocardium, such as decreased ejection fraction, cardiac index, and systolic output index [19]. In addition, it is noteworthy that adult patients often have gastrointestinal symptoms that are also common in children, and in the pediatric population, the complete clinical presentation of KD can be described [50].

Magnetic resonance imaging of the heart in pediatric patients demonstrates diffuse myocardial edema without the signs of replacing fibrosis or focal necrosis of the heart muscle. Acute myocarditis occurs less than 1 week after the onset of fever and gastrointestinal symptoms. These data support the indication of postinfectious myocarditis in children and adolescents with COVID-19 [8].

With respect to the clinical presentation, all pediatric patients with Kawa-COVID-19 presented with fever or chills, 97% of them had tachycardia, 80% had symptoms of gastrointestinal tract damage, 60% had a rash, 56% had signs of conjunctivitis, and 27% had mucosal changes. Increased levels of C-reactive protein, D-dimer, and troponin were found in 100%, 91%, and 71% of the patients, respectively. Furthermore, 62% of the patients received vasopressor therapy, 53% of the patients had signs of myocarditis, and 80% of the patients were hospitalized in the intensive care unit [11]. All patients examined had an increase in cardiac inflammatory markers (C-reactive protein, ferritin, troponin I, creatine kinase, and pro-brain natriuretic peptide). Transient heart valve failure was registered in 67% of the patients. Left ventricular ejection fraction was reduced in 80% of the patients, and fractional shortening was noted in 53% of the cases. Coronary artery abnormalities were revealed

in 93% of the children. Pathological changes on the electrocardiogram were detected in 60% of the patients [42]. All children received an inotropic support (adrenaline, milrinone, dobutamine, norepinephrine, etc.) [14, 16, 58]. Similar findings were demonstrated in a cohort study of 16 pediatric patients in France [39].

In most cases, MIS-C developed 2–4 or 2–6 weeks after the acute phase of COVID-19 [9, 15, 21]. This new postviral systemic inflammatory disease supposedly arises from an excessive adaptive immune response of the body [26]. In this regard, the researchers call for alertness of the clinicians regarding the cytokine release syndrome associated with the COVID-19 [56]. The revealed relationship between MIS-C and COVID-19 infection suggests that the pathogenesis of MIS-C proceeds according to the postinfectious immune dysregulation type [37].

Children become infected with COVID-19 as often as adults, but in them, the disease is mostly asymptomatic or has a milder course, possibly due to the peculiarities of the immune response of the child's body [39, 45]. Although children are mostly spared from severe respiratory injury (at least it occurs much less frequently in children than in adults), they may develop MIS-C associated with COVID-19, with a disease course similar to that of KD [20]. The inflammatory response in MIS-C differs from the classic cytokine storm observed in severe acute COVID-19 cases primarily in the fact that, unlike it, it affects the respiratory tract much less frequently. Having common signs with KD, it is distinguished at the same time by peculiarities of the response of T cell subpopulations, interleukins, and biomarkers associated with vascular damage. The formation of autoantibodies plays a significant role in the pathogenesis of MIS-C [9] (Fig. 1).

In terms of treatment, most publications report the use of steroids only in patients with severe clinical manifestations of the disease or lack of response to primary intravenous administration of immunoglobulin, and only in a small number of studies, steroids were prescribed to all the patients (14%) [13]. In the treatment of children with Kawa-COVID-19, high doses of intravenous immunoglobulin (2 g per kg of the body weight) are used. A repeated course of intravenous immunoglobulin is possible. Most researchers agree that glucocorticosteroids and intravenous immunoglobulin are the first line of therapy for MIS-C associated with COVID-19 infection in children. In some cases, for respiratory distress, an interleukin-1 receptor antagonist (anakinra) [39] could be used, which is also used in KD [25], and in severe cases, the use of an interleukin-6 receptor antagonist (tocilizumab) has been reported [4, 39]. The use of aspirin in the treatment of children with MIS-C associated with COVID-19 infection, which is widely used by patients with KD, is rarely described in the literature; however, the fact that it has a quick positive effect in some cases [39, 50] may be a basis for further research in this direction in both pediatric and adult populations.

In patients with true KD, especially if treatment with steroids and immunoglobulin is ineffective, plasmapheresis could be used [28, 38]. Moreover, in some cases wherein large doses of immunoglobulins are administered (such doses are used in the treatment of MIS-C), there are risks of acute hemolysis [31].

Currently, plasmapheresis is a widely recognized method for the treatment of diseases such as myasthenia gravis, Guillain–Barre syndrome, and thrombotic microangiopathy. It is also actively used in patients with kidney disease. Pathological factors that could be eliminated by plasmapheresis include autoantibodies, complement products, lipoproteins, immune complexes, cryoglobulin, myeloma protein, protein-bound toxins, cell platelets, and leukocytes [22].

A similar treatment approach could be used in MIS-C cases, especially given the significant manifestations of endotoxemia accompanying the severe course of COVID-19, which may require the use of extracorporeal detoxification methods, mainly plasmapheresis [52, 54]. On the other hand, the autoimmune nature of MIS-C also creates a pathogenetic basis for the use of plasmapheresis to eliminate the antibodies and other large molecular toxic metabolites that cannot be removed by the kidneys [24, 35, 54]. Using extracorporeal detoxification during the acute course of COVID-19 infection in severely predisposed patients even before the development of MIS-C or at the very beginning of the syndrome manifestation, one could possibly prevent the critical conditions and long-term complications associated with the disease [33, 40, 49, 55]. This method of treatment, which could be used in an outpatient basis, can also be used in patients with MIS-C associated with a previous infection with COVID-19. The author's photo clearly demonstrates the ease and accessibility of plasmapheresis performed for two siblings with MIS associated with COVID-19 during an outpatient visit to the gravitational blood surgery department of the First Pavlov Saint Petersburg State Medical University (Fig. 2).

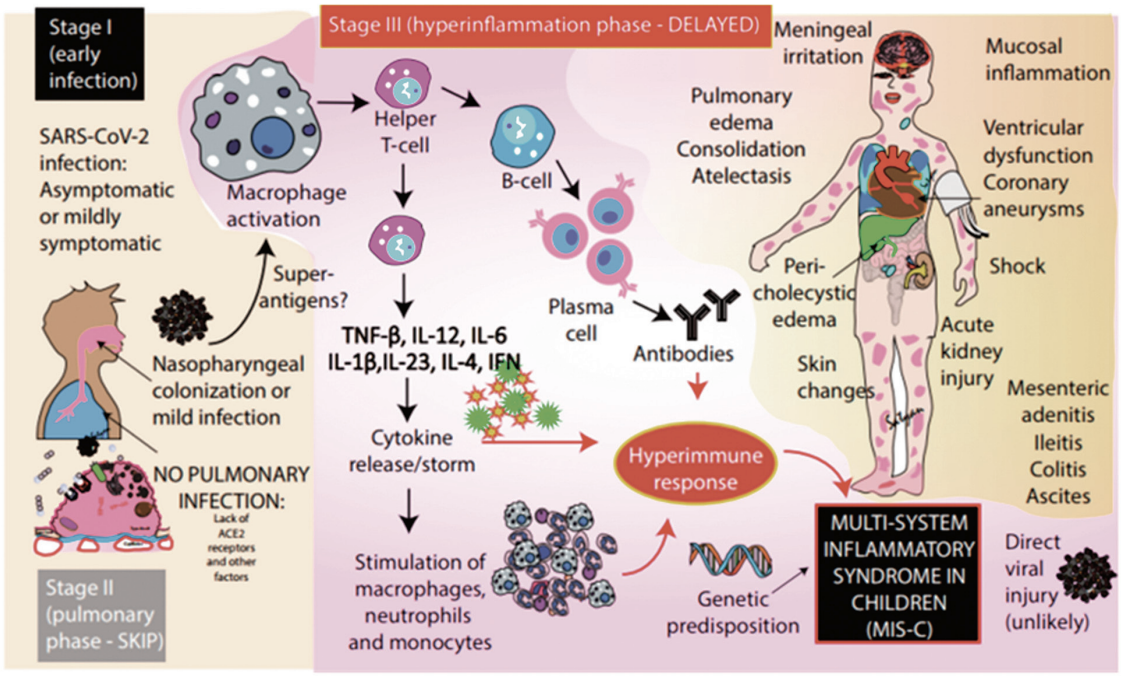

Fig. 1. Pathogenesis of multisystem inflammatory syndrome in children [9]. ACE2 – receptors for angiotensin-converting enzyme 2; TNF-β – tumor necrosis factor β; IL – interleukins

Рис. 1. Патогенез мультисистемного воспалительного синдрома у детей [9]. ACE2 — рецепторы ангиотензинпревращающего фермента 2; TNF-β — фактор некроза опухоли β; IL — интерлейкины

Fig. 2. Outpatient plasmapheresis procedure for children with multisystem inflammatory syndrome

Рис. 2. Проведение амбулаторной процедуры плазмафереза детям с мультисистемным воспалительным синдромом

In conclusion, it could be noted that although children usually present a mild form of COVID-19, some of them, in the delayed period (several weeks after an acute infection of the COVID-19), may develop severe inflammatory disease that has clinical manifestations similar to the toxic shock syndrome, KD, currently classified as MIS-C. The syndrome could only have a temporary relationship with COVID-19 infection. In the future, new associations of similar clinical manifestations with the other infectious (or noninfectious) diseases may appear. But at present, most children in the described cohorts with MIS-C are associated with COVID-19 infection [39, 51]. However, despite the revealed prominent similarities between MIS-C and KD, these diseases are different. Active attempts are being made to further differentiate them clinically as well as serologically, although based on the materials used in the review of the publications, it cannot be said that these attempts are completely successful. The similarity of the two diseases has prompted attempts to use the full range of the drugs used in the counteraction against KD in the treatment of children and adults with MIS-C.

It is believed that an excessive adaptive immune response with the formation of autoantibodies underlies the pathogenesis of MIS-C [41]. Treatment is based on the use of anti-inflammatory therapy, including glucocorticosteroid and aspirin administrations; prescription of high doses of intravenous immunoglobulin; and possible use of interleukin-1 and -6 receptor antagonists; however, endotoxicosis and the autoimmune nature of the disease create a pathogenetic prerequisite for the possible use of plasmapheresis in a complex therapy, which was proven to be beneficial according to the publications analyzed in the review [34].

ADDITIONAL INFORMATION

Author contributions. All the authors confirm the compliance of their authorship with the international ICMJE criteria (all authors have made a significant contribution in the development of the concept, research, and preparation of the article and have read and approved the final version of the manuscript before its publication).

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