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2025 | Vol 30 | Issue 2 | Page 31-35
Journal Homepage: https://zkdx.ch/
DOI: 10.1654/zkdx.2025.30.2-04

Changes in the Cardiovascular System after Covid-19 in Children

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Abstract
Medical research is being conducted around the world to study the pathogenesis of the SARS CoV-2 virus, its etiology, normal long-term activity of people, various strains of the virus, vaccination issues, as well as the prevention of various procedures during the recovery process after an illness. In this regard, it was of interest to conduct follow-up studies of the course of post-COVID syndrome: the clinical course and general clinical studies of children after suffering from COVID-19 of varying severity, who was undergoing outpatient treatment at Polyclinic No. 33. Hematological, biochemical, immunoenzyme and instrumental studies were carried out at the TMA Children's Clinic.

Keywords
Post-Covid syndrome, Children, Cardiovascular system, Myocardial damage, Diagnosis, Complications, EKG, EcoKG children of different ages

1. Introduction
The COVID-19 pandemic caused by SARS-CoV-2 is spreading at an alarming rate worldwide, creating a global health emergency. According to the World Health Organization, “most patients with COVID-19 with mild (40%) or moderate (40%) coronavirus disease, approximately 15% experience pneumonia with the development of atypical acute respiratory distress syndrome, and 5% have an extremely severe course with complications such as sepsis and septic shock, thromboembolism and/or multiple organ failure, including acute kidney and heart failure” [14]. Emerging and reemerging infectious diseases are persistent and have gained particular importance in recent years [11]. This was confirmed during the COVID-19 pandemic [14]. Coronaviruses have been known for a long time, can infect various animal species and humans, are strictly species-specific, and cause mainly respiratory and gastrointestinal tract diseases [7]. In humans, disease is caused by four genotypes of circulating single-stranded RNA-containing coronavirus viruses (HCoV2-229E, HCoV-OC43, HCoV-NL63 and HCoVHKU1B, classified in pathogenicity group II) [2, 4, 8]. The viral genome encodes four structural proteins: spike protein (S), membrane protein (M) and nucleocapsid protein (N) [6, 8, 13]. The virus uses the receptor-binding domain (RBD) of the S1 protein and angiotensin-converting enzyme 2 (ACE-2) to enter the cell. The binding of this domain to the receptor promotes membrane fusion, and the remaining viral proteins facilitate genome stabilization and viral replication [10]. ACE-2 receptors are mainly localized in alveolar macrophages and dendritic cells, cells of the upper and lower respiratory tract, intestinal epithelial cells, myocardium, endothelial cells, renal tubule cells, and neurons of the brain, which determines the clinical features of the course of the disease and the routes of infection. Compared with adults, children may be less susceptible to COVID-19 infection due to reduced function of ACE and their receptors[15]. Based on the data obtained, it can be concluded that moderate to severe COVID-19 is typical for adolescent children. This group of children shows a pronounced induction of IL-6. [16, 17, 18]

According to statistics, a general susceptibility to SARS-CoV-2 is assumed, and cases of severe infection and fatal outcomes are typical mainly in the elderly. According to the authors, this disease in children is characterized by a milder course and very few complications and adverse outcomes. An analysis of 45 references on the incidence of this infection in children demonstrated an incidence of 1-5%, characterized by a mild course and the absence of vertical transmission of the infection [1]. According to other authors, this figure may be slightly higher in countries with higher proportions of children and adolescents. The largest study of 1,787,680 confirmed cases of COVID-19 testing conducted

by the U.S. Centers for Disease Control and Prevention (CDC) showed that the proportion of children and adolescents under 18 years of age was 3.2%, with low susceptibility to infection and a mild course [12]; H.P. Alimova et al. (2021), the course of infection and the development of the clinical picture depend on the antemortem background, the presence of concomitant pathologies, overlapping infections, the nature of the immune response, and the age of the sick child [1].

The Aim of the Study

Assess the cardiovascular status of children recovering from COVID-19 according to the severity of the infection process.

2. Material and Research Methods

Thecasehistoriesof103childrenofvariousages admitted with a diagnosis of novel coronavirus infection COVID-19 between September and November 2020 was retrospectively analyzed. There were 58 boys (56.3%), girls - 45 (43.7%).

3. Result and its Discussion

Previous studies have shown that the frequency of complaints of pain in the heart area was reported by 2 (13.3%) of 15 children who had a mild form of coronavirus infection, 20 (43.4%) of 53 children who had a moderate form, and all 20 children (100.0%) who had a severe form of the infectious process (see Fig. 1.1). Therefore, the main goal of this chapter is to provide information on biochemical indicators reflecting the state of cardiac tissue in children who have had coronavirus infection depending on the severity of its course.

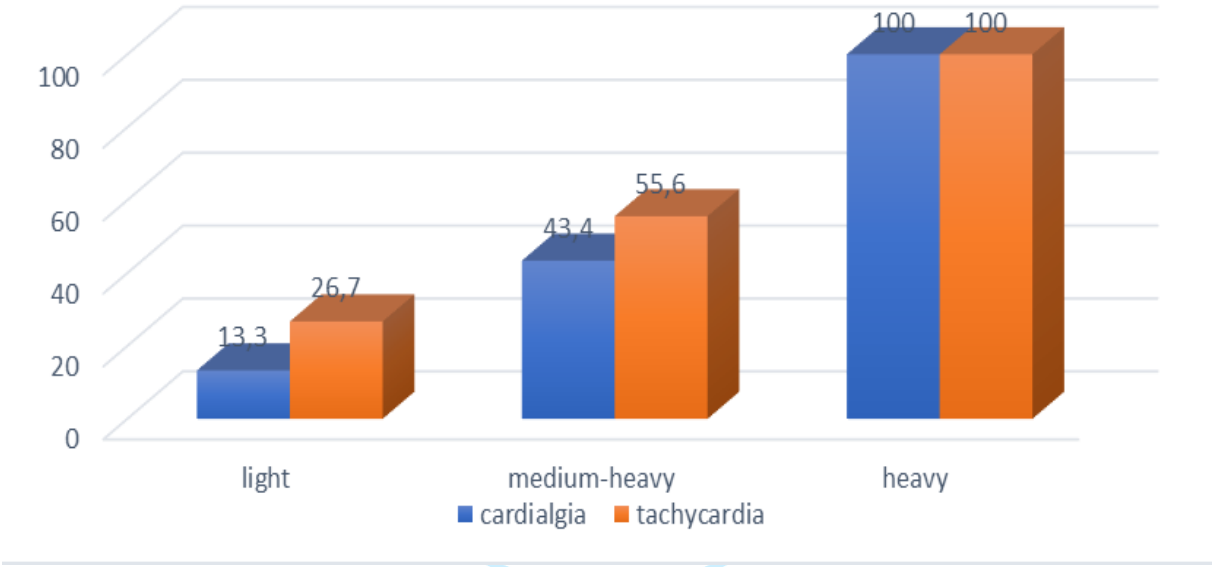

Fig. 1.1 Frequency of complaints of pain in the heart area and increased heart rate in children who have had coronavirus infection, depending on the severity of the pathology.

Complaints of increased heart rate were reported by 100% of children who had suffered a mild and severe form of coronavirus infection. Therefore, we compared electrocardiographic parameters in children affected by coronavirus infection according to the severity of the course of the disease. It must be said that the deterioration of myocardial enzyme activity in serum is indicative of a complex process in response to infectious and inflammatory processes that develop hypoxia. In the children with COVID-19 we studied, radiography and MSCT showed the development of lung damage of varying severity, the presence of 'ground glass' and increased grade I and II cardiothoracic indices. On ECG, myocarditis was characterized by combined sinus tachyarrhythmias and brady arrhythmias with rhythm and conduction disturbances, complete or incomplete block of the bundle of His in the left and especially in the right leg, and decreased amplitude of the QRS complex teeth, especially in children with severe COVID-19 (see Table 1.1). Combined rhythms of sinus tachycardia and bradyarrhythmia and conduction disturbances were most frequently observed in ECGs of children with moderate to severe disease [10].

Table 1.1 Frequency of ECG changes in children who have had COVID-19 depending on the severity of the disease

Indicators	Mild course, n=30		Moderate-severe course, n=53		Severe course, n=20	
	N	%	n	%	n	%
Morphology of the ventricular complex						
Depolarization phase:	0	0,0	2	3,8	5	25,0
- pathological Q wave	2	6,6	17	32,0	13	65,0
- low QRS voltage	0	0,0	2	3,8	6	30,0
Repolarization phase:- changes in T wave	1	3,3	9	16,9	4	20,0
Cardiac conduction disorders						
Arrhythmia	2	6,6	10	18,9	6	30,0
Sinus tachycardia	4	26,7	11	20,8	7	35,0
Sinusov. bradycardia	4	26,7	17	32,1	6	30,0
AV block	0	0,0	0	0,0	0	0,0
- blockade of LBP	1	3,3	6	11,3	4	20,0
-blockade of PNPG	2	6,6	20	37,7	14	70,0
Extrasystole	0	0,0	8	15,0	4	20,0
SSSU	0	0,0	0	0,0	0	0,0
Ventricular hypertrophy						
Right ventricle	1	6,7	7	13,2	2	10,0
Left ventricle	0	0,0	9	17,0	4	20,0
Both sections	0	0,0	0	0,0	0	0,0
Metabolic disorders	1	6,7	4	7,5	5	25,0

The most common intraventricular conduction disturbances were complete and incomplete right bundle branch block and, somewhat less commonly, left bundle branch block. In children with severe COVID-19, there was a decrease in the amplitude of the QRS complex teeth, more pronounced in standard and enhanced unipolar limb leads, indicating the acute and widespread nature of myocardial damage, especially in children with PMIS. To better characterize the myocardial status, a mathematical analysis of ECG parameters according to the severity of coronavirus infection in children was performed (see Table 1.2). The results showed an increase in heart rate, R-R and Q-T distances, the severity of which depends on the severity of COVID-19. In addition, changes were also detected in other ECG parameters indicating the presence of myocardial damage, especially in children with severe COVID-19 [10].

Table 1.2 Quantitative ECG indicators in children who have had COVID-19 depending on the severity of the disease, M±m

Indicators	Mild course, n=30	Moderate-severe course, n=53	Severe course, n=20
Heart rate, beats/min	106,13±5,72	102,49±4,31	94,5±7,26
R-R	0,587±0,035	0,625±0,024	0,670±0,03
P	0,065±0,002	0,067±0,002	0,071±0,002
P-Q	0,123±0,004	0,122±0,002	0,127±0,005
QRS	0,073±0,003	0,071±0,001	0,082±0,005
Q-T	0,290±0,012	0,294±0,006	0,312±0,010

In ECG, children with moderate and severe disease showed the most combined rhythm, conduction and ventricular hypertrophy disorders. Thus, disorders in the repolarization phase, manifested by a change in the T wave, were detected in 26 and 13% of those examined at the age of junior and senior school age, and ST complex - in 37.5; 33; 55 and 13% of pre-school, preschool, junior and senior school children who had recovered from COVID-19, respectively.

ECG changes in children with coronavirus infection can be varied and reflect both the direct effects of the virus on the cardiovascular system and the indirect effects of general inflammation or stress. The most commonly observed ECG changes in children with COVID-19 are: Sinus tachycardia [97; 35]: This is the most common change, which may reflect the body's physiological response to fever, dehydration, or stress from infection. ST segment and T-wave changes: These changes may indicate myocarditis or myocardial ischemia. Myocarditis may be a direct consequence of the viral infection or may occur as part of the multisystem inflammatory syndrome in children (MIS-C) associated with COVID-

19. Prolongation of the QT interval [7; 15]: This may be caused by the disease itself or a side effect of some antiviral and antiarrhythmic drugs used in the treatment of COVID-19. If symptoms or suspected cardiac complications occur in children with COVID-19, a thorough ECG examination should be performed and, if necessary, additional diagnostic procedures such as echocardiography should be performed to assess the structure and function of the heart. This will allow timely detection of potential complications and initiation of appropriate treatment.

From the data obtained, it can be concluded that changes in cardiovascular parameters in childrenaffectedbyCOVID-19 are typical of adolescents: Depending on the severity of COVID-19, total LDH and CPK activity in serum increases, indicating the presence of tissue hypoxia, which is indicated by severe muscle pain. In addition, depending on the severity of COVID-19, the CPK-MB fractionis elevated, indicating myocardial damage in the children studied. This is manifested by a violation of ventricular complex morphology, cardiac conduction and ventricular overload. Overall, further studies are needed to better understand the consequences of the pediatric-acquired form of COVID-19 and these children require strict follow-up measures and continuous monitoring. Hemodynamic studies show signs of left ventricular systolic dysfunction in up to 56% of COVID-19 patients (Table 1.4). According to Li, B., Yang, J., Zhao, F., et al, left ventricular diastolic dysfunction was detected in 57% of pediatric COVID-19 cases with almost the same frequency [7; 9]. Moreover, the development of left ventricular diastolic dysfunction in COVID-19 is associated with adverse outcomes [10].

Systolic dysfunction of the left ventricle is characterized by a decrease in the contractility of the heart muscle. This condition may manifest itself in the following ways: fatigue and weakness - due to insufficient blood supply to the tissues. Shortness of breath-especially during physical exertion, since the heart cannot effectively pump blood to meet the body's need for oxygen. Decreased physical activity - children may become less active due to fatigue and shortness of breath. Echocardiography is the main diagnostic method that allows evaluating the structural and functional parameters of the heart, including the ejection fraction (to evaluate systolic function) and the parameters of diastolic filling of the LV.

Table 1.3 EchoCG indicators in children who have had COVID-19 depending on the severity of the disease, M±m

Indicators	Mild course, n=30	Moderate-severe course, n=53	Severe course, n=20
Aortic root diameter, mm	16,6±6,5	14,6±6,5	18,6±6,5
Left atrium volume, mm	21,2±12,4	22,2±12,4	23,2±12,4*
Right ventricular diameter, mm	14,5±7,2	15,5±7,2	16,5±7,2*
Left ventricular fraction (LVEF)	65,2±14,6*	61,2±12,6	58,6±11,8
Interventricular septum thickness, mm	7,1±2,4	7,1±2,4	7,1±2,4
Left ventricular posterior wall thickness, mm	7,2±1,9	7,2±1,9	7,2±1,9
LV end-systolic diameter, mm	21,4±6,1	22,4±6,2	23,4±6,6
LV end-diastolic diameter, mm	32,5±12,7	32,5±12,5	33,5±11,5
LV end-systolic volume, ml	17,2±4,8	17,2±4,8	17,2±4,8*
LV end-diastolic volume, ml	44,6±12,8	44,6±12,8	44,6±12,8*
Stroke volume, ml	49,4±15,4*	46,4±13,4	41,4±11,0
Myocardial mass, g	56,2±24,5	52,2±24,1	59,2±24,4
Systolic dysfunction, %	11	18	27

Children with severe COVID-19 have an increase in the aortic root diameter (18.6 ± 6.5 mm) compared to children with mild (16.6 ± 6.5 mm) and moderate (14.6 ± 6.5 mm) disease. A gradual increase in the left atrium volume from mild (21.2 ± 12.4 mm) to severe (23.2 ± 12.4 mm) indicates a possible increase in the load on the left atrium in response to the disease. Children with severe disease have an increase in the right ventricular diameter (16.5 ± 7.2 mm), which may indicate an increase in pressure in the pulmonary circulation. A decrease in EF from 65.2% in children with mild disease to 58.6% in children with severe disease indicates a decrease in LV systolic function. Does not change depending on the severity of the disease, which indicates the stability of these structural components of the heart. A slight increase in the size of the LV in systole and diastole with increasing severity of the disease. Volumes remain stable regardless of the severity of the disease. A decrease in stroke volume from mild (49.4 ± 15.4 ml) to severe (41.4 ± 11.0 ml) may reflect a decrease in the efficiency of the heart pump. An increase in myocardial mass in children with severe disease (59.2 ± 24.4 g) may be a consequence of cardiac muscle hypertrophy. Based on the data obtained, it can be concluded that with increasing severity of COVID-19 in children, significant changes in the structure and function of the heart are observed, including an increase in the volumes of the atria and ventricles, a decrease in the ejection fraction and an increase in

myocardial mass. These changes require close monitoring and may require specific cardio logical support during the recovery process.

Another frightening cardiac complication in children with COVID-19 is the development of rhythm disturbances, the frequency of which reaches 12% in severely affected patients. In most cases, about 6-8% of COVID-19 patients experience atrial fibrillation. Atrial fibrillation usually develops within the first three days of illness. Ventricular arrhythmias are less common (about 2%). Thus, based on the analysis and literature data, it can be concluded that the heart plays an important role in the etiology of multiple organ failure in children with COVID-19.The main clinical manifestations of cardiac dysfunction are left and/or right ventricular failure, insufficient cardiac output and the development of arrhythmias. The development of cardiac disorders and the occurrence of cardiovascular failure are based on a complex of various mutually aggravating reactions and mechanisms, including the direct action of microorganisms, activation of inflammatory and anti-inflammatory cytokines associated with the development of a systemic inflammatory response, and disruption of cardiomyocyte structure and function. This is a promising method to study the etiology of heart failure and its relationship with carcinogenesis in COVID-19 patients [10].

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