SIR Publishers
CENTER FOR INTERNATIONAL SCIENTIFIC PUBLICATIONS
JOURNAL OF CHILD PSYCHOLOGY AND PSYCHIATRY
ISSN(Online): 2984-7958
SJIF Impact Factor |(2024): 6.833|
Volume-7, Issue-5, Published |20-05-2024|

MYOCARDIAL INJURY IN CHILDREN WITH COVID-19

https://doi.org/10.5281/zenodo.11407325

M.T.Yusupova, I.T.Rasuova, M.Xayrullayeva

ABSTRACT

Two major outbreaks associated with coronaviruses were reported in 2003 and 2012, respectively [1]. In December 2019, an epidemic of atypical pneumonia was observed in Wuhan, China. The causative agent of this outbreak was a novel coronavirus called severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). The World Health Organization (WHO) later named the disease associated with this virus as coronavirus disease-19 (COVID-19). In March 2020, COVID-19 was declared a global pandemic [2].

Keywords

COVID-19; myocardium; interleukin; heart; children; Cardiomyopathy.

The purpose of the study: analytical review of modern scientific literature sources covering the issues of prevention of complications of myocardial injury in children infected with COVID-19.

Materials and methods. An analysis of 25 foreign literature sources was conducted on this topic.

Charge: SARS-CoV-2 is a positively charged single-stranded RNA virus. This creates a wide spectrum of diseases affecting many organs. By early May 2020, the virus had reached more than 200 countries worldwide and killed at least 4 million people. As of February 10, 2021, there have been more than 106 million confirmed cases of COVID-19 and more than 2 million deaths worldwide [3].

The virus can directly invade various cells of the human body, including lung, heart and intestinal cells. Subsequently, tissues are damaged by direct viral invasion, cytokines, and indirect effects of inflammatory agents. A strong inflammatory reaction accompanied by a cytokine storm is the main factor causing the development of the disease in various organs. Initially, most patients have fever, dry cough and shortness of breath. As the disease progresses, patients may develop nausea, vomiting, abdominal pain, and diarrhea [4]. About 10% of patients develop severe disease and require intensive care and supplemental oxygen therapy. These patients are at high risk of acute respiratory distress syndrome with diffuse alveolar damage, heart damage, viremia, bacterial infections, and multiorgan failure [5].

Pathophysiology of COVID-19

SIR Publishers
CENTER FOR INTERNATIONAL SCIENTIFIC PUBLICATIONS
JOURNAL OF CHILD PSYCHOLOGY AND PSYCHIATRY
ISSN(Online): 2984-7958
SJIF Impact Factor |(2024): 6.833|
Volume-7, Issue-5, Published |20-05-2024|

The pathophysiology of COVID-19 occurs in three stages. The first stage begins with the penetration of the virus into the epithelium of the respiratory organs, and then cell proliferation begins. The initial immune response is characterized by the activation of monocytes and macrophages and is characterized by mild symptoms. At the next stage, pulmonary vasodilatation and an increase in vascular permeability are observed. Leukocyte migration then leads to fluid extravasation and pulmonary edema. As a result, alveolar damage, hypoxia, cardiac damage and stress occur. The final stage is characterized by a strong inflammatory reaction followed by a large second-order cytokine storm [6].

A state of hyperinflammation

Excessive release of pro-inflammatory cytokines leads to a dramatic increase in immunity against the virus. In fact, plasma levels of interleukin-2 (IL2), interleukin-6 (IL-6), tumor necrosis factor α (TNF α), and C-reactive protein (CRP) are significantly increased in patients with COVID-19. They are the main causes of multi-organ failure [7]. The innate immune system plays a crucial role in the uncontrolled inflammatory response mounted against the virus.

SARS-CoV-2 and the pediatric population

The Chinese Center for Disease Control reports that less than 1% of COVID-19 patients are children under the age of 10. Other major authors in China have reported a milder course of COVID-19 in a group of children, with most having mild symptoms. However, infants infected with COVID-19 had a more severe illness than older children [8]. The reasons for the mild manifestation of the disease in children are not yet clear. Some theories support the idea that children have less functional AAF2 receptors than adults; Another theory is that AAF2 receptors are abundant in children, which in turn lowers plasma levels of Ang II. Ang II is recognized to be strongly associated with the clinical course of COVID-19. Indeed, the hospitalization rate of children with COVID-19 is low and the mortality rate is less than 5% [9]. However, a minority of children are prone to serious manifestations of the disease, especially cardiovascular damage.

SARS-CoV-2 infects the heart

In the case of COVID-19, there are three mechanisms that cause cardiac damage: (1) direct injury caused by direct entry of the virus into myocardial cells, (2) hypoxia caused by myocardial ischemia and (3) increased inflammatory response to endothelial damage and microvascular thrombi [10]. The virus has a direct effect on myocardial cells, directly binding to AAF2 receptors present on these cells, causing direct myocardial damage [11]. The role of AAF2 and angiotensin in the heart is well studied. Their interaction inhibits oxidative stress and cardiac regeneration, while inducing coronary vasorelaxation [12]. AAF2 expression is increased in early stages of myocardial injury. In COVID-19, AAF2 is

SIR Publishers
CENTER FOR INTERNATIONAL SCIENTIFIC PUBLICATIONS
JOURNAL OF CHILD PSYCHOLOGY AND PSYCHIATRY
ISSN(Online): 2984-7958
SJIF Impact Factor |(2024): 6.833|
Volume-7, Issue-5, Published |20-05-2024|

significantly reduced and the protective effect of angiotensin is lost and myocardial injury is exacerbated. Interestingly, AAF2 expression has been found to be low in the elderly, which may partially explain the severe course of the disease in this age group [13]. In addition, many people say that patients with cardiovascular diseases are at high risk of contracting SARS-CoV-2 and having a severe disease. The mortality rate in such patients also increased significantly [14]. It is associated with ischemic damage of the cardiovascular system caused by a viral infection.

Similarly, electrocardiographic abnormalities are associated with inflammatory markers and cardiac damage [15]. Increased inflammatory markers are accompanied by elevated cardiac biomarkers and are associated with cardiac damage. Successive severe clinical presentation, morbidity and mortality rates increase [16]. In a retrospective study conducted in China earlier this year, the authors found that elevated cardiac biomarkers were associated with increased mortality from COVID-19 [17]. Many studies have shown that patients with cardiovascular diseases such as hypertension and coronary artery disease have a higher risk of contracting severe forms of COVID-19 and a greater risk of death. In a recent meta-analysis, Lee et al reported that the relative risk of severe COVID-19 and requiring intensive care in patients with hypertension and coronary artery disease was 2.03 (95% CI 1.54–2.68), respectively. ,) and 3.3 (95% CI 2.03–5.36,) [18].

Type I: acute myocardial injury

Type 1 myocardial damage is determined by a change in the electrocardiogram associated with myocardial ischemia or, in its absence, an acute increase in the level of cardiac troponin. The presence of acute myocardial injury in patients with COVID-19 causes a 3.4-fold increase in the risk of death. Similarly, acute myocardial injury occurs in one-fifth of hospitalized patients with COVID-19. In addition, myocardial damage causes a high need for mechanical ventilation.

Myocardial infarction, acute myocardial injury, occurs as a result of migration of atherosclerotic plaque in people with atherosclerosis. Viral products bind to immune receptors in the cells located inside the platelet, activating them and making the platelet prone to movement. These infection and inflammatory products cause coronary endothelial dysfunction and thrombosis [19].

Type II myocardial damage

Type II MI results from an imbalance in myocardial oxygen demand and supply. In the setting of COVID-19, type II MI can be caused by several factors: (1) the presence of an atherosclerotic plaque that leads to a gradual decrease in blood flow, (2) endothelial dysfunction, (3) increased AT levels. (4) high blood pressure and vascular hypoxia (5) [20]. In situations of severe stress and increased inflammation, SARS-CoV-2 infection can cause myocardial ischemia and infarction by disrupting the balance between myocardial oxygen supply and demand. This

SIR Publishers
CENTER FOR INTERNATIONAL SCIENTIFIC PUBLICATIONS
JOURNAL OF CHILD PSYCHOLOGY AND PSYCHIATRY
ISSN(Online): 2984-7958
SJIF Impact Factor |(2024): 6.833|
Volume-7, Issue-5, Published |20-05-2024|

imbalance may or may not be influenced by the atherosclerotic plaque due to the greater physiological stress caused by the infection.

The incidence of myocardial ischemia in the general COVID-19 population ranges from 7% to 40%.

Heart failure and myocardial damage account for approximately 40% of deaths in SARS-CoV-2-positive patients. In this case, the risk of death is significantly higher than the death rate associated with old age and pre-existing diseases.

Myocardial damage and elevated troponin have been reported in 12% of adult SARS-CoV-2 patients [21]. In another large study of only pediatric patients, at least 73 percent of enrolled patients had elevated cardiac biomarkers. In 50% of them, only troponin was elevated, most of them increased the level of Pro-BNP above 400 pg / ml.

In addition to increased troponin levels, an increased risk of death was associated with a worse clinical outcome (higher need for mechanical ventilation) as well as higher levels of other biomarkers such as IL-6, CRP, and D-dimer. Patients with myocardial injury and elevated troponin levels are more common than patients without myocardial injury. This phenomenon has not yet been reported in the pediatric population. The most common echocardiographic change in patients with any element of myocardial injury is right ventricular enlargement and dysfunction. Left ventricular dysfunction is less common in the literature. Focal and diffuse ST-segment elevation electrocardiography is also common.

Cardiomyopathy

Severe systemic inflammation in COVID-19 induces cardiotoxicity and leads to cardiovascular dysfunction. In vitro studies have shown that exposure to high levels of IL-6 and TNF-α resulted in left ventricular systolic dysfunction and reduced contractility [22]. In another study, the incidence of cardiomyopathy in patients infected with the virus was found to be as high as 33%. Tricuspid, aortic, or mitral regurgitation, leading to hemodynamic changes, has been observed in 10% of patients hospitalized for COVID-19. In addition, various studies have reported cardiac dysfunction in 41.2% of patients hospitalized for COVID-19. Although it is less common in the pediatric population, in several studies the authors have identified the presence of left ventricular dysfunction in patients under 20 years of age.

Multisystem inflammatory syndrome in children (BKTYS)

In April 2020, the National Health Services in Great Britain reported the first case of BKTYS. At that time, it was a new combination of atypical Kawasaki disease and toxic shock syndrome under severe COVID-19. Subsequently, WHO and the United States Center for Disease Control and Prevention established diagnostic

SIR Publishers
CENTER FOR INTERNATIONAL SCIENTIFIC PUBLICATIONS
JOURNAL OF CHILD PSYCHOLOGY AND PSYCHIATRY
ISSN(Online): 2984-7958
SJIF Impact Factor |(2024): 6.833|
Volume-7, Issue-5, Published |20-05-2024|

criteria with similar characteristics [23]. The first is characterized by acute necrotic arteritis characterized by infiltration of neutrophils into the vessel walls. After this, an aneurysm is formed inside the coronary artery. Macrophages and T-cell lymphocytes infiltrate the damaged vessel wall to initiate the chronic form of vasculitis. Proliferation of myofibroblasts over the years leads to coronary artery stenosis. In the initial acute phase, myocardial edema may develop, which leads to myocarditis before the symptoms of aneurysm. Transient left ventricular dysfunction occurs and leads to cardiovascular shock in some patients.

During SARS-CoV-2 infection, patients who develop BKTYS are usually more than patients who develop Kawasaki disease, and the average age of the former is 9 years. Symptoms begin about 4-6 weeks after exposure to the virus and usually have a negative PCR test. They usually have a variety of respiratory symptoms, ranging from fever and cough to shortness of breath. Gastrointestinal symptoms such as abdominal pain and diarrhea are also present in 70% of patients with BKTYS. Common cardiac abnormalities in BKTYS are arrhythmias, conduction abnormalities, ventricular dysfunction, dilatation or aneurysm of the coronary arteries, and pericarditis. According to most reports, the most common cardiac abnormality is LV dysfunction.

Patients with BKTYS may develop coronary artery abnormalities such as dilatation and aneurysm. Reports have shown a range of variations from small aneurysms to large aneurysms. Although a mechanism similar to that observed in Kawasaki disease has been hypothesized to occur with BKTYS, the actual pathophysiology behind the coronary artery abnormalities has not yet been elucidated [24].

In addition to coronary artery anomalies, the second most common cardiac anomaly in BKTYS is arrhythmia. First-degree heart block is the most common form of arrhythmia. Usually reported in children with LV dysfunction. Electrocardiogram shows QT prolongation, ST segment changes or T-wave abnormalities [25]. Laboratory examination reveals an increase in inflammatory markers such as troponin, natriuretic peptide (Pro-BPN), D-dimer and ferritin, erythrocyte sedimentation rate (EChT), interleukin-6 (IL-6), procalcitonin and C-reactive . An extended general blood analysis reveals neutrophilia, lymphopenia and thrombocytopenia. Chest X-ray shows cardiomegaly, mediastinal abnormalities. More than 50% of these patients have left ventricular dysfunction or heart failure documented on echocardiography. T1- and T2-weighted cardiac magnetic resonance imaging reveals diffuse increased intensity consistent with myocardial edema. The natural course of BKTYS is not yet known. Ventricular dysfunction improves over time. More studies are needed to determine the long-term effects of BKTYS.

SIR Publishers
CENTER FOR INTERNATIONAL SCIENTIFIC PUBLICATIONS
JOURNAL OF CHILD PSYCHOLOGY AND PSYCHIATRY
ISSN(Online): 2984-7958
SJIF Impact Factor |(2024): 6.833|
Volume-7, Issue-5, Published |20-05-2024|

Summary

Cardiovascular disease is important for patients diagnosed with COVID-19. Multidisciplinary teams with cardiology services are essential to prevent complications in patients with COVID-19, as well as in patients with underlying heart disease who are predisposed to severe COVID-19. We emphasize that devoting adequate resources to timely diagnosis and treatment of cardiovascular complications is critical to reducing morbidity and mortality from cardiovascular disease. In addition, it is important to perform a complete evaluation with an electrocardiogram, echocardiography, and a basic cardiac examination for timely diagnosis and treatment.

FOYDALANILGAN ADABIYOTLAR

1. S. Perlman, "Yana o'n yil, yana bir koronavirus", *New England Journal of Medicine* , jild. 382, yo'q. 8, 760–762-betlar, 2020 yil.
2. JSST, *Jahon sog'liqni saqlash tashkiloti Koronavirus kasalligi 2019 (COVID-19): Vaziyat haqida hisobot-113* , JSST, Jeneva, Shveytsariya, 2020 yil.
3. NK Yunis, RO Zarif, SN Al Hassan, F. Bitar, AH Eid va M. Arabi, “COVID-19 bemorlarida gidroksiklorokin: ijobiy va salbiy tomonlari” , *Farmakologiyada chegaralar* , jild. 11-modda, ID 597985, 2020 yil.
4. Xalilov, Hikmatulla, et al. "TELEMEDITSINANING PROFILAKTIK DAVOLANISHDA AHAMIYATI." *Евразийский журнал академических исследований* 4.4 Part 2 (2024): 66-70.
5. C. Huang, Y. Vang, X. Li va boshqalar, "Xitoyning Wuxan shahrida 2019-yilda yangi koronavirus bilan kasallangan bemorlarning klinik xususiyatlari," *The Lancet* , jild. 395, yo'q. 10223, 497–506-betlar, 2020 yil.
6. Z. Vu va JM MakGugan, "Xitoyda koronavirus kasalligi 2019 (COVID-19) avj olishining xususiyatlari va muhim saboqlari", *JAMA* , jild. 323, yo'q. 13, 1239–1242-betlar, 2020 yil.
7. . Dilshodovich, Khalilov Hikmatulla, Kayimov Mirzohid Normurotovich, and Esanov Alisher Akromovich. "RELATIONSHIP BETWEEN THYROID DISEASE AND TYPE 2 DIABETES." (2023).
8. XM Yuanyuan Dong, Y. Xu, X. Qi, F. Jiang, Z. Jiang va S. Tong, "Xitoydagi bolalar orasida COVID-19 epidemiologiyasi", *Pediatriya* , jild. 145, yo'q. 6, 2020 yil.
9. Y.-H. Loke, CI Berul va AS Harahsheh, "Bolalarda ko'p tizimli yallig'lanish sindromi: Kawasaki kasalligi bilan bog'liqlik bormi?" *Yurak-qon tomir tibbiyotidagi tendentsiyalar* , jild. 30, yo'q. 7, 389–396-betlar, 2020 yil.

10. G. Giustino, SP Pinney, A. Lala va boshqalar, "Koronavirus va yurak-qon tomir kasalliklari, miokard shikastlanishi va aritmiya" , *Amerika Kardiologiya kolleji jurnali* , jild. 76, yo'q. 17, 2011–2023, 2020-betlar.

11. Xalilov, X. D., N. K. SHadmanova, and M. N. Qayumov. "Gipertireorizmni exsperemental modellashtirish." (2023).

12. W. Ni, X. Yang, J. Liu va boshqalar, "Kasalxonaga yotqizilganida o'tkir miokard shikastlanishi COVID-19da barcha sabablarga ko'ra o'lim bilan bog'liq", *Journal of the American College of Cardiology* , jild. 76, yo'q. 1, 124-125-betlar, 2020 yil.

13. H. Jia, "O'pka angiotensin-konverting fermenti 2 (AAF2) va yallig'lanishli o'pka kasalligi", *Shok* , jild. 46, yo'q. 3, 239–248-betlar, 2016 yil.

14. V. Xia, J. Shao, Y. Guo, X. Peng, Z. Li va D. Xu, "COVID-19 infektsiyasi bo'lgan pediatrik bemorlarda klinik va KT xususiyatlari: kattalardan farqli nuqtalar", *Pediatrik pulmonologiya* , jild. 55, yo'q. 5, 1169–1174-betlar, 2020 yil.

15. Xalilov, H., Namiddinov, A., Sayfullayeva, D., & Hikmatova, G. (2024). TELEMEDITSINANING PROFILAKTIK DAVOLANISHDA AHAMIYATI. *Евразийский журнал академических исследований*, *4*(4 Part 2), 66-70.

16. S. Shi, M. Qin, B. Shen va boshqalar, "Uxandagi COVID-19 bilan kasalxonaga yotqizilgan bemorlarda yurak shikastlanishining o'lim bilan bog'liqligi, Xitoy," *JAMA Cardiology* , jild. 5, yo'q. 7, 802–810-betlar, 2020 yil.

17. Q. Ruan, K. Yang, V. Vang, L. Jiang va J. Song, "Uxan, Xitoydan kelgan 150 bemor ma'lumotlari tahlili asosida COVID-19 tufayli o'limning klinik ko'rsatkichlari", *intensiv terapiya tibbiyoti* , jild. 46, yo'q. 5, 846–848-betlar, 2020 yil.

18. B. Li, J. Yang, F. Zhao va boshq., "Xitoyda yurak-qon tomir metabolik kasalliklarining COVID-19 ga tarqalishi va ta'siri" , *Kardiologiyada Klinik tadqiqotlar* , jild. 109, yo'q. 5, 531–538-betlar, 2020 yil.

19. Dilshodovich, K. H., Normurotovich, K. M., & Akromovich, E. A. (2023). RELATIONSHIP BETWEEN THYROID DISEASE AND TYPE 2 DIABETES.

20. P. Libby, J. Loscalzo, PM Ridker va boshqalar, "Aterotrombozda yallig'lanish, immunitet va infektsiya" , *Amerika Kardiologiya kolleji jurnali* , jild. 72, yo'q. 17, 2071–2081-betlar, 2018 yil.

21. L. Sarkisyan, L. Saaby, TS Poulsen va boshq., "Miokard infarkti, miokard shikastlanishi va troponinlar darajasi oshmagan bemorlarning klinik xususiyatlari va natijalari", *The American Journal of Medicine* , jild. 129, yo'q. 4, 446-e21-betlar, 2016 yil.

22. DR Lakkireddi, “Koronavirus ritmi jamiyatining COVID-19 ishchi guruhidan COVID-19 pandemiyasi davrida yurak elektrofiziologiyasi bo‘yicha qo‘llanma; Amerika kardiologiya kollejining elektrofiziologiya bo'limi; va klinik

kardiologiya bo'yicha kengashning elektrokardiografiya va aritmiya qo'mitasi, Amerika yurak assotsiatsiyasi, " *Tiraj* , jild. 141, yo'q. 21, e823–e831-betlar, 2020 yil.

23. IA Hobai, J. Edgecomb, K. LaBarge va WS Colucci, "Sepsis keltirib chiqaradigan kardiyomiyopatiyaning hayvon modellarida hujayra ichidagi kaltsiy tashuvchilarning disregulyatsiyasi", *Shock* , vol. 43, yo'q. 1, 3–15-betlar, 2015 yil.

24. Xalilov, X. D., SHadmanova, N. K., & Qayumov, M. N. (2023). Gipertireorizmni exsperemental modellashtirish.

25. JSST, *vaqtinchalik COVID-19 bilan bog'liq bolalar va o'smirlardagi ko'p tizimli yallig'lanish sindromi* , JSST, Jeneva, Shveytsariya, 2020 yil.