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# Evaluation of Children Who Develop Myocarditis Due to Coronavirus Disease 2019

## Koronavirüs Hastalığı 2019 (COVID-19) Nedeniyle Miyokardit Gelişen Çocukların Değerlendirilmesi

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## Abstract

**Background:** The aim of this study is to share the data of patients who developed myocarditis due to Coronavirus disease 2019 (COVID-19).

**Materials and Methods:** The data of 50 children who were diagnosed with myocarditis due to Coronavirus disease 19 between April 2020 and January 2022 were retrospectively evaluated.

**Results:** Twenty-four (48%) of the patients were diagnosed with multisystem inflammatory syndrome (MIS-C). Twenty-six (52%) patients had fever and 14 (28%) patients had chest pain, which were the most common complaints. Eight (16%) patients had systolic dysfunction. Pericardiocentesis was performed in one patient due to cardiac tamponade. In four (8%) of the patients, acute fulminant myocarditis developed and intensive care was required. One patient required intubation and was later extubated. Twenty-one (42%) patients were treated with intravenous immunoglobulin alone, and 8 patients were treated with intravenous immunoglobulin + steroids. Four (8%) patients received intravenous inotropic therapy. Twenty-one (42%) patients did not receive any treatment and recovered with bed rest only. None of our patients died.

**Conclusions:** In our study, approximately half of the pediatric patients who had developed myocarditis due to Coronavirus disease 2019 recovered with bed rest only. However, as shown in our study, it may progress to fulminant myocarditis in some patients. Therefore, even if such patients are stable, they need to be followed up closely since mortality can be prevented at a high rate when the appropriate treatments are rapidly provided to patients in need.

**Keywords:** Children, Coronavirus Disease 2019, Myocarditis, Fulminant

## Öz

**Amaç:** Bu çalışmanın amacı, Koronavirüs hastalığı 2019 (COVID-19) nedeniyle miyokardit gelişen hastaların verilerini paylaşmaktır.

**Materyal ve metod:** Nisan 2020 ile Ocak 2022 tarihleri arasında COVID-19'a bağlı miyokardit tanısı alan 50 çocuğun verileri retrospektif olarak değerlendirildi.

**Bulgular:** Hastaların 24'ünde (%48) multisistem inflamatuar sendrom (MIS-C) saptandı. En sık görülen yakınmalar ateş (26 hasta, %52) ve göğüs ağrısı (14 hasta, %28) idi. Sekiz hastada (%16) sistolik disfonksiyon mevcuttu. Bir hastaya kardiyak tamponad nedeniyle perikardiyosentez yapıldı. Dört hastada (%8) akut fulminan miyokardit gelişti ve yoğun bakım gereksinimi oldu. Bir hasta entübasyona ihtiyaç duydu ve sonrasında ekstübe edildi. Yirmi bir hasta (%42) yalnızca intravenöz immünoglobulin ile tedavi edildi, 8 hasta intravenöz immünoglobulin + steroid aldı. Dört hastaya (%8) intravenöz inotropik tedavi uygulandı. Yirmi bir hasta (%42) ise herhangi bir tedavi almadan sadece yatak istirahati ile iyileşti. Hiçbir hastamız kaybedilmedi.

**Sonuç:** Çalışmamızda, COVID-19'a bağlı miyokardit gelişen pediatrik hastaların yaklaşık yarısının yalnızca yatak istirahati ile iyileştiği görüldü. Bununla birlikte, çalışmamızın da gösterdiği gibi bazı olgularda fulminan miyokardite ilerleyebilmektedir. Bu nedenle, bu tür hastalar stabil görünseler bile yakından izlenmelidir; çünkü uygun tedavi, ihtiyaç duyan hastalara hızlıca uygulandığında mortalite büyük ölçüde önlenebilmektedir.

**Anahtar Kelimeler:** Çocuklar, Koronavirüs Hastalığı 2019 (COVID-19), Miyokardit, Fulminan

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## Introduction

Coronavirus disease 2019 (COVID-19) usually has a mild course in children. Nevertheless, it can result in multisystem inflammatory syndrome in children (MIS-C), a condition that may be fatal if not promptly recognized. Furthermore, COVID-19 can also affect the heart and lead to myocarditis. Myocarditis is defined as inflammation of the cardiac muscle, causing myocardial damage in the absence of ischemia (1,2). Among the potential viral triggers of myocarditis, adenovirus, parvovirus B19, Epstein-Barr virus, and cytomegalovirus are considered significant (3,4). Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) has also emerged as a possible infectious cause.

The pathophysiology of viral myocarditis involves both direct viral injury and immune-mediated cell death (1). In the early phase, high viral replication may directly harm cardiomyocytes. Immune-mediated injury is driven by natural killer cells, macrophages, and T lymphocytes infiltrating the myocardium (5). Additionally, cytokines such as interleukin (IL)-1β and IL-17 contribute to cardiac remodeling and fibrosis, ultimately leading to ventricular dilatation and heart failure (6,7). The presence of fibrosis in the myocardium disrupts the cardiac conduction system, thereby increasing the likelihood of arrhythmias (8).

SARS-CoV-2 infects host cells via angiotensin-converting enzyme 2 (ACE2) receptors. While these receptors are highly expressed in alveolar type II epithelial cells—explaining the pulmonary manifestations of COVID-19—they are also present in cardiomyocytes (9,11).In one reported case, endomyocardial biopsy (EMB) identified SARS-CoV-2 viral particles in the myocardium of a COVID-19 patient (12). This finding demonstrates that the virus can directly cause myocarditis by infecting heart muscle cells (13). Alternatively, SARS-CoV-2 may induce myocardial damage through infection of cardiac endothelial cells (14).

Systemic inflammation has also been implicated in COVID-19-associated myocarditis. Interleukin-6 (IL-6) is a central mediator of the cytokine storm. In certain patients, excessive release of proinflammatory cytokines results in a dysregulated immune response (15,16). This hyperinflammatory state may promote coronary artery thrombosis (17) and directly contribute to myocardial damage and myocarditis.

Clinically, COVID-19 most commonly presents with pneumonia, manifesting as fever, cough, and dyspnea (18). However, it has also been shown to cause cardiovascular complications, particularly myocarditis. The clinical spectrum of COVID-19-related myocarditis ranges from mild disease to severe, fulminant presentations. Therefore, physicians must be vigilant in reconizin these cases, as failin to provide timely treatment can lead to life-threatenin outcomes (19).

The objective of this study is to enhance understandin of COVID-19-associated myocarditis in children and to emphasize timely and appropriate manaement in order to prevent fatal complications.

## Materials and Methods

***Study Population***

In the study, patients between the aes of 0-18, who were dianosed with myocarditis due to COVID-19 in the Department of Pediatric Cardioloy of our hospital between April 2020 and January 2022, were included. Ethical approval was obtained from the Ethics Committee of the Dicle University Center (approval no: 75, date: March 17, 2022). The sociodemoraphic characteristics (ae, ender), complaints, clinical findings, hemodynamic features, history of comorbidities, lenth of hospital stay, severity and proression of the disease, telecardioraphy and electrocardioraphy results, and laboratory data (hemoram, biochemistry, C-reactive protein, NT-proBNP, CK-MB, troponin), treatments and outcome of the patients were evaluated retrospectively. Informed consent was obtained from the parents of the patients.

The dianostic criteria for acute myocarditis included elevated troponin levels, ST sement elevation or depression on electrocardioram (ECG), reduced left ventricular function (ejection fraction <55%) with reional wall motion abnormalities on echocardioraphy (20).

Multisystem inflammatory syndrome in children (MIS-C) is a clinical condition that develops followin SARS-CoV-2 infection and is characterized by excessive and widespread inflammation affecting multiple oran systems (an abnormal increase in the body's immune response). This syndrome can cause inflammation in multiple oran systems such as the heart, blood vessels, diestive system, kidneys, skin, eyes, and nervous system, and can lead to severe illness (21).

Acute fulminant myocarditis is a clinical condition characterized by sudden-onset and severe inflammation of the heart muscle, leadin to significant heart failure, hypotension, and/or cardioenic shock (22).

Shock was defined as the concurrence of tachycardia and one of the followin symptoms: systemic arterial hypotension (Pediatric basic life support alorithm), cold extremities, weak peripheral pulse, a capillary refill time of >3 seconds, oliuria, or an arterial blood lactate level of >2 mmol/L (23).

Standard microbioloical screenin was performed. The

common etiologies of acute myocarditis were systematically screened. SARS-CoV-2 was diagnosed by examining the results of polymerase chain reaction (PCR) test, performed by collecting nasopharyngeal swab specimen, and by detecting SARS-CoV-2 antibodies (IgG and IgA) in blood.

***Statistical Analysis***

Statistical analyses were performed using IBM SPSS Statistics for Windows. Descriptive statistics were expressed as number (n), percentage (%), mean±standard deviation (SD), or median (minimum-maximum), as appropriate. The normality of continuous variables was assessed using the Shapiro-Wilk and Kolmogorov-Smirnov tests. Variables with normal distribution were reported as mean±standard deviation (SD), whereas variables that did not follow a normal distribution were presented as median (minimum-maximum). A p value <0.05 was considered statistically significant.

## Results

In the study, 22 (44%) of the patients who developed myocarditis due to the novel Coronavirus (COVID-19) were female, and 28 (56%) were male. The mean age of the patients was 112.3±53.75 months. The mean length of hospital stay was found as 5.5±6.46 days. 26 (52%) of the patients had fever and 14 (28%) patients had chest pain, which were the most common complaints. Hypotension was detected in 4 (8%) patients (Table 1). 39 (78%) patients had COVID-19 antibody positivity, 8 (16%) patients had COVID-19 polymerase chain reaction positivity, and 3 (6%) patients had a history of contact with COVID-19 infected individuals. 24 (48%) of the patients were diagnosed with MIS-C. The median troponin level was found as 1817 (0.7-14543 ng/L) ( the median CK-MB level was found as 8.2 (0.5-81 µg/L) and the median proBNP level was found as 5196 (10-35000) (Table 2). 17 (34%) patients had pathological ECG results; the most common pathological ECG findings were sinus tachycardia (n=9, %18) patients) and ST-T changes (in 8 patients). No malignant arrhythmias were detected. 8 (16%) patients had systolic dysfunction. The mean left ventricular ejection fraction (%) of all patients was 65.2 (55-75). At the time of discharge, it was observed that the patients had completely recovered from systolic dysfunction. In 13 (26%) of the patients, mitral regurgitation (5 patients had moderate mitral regurgitation, and 8 patients had mild mitral regurgitation) was detected. Pericardiocentesis was performed in one patient due to cardiac tamponade. In one patient, dilatation of the left coronary artery was detected ( z score:+3.1). At the time of discharge, coronary dilatation was found to be improved. In addition, our patients experienced dermatological, gastrointestinal, and neurological system involvement. All patients were hospitalized for treatment. Four patients presented with signs of fulminant myocarditis. In 4 (8%) of the patients, intensive care was required. One patient required intubation and was later extubated. 21 (42%) patients were treated only with intravenous immunoglobulin, and 8 patients were treated with intravenous immunoglobulin + steroids. 4 (8%) patients received intravenous inotropic therapy. 21 (42%) patients did not receive any treatment and recovered with bed rest only. None of our patients had complications or died.

**Table 1.** Demographic, clinical and hemodynamic data

| | |
|---|---|
| Age (month), median (range) | 112.3 (1-216) |
| Length of hospital stay (days), median (range) | 5.5 (1-30) |
| Glasgow Coma Scale, median (range) | 14.6 (9-15) |
| Left ventricular ejection fraction (%), median (range) | 65.2 (45-75) |
| Gender, | Variable n (%) |
| Male | 28 (56) |
| Female | 22 (44) |
| **Clinical findings** | |
| Fever | 26 (52) |
| Chest pain | 14 (28) |
| Palpitation | 1 (2) |
| Skin rash | 2 (4) |

| **Table 1.** Continued | |
|---|---|
| Abdominal pain | 6 (12) |
| Headache | 3 (6) |
| Vomiting | 1 (2) |
| Difficulty in breathing | 3 (6) |
| Cough | 2 (4) |
| **Hemodynamic features** | |
| Tachycardia | 9 (18) |
| Systemic arterial hypotension | 4 (8) |
| n: Number | |

| **Table 2.** Laboratory and treatment data | | |
|---|---|---|
| **Laboratory values, median (range)** | **Mean ± SD** | **Median (Min-max)** |
| Haemoglobin, g/dL (12.9-14) | 12.0±1.9 | 45-458 |
| Platelet count, $10^3$ /µL (155-368) | 238 | 0.36-11.1 |
| Lymphocyte count, $10^3$ /µl (1.09-2.99) | 2.45 | |
| White blood cell count, $10^3$ /µl (4.19-9.43) | 10.2±3.5 | |
| Sodium, mmol/L (136-146) | 134±4 | |
| Potassium, mmol/L (3.5-5.1) | 4.3±0.6 | |
| Chlorine, mmol/L (101-109) | 103±5 | |
| Calcium, mg/dL (8.8-10.6) | 9.3±0.9 | |
| Magnesium, mg/dL (1.8-2.6) | 2.0±0.2 | 0.02-27.70 |
| C-reactive protein, mg/L (0-5) | 8.8 | 0.7-14543 |
| Troponin, ng/L (0-11) | 1817 | 0.5-81 |
| Creatine Kinase-MB (mass), µg/L(0.6-6.3) | 8.2 | 10-35000 |
| NT-proBNP, pg/mL ( 100 µg/L) | 5196 | |
| **Evidence of COVID (RT-PCR, antigen test or serology positive) or likely contact with patients with COVID** | | |
| COVID-19 PCR positivity, n (%) | 8 (16) | |
| COVID-19 IgG positivity, n (%) | 39 (78) | |
| COVID-19 contact history, n (%) | 3 (6) | |
| **Highest level of care** | | |
| General pediatric unit, n (%) | 46 (92) | |
| Intensive care unit, n (%) | 4 (8) | |

| **Table 2.** Continued | |
|---|---|
| **Treatments** | |
| Patients who did not receive treatment and were put on bed rest only, n (%) | 21 (42) |
| Only one dose IVIG, n (%) | 21 (42) |
| IVIG + steroids, n (%) | 8 (16) |
| Inotropes and vasoactive drugs, n (%) | 4 (8) |
| Mechanical ventilation, n (%) | 1 (2) |
| n: Number, COVID: Coronavirus dissease, IVIG: Intravenous immunoglobulin, NT-proBNP: N-terminal (NT)-prohormone brain natriuretic peptide, RT-PCR: Reverse transcriptase-polymerase chain reaction, SD: Standard deviation | |

## Discussion

Although COVID-19 is benign in children compared to adults, there were some reports of cardiac involvement resembling MIS-C and often the features of Kawasaki disease. Some of these patients were treated with intravenous immunoglobulin (IVIG) and steroids (24). In some patients, myocarditis may develop due to COVID-19. The underlying pathophysiological mechanism of COVID-19-related myocarditis is currently unclear (25). The occurrence of COVID-19-related myocarditis on an average of 4 weeks after the disease and the significantly high levels of IgG and IgA strongly suggest that the myocardium is affected by the immune response following the viral infection (26). In addition, the improvement of cardiac functions and the decrease of inflammatory markers after receiving IVIG therapy also support this.

It is known that ACE-2 is widely produced in cardiomyocytes, cardiac fibroblasts, and coronary endothelial cells. The SARS-CoV-2 virus uses the ACE-2 receptor for host cell entry. These data suggest that SARS-CoV-2 may directly cause myocardial damage. COVID-19 usually progresses with mild respiratory symptoms in children (27). When acute myocarditis develops in patients with COVID-19, the approach to these patients is no different from that for other viral myocarditides. Symptomatic treatment is applied according to the severity of myocarditis. The treatment of myocarditis includes managing myocardial inflammation and its complications. The efficacy of intravenous immunoglobulins in the treatment of viral myocarditis has been investigated. It has been found that IgG, IgA and IgM immunoglobulins have anti-inflammatory activity and are effective in clearing pathogens from the myocardium (28). Hu et al. (26) used a combination of glucocorticoid and immunoglobulin therapy to successfully treat COVID-19-related myocarditis. In a meta-analysis, it was stated that IVIG therapy in acute myocarditis significantly improved left ventricular functions and decreased mortality. Furthermore, it was also found that IVIG therapy increased the survival time in fulminant myocarditis (29). Fulminant myocarditis is not common in other viral myocarditides. The reason for this has not been clarified yet. Therefore, histological cardiac evaluations are needed to better elucidate the COVID-19 disease. The underlying mechanism of heart damage remains unclear, as endomyocardial biopsy is not performed in most of the patients. In our study, endomyocardial biopsy was not performed either, as it is not a routinely performed procedure in pediatric patients with myocarditis and was recommended only for critically ill patients who were unresponsive to treatment.

In children presenting with the symptom of fever, who then rapidly develop shock and malignant arrhythmia without fever and respiratory symptoms, acute myocarditis due to COVID-19 disease should be considered. Although rare, acute fulminant myocarditis was recently reported in children, and these patients were treated in intensive care units. In these cases, severe left ventricular failure developed, for which intravenous immunoglobulin and steroid treatments were administered (30,31). In our study, acute fulminant myocarditis developed in four of the patients and all of them received inotropic therapy, IVIG and steroid therapy. All of the four patients recovered without any complications. There are no comprehensive randomized controlled trials demonstrating the efficacy of antiviral drugs in SARS-CoV-2 myocarditis, and currently available antivirals are not approved as specific treatments for myocarditis (32).

There are no clear data on the use of corticosteroids in the treatment of COVID-19-related myocarditis. In the literature,

it has been stated that COVID-19 may cause cardiac damage by causing cytokine release syndrome and activating the inflammatory response in both adult and pediatric patients (33). Corticosteroids have often been used in adult patients with critical illnesses associated with SARS-CoV-2 to reduce inflammation (34). Prednisolone is considered to be effective in the treatment of viral myocarditis in the absence of viral replication. In a systematic review by Sawalha et al. (35), it was found that patients with COVID-19-related myocarditis improved with the use of corticosteroids. However, another study argued that corticosteroids did not reduce mortality in viral myocarditis (36). In a study, it was determined that the combination of tocilizumab and favipiravir significantly reduced inflammation that was caused by cytokine storm (37). Since none of the patients included in our study received antiviral therapy, no data on antiviral therapy were included in the study.

All types of arrhythmias may occur in patients with COVID-19-related myocarditis. In a case presentation by Kohli et al., it was shown that the patient developed atrial fibrillation and cardioversion was required (31). In the study conducted by Kesici et al. (30), extracorporeal membrane oxygenation (ECMO) was performed in an infant with fulminant myocarditis, but the patient was died. In the study of Tseng et al. (38), in the follow-up of a patient who developed sustained ventricular tachycardia, ECMO was performed. In patients with fulminant myocarditis presenting with cardiogenic shock, the use of inotropes and mechanical ventilator support, and in patients with cardiac arrhythmia, antiarrhythmics such as amiodarone may be required. If these arrhythmias cannot be managed, a cardioverter defibrillator or cardiac pacing may be needed. Since none of our patients developed malignant arrhythmia, antiarrhythmic therapy was not administered. Although we attribute the absence of arrhythmias in our patients to early diagnosis and treatment, We believe that when the number of patients increases, some patients may develop arrhythmias.

### *Study Limitations*

Although cardiac magnetic resonance imaging (CMR) is an important procedure for the diagnosis of myocarditis, it could not be performed at our hospital. Even if performing CMR was possible, it would not change the clinical management of the disease; moreover, healthcare personnel had to be protected from exposure to COVID-19. Endomyocardial biopsy was not performed in our patients, as it is not a routinely performed procedure in pediatric patients with myocarditis and was recommended only for critically ill patients who are unresponsive to treatment. The lack of CMR and endomyocardial biopsy may be a limitation of our study.

## Conclusion

Myocarditis due to COVID-19 is an important complication that is worsening the prognosis in patients with SARS-CoV-2 infection. While some patients with COVID-19-related myocarditis may be asymptomatic, some may present with fulminant myocarditis and malignant arrhythmias. Although there are not enough data in the publications on the treatment of COVID-19-related myocarditis, it is thought that the combination of IVIG and corticosteroid significantly reduces mortality in fulminant myocarditis. Therefore, clinicians should be careful in terms of arrhythmias, signs of heart failure and life-threatening fulminant myocarditis; and appropriate treatments should be initiated as quickly as possible.

***Ethical Approval:*** *Ethical approval was obtained from the Ethics Committee of the Dicle University Center (approval no: 75, date: March 17, 2022).*

***Author Contributions:***

*Concept: H.B., M.T., A.K.*

*Literature Review: H.B., M.T., D.U., A.A., K.Y., F.A., K.Ş.*

*Design: K.Y., F.A., K.Ş.*

*Data acquisition: D.U., A.A., K.Y.*

*Analysis and interpretation: H.B., M.T.*

*Writing manuscript: M.T., D.U.*

*Critical revision of manuscript: F.A., K.Ş.*

***Conflict of Interest:*** *The authors have no conflicts of interest to declare.*

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