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Dilated Cardiomyopathy Post COVID-19 Infection in Children: Case Report

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Abstract
Introduction: *Dilated cardiomyopathy (DCM) is a disorder of cardiac muscle characterized with ventricular dilatation and decreased diastolic function. Although the most common cause of DCM is idiopathic, several cases of DCM in COVID-19 infection have been reported.* ***Case report:*** *A 2 years-old boy presented with shortness of breath and cough for 2 weeks, especially during activity. Physical examination revealed hypertension, tachypnoea, fever, crackles in both lungs, and intracoastal retractions. Chest x-ray showed cardiomegaly (CTR 0.63) and minimal pulmonary enema as well as inhomogeneous perihilar and right paracardial opacity with ill-defined borders and air bronchogram. SARS-CoV-2 IgG was positive. Echocardiography confirmed DCM, pulmonary hypertension, and minimal pericardial effusion.* ***Discussion:*** *Echocardiographic for ventricular dysfunction is considered the best method for evaluation and diagnosis. Treatment for DCM depends on the degree of myocardial remodelling and the clinical symptoms. The latest management algorithm recommends the use of ACE-Inhibitors, B-Blockers, and Diuretics. The patient experienced improvement after 2 weeks of treatment with furosemide, captopril, spironolactone, and digoxin therapy.* ***Conclusion:*** *Diagnosis and management of DCM depends on the clinical findings and imaging, and requires subspecialty management. Since the COVID-19 pandemic, it is necessary to consider COVID-19 infection in children with symptoms of respiratory tract infection.*

Keywords: Dilated Cardiomyopathy; COVID-19; Paediatric; Case Report;

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Introduction

Cardiomyopathy is uncommon in children but carries a high risk of morbidity and mortality. Cardiomyopathy is the most common indication for heart transplantation in childhood, particularly in children over the age of one. Paediatric cardiomyopathy can be caused by a variety of factors, including genetic variations affecting the myocardium and systemic diseases that cause diffuse myocardial damage. Population-based studies in the United States, Finland, and Australia estimated the incidence of primary cardiomyopathy in children at 1 case per 100,000 person-years in individuals aged <20 years (Lipshultz et al., 2019)

The initial 1980 World Health Organization classification divided cardiomyopathy into three categories based solely on phenotype: dilated, hypertrophic, and restrictive (Lipshultz et al., 2013) Dilated cardiomyopathy (DCM) is a heart muscle disorder characterized by ventricular dilation and reduced diastolic function. Although the most common cause of DCM is idiopathic, there have been reports of DCM caused by COVID-19 infection. SARS-CoV-2 reports from China revealed myocardial damage in some patients.

The lack of significant left ventricular dilatation, which is common in paediatric DCM patients, is also an unusual finding. Left ventricular size and function returning to normal within 2 weeks after symptoms appear suggests reversible acute myocardial damage caused by SARS-CoV-2 (Sharma et al., 2020). The prognosis for children with DCM is poor, with a 5-year survival rate of around 30-50%. Treatment for DCM depends on the degree of heart muscle remodelling and the clinical symptoms that appear. The latest management algorithm recommends the use of ACE-Inhibitors and B-Blockers as well as the administration of diuretics (Mahmaljy et al., 2023)

Case Presentation

A two-year-old boy came to the Emergency Room with his parents, complaining of shortness of breath and coughing. Shortness of breath began two weeks before entering the hospital. Shortness of breath and coughing are experienced, particularly when the child is active. Fever has been present for three days prior to hospitalization. All anamnesis also revealed a history of persistent coughing and shortness of breath. Defecation and urination are normal.

There are no family members of the patient who have the same condition as the patient. The patient was born normally in the hospital with the assistance of a midwife, and it was reported that the patient began crying immediately. The patient's basic immunization records are complete. The patient has a normal growth and development history. On physical examination, the patient was found to be short of breath, compos mentis. On examination of vital signs, blood pressure was found to be 144/76 mmHg, pulse rate 84 times/minute, respiratory rate 38 times/minute, body temperature 37.7˚ C, SpO2 95% room air.

On examination of the head, eyes, ears, nose, and neck were within normal limits. Cardiac physical examination was within normal limits. On lung examination, rough wet rhonchi were found in both lungs accompanied by intracoastal retractions. On physical examination of the abdomen, genitalia and extremities, no abnormalities were found. On examination the skin was found to feel warm, no cyanosis, no jaundice, good skin turgor, no erythema marginatum, no subcutaneous nodules. The patient's anthropometric status showed a body weight of 12.6 kg (Weight-for-age -1 SD to +1 SD), height 86 cm (Height-for-age -2 SD to -1 SD), body mass index 17.0 (BMI-for-age -1 SD to +1 SD). Based on this data, it is said that the patient has a weight and height appropriate for his age with good nutritional status.

In this patient, a complete blood laboratory examination was carried out and the haemoglobin result was 11.9 g/dL; haematocrit 35.6%; leukocytes 9.8 x 103/uL; erythrocytes 4.38 x 106/uL; platelets 420,000/µL; procalcitonin 0.41%; total lymphocytes 4.02 x 103/mm3. On imaging examination, there was cardiomegaly (CTR: 0.63) accompanied by minimal pulmonary enema and an inhomogeneous semi-opaque opacity in the perihilar and right paracardial areas with indistinct boundaries accompanied by an air bronchogram. Then the patient was referred to referral hospital for further examination. An echocardiography examination revealed widening of the heart chambers with an LVID of 34.3 mm; Mild TR, PG 40 mmHg; Mild PR, PG 17 mmHg; decreased ventricular contraction EF 24-30% MV E/A 2.4, TAPSE 8 mm. The result of echocardiography examination was dilated cardiomyopathy and pulmonary hypertension accompanied by minimally pericardial effusion. A laboratory test at the referral hospital showed a positive result for SARS-CoV-2 IgG. The patient was treated with Furosemide injection 0.5 mg/KgBW/12 hours; Captopril 0.3-0.5mg/KgBW/12 hours; Spironolactone 12.5 mg/12hours; Digoxin 50 mcg/12 hours.

After 2 weeks of treatment, an echocardiography evaluation was carried out with results of improvement in dilated cardiomyopathy and pulmonary hypertension. The patient is planned for outpatient treatment with continued oral therapy with the addition of Methylprednisolone 2 mg/KgBW/day for up to 2 weeks tapering off. During the control, a radiological examination was carried out and the heart size returned to normal (CTR: 0.50).

Discussion

Dilated cardiomyopathy (DCM) is a phenotype characterized by dilatation of the left ventricular chamber and decreased systolic ejection without an increase in left ventricular (LV) wall thickness (Hsu & Canter, 2010). The National Australian Childhood Cardiomyopathy Study (NACCS) and the North American Paediatric Cardiomyopathy Study (PCMR) show similarities in terms of incidence of cardiomyopathy, high prevalence of cases in the first year of life, and incidence according to ethnic and gender differences. In both studies, DCM accounted for more than 50% of the total number of cardiomyopathies observed. The annual incidence of the DCM phenotype is 0.57/100,000. Both studies found that infants (<1 year) had the largest proportion of cases.

A similar proportion of DCM cases have a family history of cardiomyopathy at the time of diagnosis. In both studies, the majority of patients had congestive heart failure at presentation (Hsu & Canter, 2010). The DCM phenotype is associated with myocarditis. Symptoms of myocarditis in children range from sudden death to mimic myocardial infarction, cardiogenic shock, and chronic heart failure. The most common causes of viral myocarditis in children with DCM are parvovirus B19, influenza, Ebstein-Barr virus, human immunodeficiency virus, coxsackie, herpes, and adenovirus. The classic viral prodrome of fever, muscle aches, and nonspecific respiratory and gastrointestinal symptoms can be found in the clinical history of paediatric patients with DCM. Echocardiograms frequently result in an accurate clinical diagnosis of myocarditis. Recent analyses have shown that a clinical diagnosis of myocarditis is associated with a significantly lower risk of mortality or heart transplantation and an increased chance of recovery when compared with children diagnosed with idiopathic DCM (Fatema & Nure Ishrat, 2019)

A family history of cardiomyopathy suggests a genetic disorder. Early studies in the 1980s showed that the number of cases of idiopathic DCM that was thought to be familial was 10%. More than 20 genes have been identified to be associated with the DCM phenotype with dominant, X-linked, recessive, and mitochondrial inheritance patterns. The clinical onset of most familial DCM occurs in adulthood, with sporadic symptoms only in infants or children (Lipshultz et al., 2013)

Mutations in proteins commonly associated with hypertrophic cardiomyopathy, such as b-myosin heavy chain, a-tropomyosin, a-cardiac actin, CTnT, cTnC, and titin may also indicate a primary DCM phenotype that is distinct from the late-stage dilated phase of hypertrophic cardiomyopathy. Recent studies on sarcomere regulatory proteins show that mutations causing hypertrophic cardiomyopathy generally increase calcium sensitivity in cardiac myofilaments, whereas mutations in the same proteins causing the DCM phenotype decrease calcium sensitivity. DCM associated with dystrophin mutations is probably the most common genetic cardiomyopathy encountered by paediatric cardiologists. The DCM phenotype commonly develops in dystrophin cardiomyopathy in mid to late adolescence. Duchenne muscular dystrophy is usually accompanied by skeletal myopathy (Fatema & Nure Ishrat, 2019)

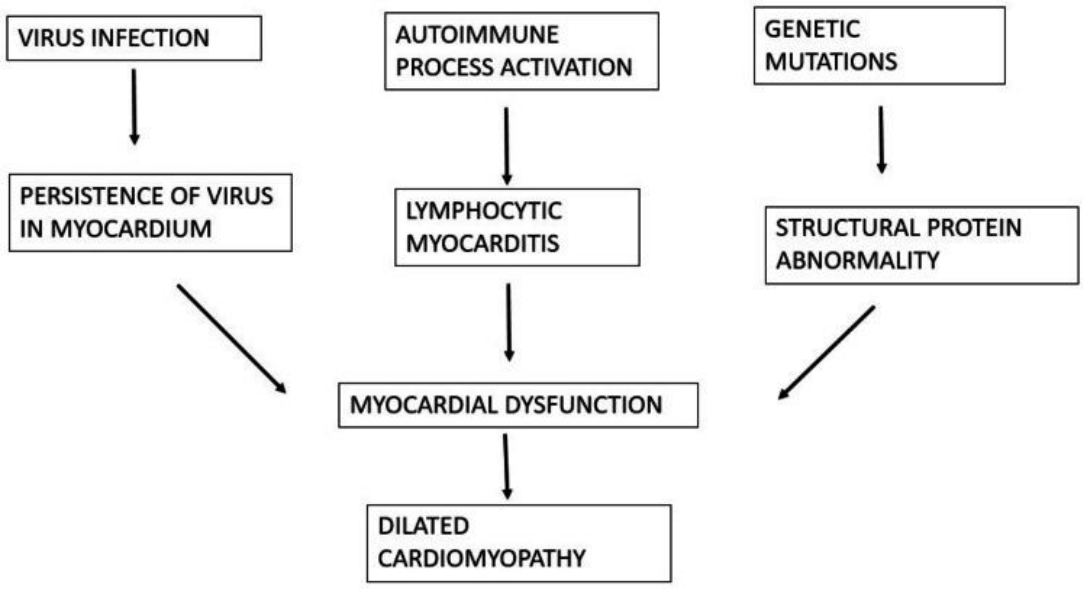

Figure 1. Etiological factors causing dilated cardiomyopathy (Mallavarapu & Taksande, n.d.)

Although the consensus supports viral infection as an initiating factor for idiopathic DCM, genetic predisposition and autoimmunity may underlie severe and progressive myocardial damage in some children. Myocardial damage causes a decrease in cardiac output and initiates a cascade of reactions. Compensatory mechanisms through the renin angiotensin system, the sympathetic system (neural and humoral), and vasodilatory molecules such as natriuretic peptides, prostaglandins, and nitric oxide. Angiotensin (Ang) II promote the secretion of aldosterone and antidiuretic hormone, thereby increasing afterload and preload. Increased preload helps by stretching myocardial cells. Sympathetic stimulation increases heart rate and myocardial contractility thereby improving perfusion of vital organs. However, long-term activation of neurohumoral mechanisms causes end-organ damage (Venugopalan et al., 2000)

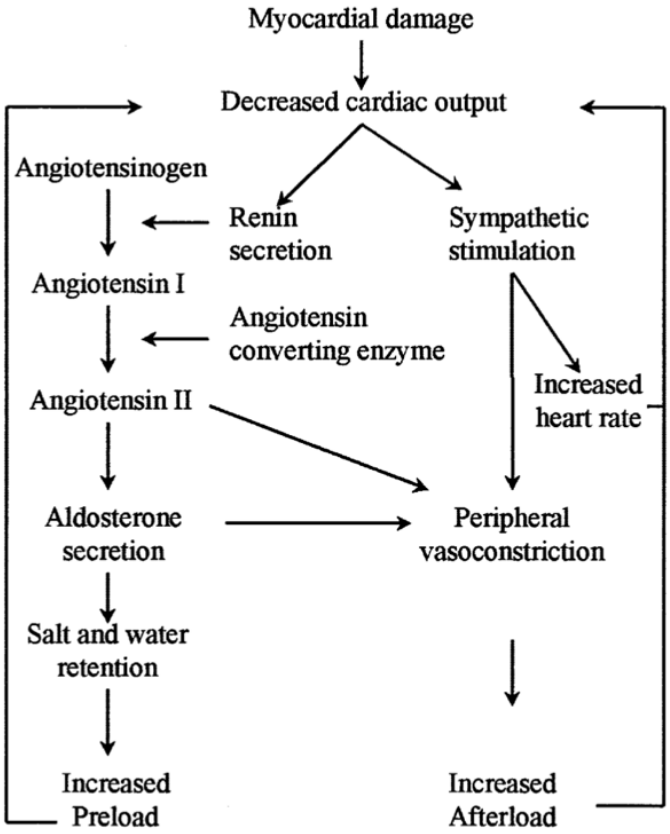

Figure 2. Mechanisms of neurohumoral compensation in chronic heart failure (Venugopalan et al., 2000)

Patients may present with symptoms of heart failure, such as sweating, shortness of breath, orthopnoea, and decreased exercise tolerance, among others. Young children usually show decreased appetite and cachexia. Sinus tachycardia, distended jugular venous pressure, pallor, and hepatomegaly are some of the clinical signs that can be seen. Further signs may include peripheral enema and abdominal distension. Sometimes a murmur of mitral regurgitation can be heard. However, the clinical signs and symptoms observed in most children with heart disease include discomfort, weakness, shortness of breath after activity, and pain in the chest. Other common signs and symptoms found in most children with cardiomyopathy include arrhythmia, inflammatory endocarditis, and congestive heart failure or sudden death due to heart failure (Mallavarapu & Taksande, n.d.)

The patient in this case was a 2-year-old boy, who came to the emergency room with chief complaints shortness of breath and coughing. Shortness of breath has been felt for 2 weeks and accompanied by coughing, especially during activities. History of fever for 3 days. There was also a history of recurrent cough accompanied by shortness of breath. On vital signs, it was found that blood pressure was elevated (144/76 mmHg), pulse 84 times/minute, tachypnoea (38 times/minute), fever 37.7˚C, and SpO2 95% room air.

On lung examination, rough wet rhonchi were found in both lung fields accompanied by intracoastal retractions. The first step in the evaluation of a newly encountered DCM phenotype is to exclude secondary cardiomyopathy. Several toxics, nutritional, endocrinologic, or electrolyte abnormalities may be associated with the DCM phenotype (Kepmenkes (KMK). In dark-skinned infants and young children, rickets should be considered because rickets is a known cause of heart failure with features of DCM. Echocardiography to rule out anomalous coronary arteries arising from the pulmonary arteries should be performed because this cardiomyopathy is curable with surgery. The electrocardiogram should be checked not only for findings (atrioventricular block, Wolff-Parkinson-White pattern) that would provide clues to a specific cardiomyopathy but also to rule out persistent tachyarrhythmias that would result in tachycardia-induced cardiomyopathy that can be treated with electrophysiological ablation (Hsu & Canter, 2010). Patients A normal birth was assisted by a midwife and was born immediately crying. Complete basic immunization history. The patient's growth and development history are within normal limits.

The genetic of dilated cardiomyopathy highlights the importance of screening family members at risk. A basic examination of families with DCM should include a detailed family history over a minimum of three generations and an initial clinical examination of first-degree relatives. Consultation with a geneticist or neurologist who specializes in muscular dystrophy may be helpful. Many cardiomyopathies can be associated with skeletal muscle findings. Skeletal muscle biopsy may be useful in infants at higher risk if endomyocardial biopsy is performed and in paediatric patients with abnormalities of skeletal muscle strength or tone (Weintraub et al., 2017)In this cases there is no family members of the patient have similar disease.

Grossly dilating all four chambers of the heart can be identified as a flabby, hypo contracting heart. On histopathology, hypertrophied muscle fibres and ninja star nuclei (irregular and hyperchromatic) can be found due to a titin gene mutation. Box-car nuclei are found in hypertension and atherosclerotic disease; Tokotsubo Cardiomyopathy; and arrhythmogenic cardiomyopathy. Tokotsubo cardiomyopathy, a type of DCM found in situations of extreme emotional stress, is characterized by selective left ventricular enlargement in which the neck of the left ventricle becomes narrow with a rounded bottom resembling Tokutsubo in Japan. Tokutsubo cardiomyopathy is more common in elderly women than men. Arrhythmogenic cardiomyopathy, an autosomal dominant disorder, is generally characterized by right ventricular dysfunction caused by plakoglobin or desmin mutations.

Naxos syndrome is an arrhythmogenic cardiomyopathy characterized by hyperkeratosis of the palms and soles of the feet (Mallavarapu & Taksande, n.d.). Chest x-ray usually shows cardiac chamber hypertrophy and signs of pulmonary venous congestion. Generally, notches in the ribs are more likely to be seen in Takaya's arteritis than in coarctation in older children with suspected heart disease. The electrocardiogram most often shows atrial fibrillation or sinus tachycardia, arrhythmia (usually ventricular), left atrial hypertrophy, and sometimes intraventricular conduction defects, and low

current. In cases of dilated or congestive cardiomyopathy, left bundle branch block (LBBB) is also encountered with deviation towards the right axis, which is not a common finding. Electrocardiography can be used to see sinus tachycardia, conduction block, and changes in left ventricular enlargement. Indistinct boundaries accompanied by air bronchogram.

Echocardiographic evaluation for the presence of ventricular dysfunction is considered the best method for the assessment and diagnosis of various types of heart disease in the paediatric and adolescent age groups. Even though two-dimensional morphological echo and ejection fraction are the critical elements for phenotypic characterization, in echo, morphologically, we can identify biventricular dilatation as an increase in left ventricular end-diastolic dimension (LVDD) and left ventricular end-systolic size (LSVD), atrial enlargement in relation to ventricular enlargement, decreased left ventricular contractility, and apical thrombus on echocardiography. It also showed a decrease in ejection fraction along with dilatation of the left ventricle with regular or thin walls (Mallavarapu & Taksande, n.d.). The patient underwent echocardiography examination with the results of dilated cardiomyopathy and pulmonary hypertension accompanied by minimally pericardial effusion.

Magnetic resonance imaging (MRI) of the heart can be performed to understand the extent of ventricular chamber dilatation, morphology, fibrosis, pumping capacity of the heart, and the origin of the irregular heartbeat, which is important in determining the type and severity of cardiomyopathy and to exclude arrhythmogenic right ventricular cardiomyopathy (ARVC). Cardiac MRI can measure left ventricular dimensions and function, including precise measurements of strain. Gadolinium, used as a contrast agent, is helpful in evaluating fibrosis and is used to provide information about the quality of myocardial tissue. In DCM, the amount of fibrosis and delay in gadolinium contrast instillation are predictors of mortality and also predict the necessity of future hospitalization (Mallavarapu & Taksande, n.d.).

Some blood tests can be used as biomarkers to diagnose genomic mutations, which is useful in management and as an indicator of prognosis. These biomarkers include ventricular natriuretic peptide and N-terminal pro-brain natriuretic peptide (NT-BNP). BNP levels increased significantly. Endomyocardial biopsy can be used in certain patients, for example patients with suspected cardiac hemochromatosis and other infiltrative or malignant diseases. On histological and microscopic examination, biopsy of endomyocardial specimens, along with electron microscopy as a complement, can provide important information to establish the diagnosis of cardiomyopathy in children. (Braunwald, 2008). Transmission electron microscopy (TEM) has been used to discover ultrastructural changes in the heart in cases of pathological remodelling, such as hypertrophy, hypertension, cardiomyopathy, and ultimately progression to heart failure.

TEM is a powerful tool to visualize cardiac ultrastructure with better resolution and magnification than traditional microscopy and imaging techniques, but also to elucidate the contribution of mitochondrial-derived signalling processes to significant myocardial dysregulation. (Collins et al., 2021)

Table 1
Diagnosis of DCM in children (Venugopalan et al., 2000)

	Features that suggest DCM
Symptoms	Shortness of breath Feeding difficulties Failure to thrive
Sign	Tachypnoea, tachycardia Hepatomegaly Evidence of fluid retention
Chest X-ray	Cardiomegaly Pulmonary venous congestion Pulmonary oedema
Electrocardiography	Hypertrophy of left ventricle with strain Low voltage complexes
Echo-Doppler Studies	Dilated left ventricle with global hypokinesia Mitral regurgitation Absence of congenital heart disease

Differential diagnosis (Table 2) will assist in the identification of the curable causes of DCM. Echo-Doppler can identify congenital heart disease, especially coarctation of the aorta, dysplastic mitral valve, and left coronary artery anomaly of the pulmonary artery. Low serum/muscle carnitine levels may indicate systemic carnitine deficiency, whereas hypoglycaemia, metabolic acidosis, or hyperammonaemia are indicators of metabolic abnormalities. Hypoketotic hypoglycaemia is suggestive of organic academia, although it is more often caused by poor intake and catabolic conditions associated with chronic congestive heart failure. When metabolic disorders are suspected, blood and urine tests need to be performed in the acute phase. Cardiac catheters and angiography are performed only in selected patients (Venugopalan et al., 2000)

Tabel 2
Differential diagnosis of DCM in children (Venugopalan et al., 2000)

Approach	**Abnormality noted**	**Possible diagnosis**
Clinical features	Encephalopathy Myopathy Dysmorphic features Hepatosplenomegaly	Metabolic disease or storage disorder
Blood investigations	Raised ESR, CRP Elevated cardiac enzymes Positive viral titres	Viral myocarditis
	Hypoglycaemia with acidosis	Glycogen storage disease, organic acidaemia's
	Hypoglycaemia without acidosis	Fatty acid oxidation defects, systemic carnitine deficiency
Electrocardiography	Supraventricular tachycardia	DCM secondary to arrhythmia
	Antero-septal infarction pattern	Anomalous coronary artery from pulmonary artery
Echocardiography and cardiac catheter studies	Congenital heart disease	DCM secondary to congenital heart disease
Myocardial biopsy	Inflammation with necrosis, positive viral antigens	Myocarditis

Children infected with COVID-19 may be asymptomatic or have fever, dry cough, and fatigue, with some upper respiratory symptoms, such as nasal congestion and runny nose; some patients experience gastrointestinal symptoms, including abdominal discomfort, nausea, vomiting, abdominal pain, and diarrhoea (Hong et al., 2020). In general, paediatric patients with COVID-19 have an excellent prognosis and usually recover within one to two weeks after the onset of the disease (Castagnoli et al., 2020)

In this case, patient was referred to referral hospital for echocardiography examination with results of dilated cardiomyopathy and pulmonary hypertension accompanied by minimally pericardial effusion. A laboratory test at the referral hospital showed a positive result for SARS-CoV-2 IgG. According to one theory, the virus enters host cells more easily by binding to the spike protein and the ACE2 receptor. The virus causes a local inflammatory reaction with infiltration of T and B lymphocytes. In the early clinical phase the replication process of the viral infection in myocardial cells causes cell damage and exposure to latent antigens in the systemic circulation - cardiac myosin which can trigger an autoimmune response (Capone et al., 2020)

Some of them can experience the second phase, associated with exacerbation of inflammation in the heart tissue even if the viral infection has completely disappeared, or post-infectious myocarditis. Persistent cell damage can result in fibrosis and ultimately progression to dilated cardiomyopathy (Vasichkina et al., 2023). Excessive interferon production and increased type 1 and 2 T cell cytokine responses are thought to contribute to myocardial dysfunction. In addition, the researchers found that the key ACE2 receptor for pathogenesis is on the surface of macrophages, and they indicated the interaction of SARS-CoV-2 with CD68+ macrophages, indicating direct viral infection of these cells (Rodriguez-Gonzalez et al., 2020). It was proven that IL-17A is required to cause acute myocarditis in dilated cardiomyopathy, and contributes to the development of foci of fibrosis in the myocardium and loss of cardiac function (Vasichkina et al., 2023)

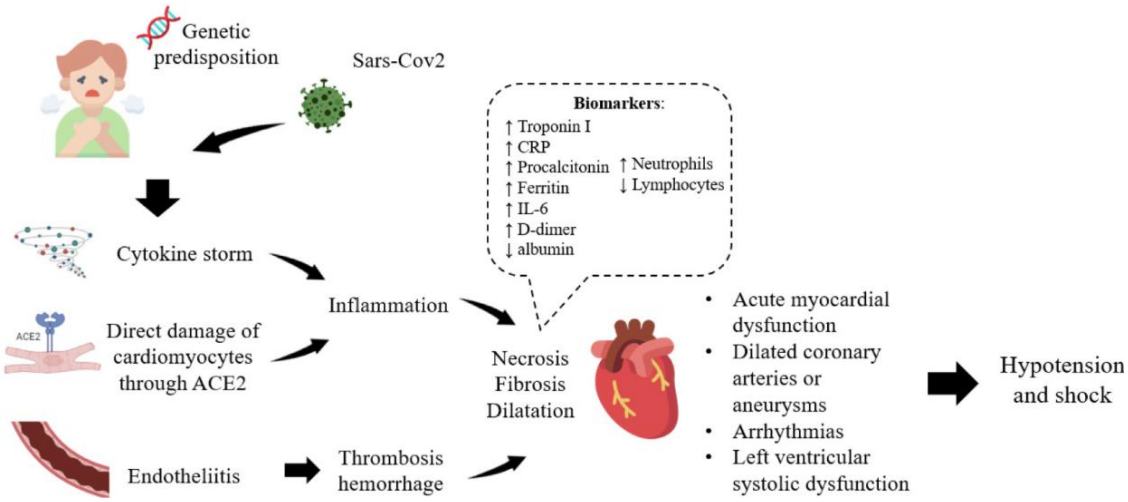

Figure 3. Mechanism of heart damage in COVID-19 (Vasichkina et al., 2023)

In adult patients with heart failure, guidelines have been developed for the evaluation and treatment of patients in the 4 stages of heart failure (A–D) that reflect disease progression. Stage A includes asymptomatic patients at high risk for heart failure without evidence of ventricular dysfunction. Stage B includes asymptomatic patients with evidence of ventricular dysfunction. Stage C includes symptomatic patients with ventricular dysfunction. Finally, stage D includes patients with refractory and symptomatic heart failure at rest (Hsu & Canter, 2010)

General health recommendations for adult patients with heart failure apply to the paediatric population. Recommendations include preventing smoking, alcohol consumption, and illegal drug use; hypertension management; control of metabolic syndrome; and sports. A 2004 consensus document outlined recommendations for physical activity and recreational sports participation in young patients with genetic cardiovascular disease, including DCM; however, the primary focus of these recommendations is the prevention of sudden death, which is rare in children with DCM. Specific exercise recommendations have not been proposed for children with DCM, but a recent pilot study confirmed the safety of exercise training in children with end-stage heart failure awaiting transplantation (Hsu & Canter, 2010)

In children at risk for heart failure, a population of childhood cancer survivors receiving anthracycline therapy and patients with Duchenne muscular dystrophy meet the criteria for stage A heart failure. Based on this small study, some physicians recommend the use of ACE inhibitor therapy in patients with dystrophy Duchenne muscle and ventricular function were normal (Fatema & Nure Ishrat, 2019)

The goal of medical therapy is to reduce the symptoms of congestive heart failure and thereby stop disease progression. The main goal of medical therapy is to control symptoms and prevent disease progression and complications, as well as improve the quality of life of symptomatic DCM patients. Drugs used in medical management include angiotensin-converting enzyme (ACE) inhibitors and beta-blocker drugs, with or without diuretics. The presence of heart failure is an indication for the use of diuretics. Vasodilators such as nitro-glycerine and ebiratide may be used. The safest drug that can be used in the paediatric age group with the fewest side effects is Carvedilol, a beta-adrenergic inhibitor with additional vasodilatory action. Inotropes are indicated if hypotension occurs. (Mallavarapu & Taksande, n.d.)

Dopamine and dobutamine must be administered intravenously to partially reverse chronic congestive heart failure and temporarily improve heart function. Dopamine and dobutamine are sympathomimetics which are included in the group of ionotropic drugs. However, excessive use of dopamine and dobutamine can cause arrhythmias and myocardial irritability. Milrinone is the drug of choice for the paediatric population. Amiodarone is a class III agent that extends the action potential and belongs to the group of antiarrhythmic drugs. It has been observed that amiodarone can be used in patients with poor myocardial excitation and contraction accompanied by symptomatic arrhythmias.

There are many nutrients beneficial to cardiovascular disease patients that can reverse myocardial dysfunction, including L-arginine, propionyl-L-carnitine, and coenzyme Q10. Recombinant human growth hormone produces an increase in left ventricular ejection fraction when administered as a subcutaneous injection or conventional therapy in children with DCM. However, growth hormone therapy has also been associated with increased somatic growth. Cardiac resynchronization therapy is used in the paediatric age group with advanced heart failure and conduction delay conditions. Heart transplantation remains the main option and is only reserved for advanced and extreme cases. Ventricular-assist devices have been shown to improve quality of life for patients in the absence of a donor heart or in patients awaiting transplantation and with advanced disease. (Lee et al., 2017)

Patients were treated with Furosemide 0.5mg/KgBW/12 hours; Captopril 0.3-0.5mg/KgBW/12 hours; Spironolactone 12.5mg/12hours; Digoxin 50 mcg/12 hours. After 2 weeks of treatment, an echocardiography evaluation was carried out with improvement in Dilated Cardiomyopathy and Pulmonary Hypertension. The patient is planned for outpatient treatment with continued oral therapy with the addition of Methylprednisolone 2mg/KgBW/day for up to 2 weeks of tapering off. During the control at the Referral Hospital, a radiological examination was carried out and the heart size returned to normal (CTR: 0.50).

DCM has one of the highest mortality rates among congenital and acquired heart diseases occurring in children. Various post-transplant complications can occur in children, including advanced kidney dysfunction, some even leading to dialysis or kidney transplantation. Infections are common during the post-transplant period, with bacterial infections most commonly occurring in the first month after transplantation and leading to death. Most mortality in children with DCM is due to progressive heart failure or complications from mechanical support or transplantation. In contrast to adults, the incidence of sudden death in children with cardiomyopathy is relatively low. (Javier Delmo et al., 2021)

In the last 2 decades, survival of children with DCM has improved, with some studies reporting 90% survival at 1 year, and 83% at 5 years after presentation. This increase is largely due to improved survival after heart transplantation and the availability of suitable ventricular assist devices to successfully bridge children with heart failure who are difficult to transplant. Recent reports from the UK and Ireland describe a 34% incidence of death or transplantation within one year in DCM patients with congestive heart failure. Although many children with acute decompensated heart failure and DCM have poor outcomes, there are many children (15%–30%) whose ventricular function can return to normal, and recovery is more likely in children with severe heart failure. Most babies with DCM die within their first year of life soon after they are diagnosed, with survival rates at one and five years of 79% and 61%, respectively. Most children die of heart failure in childhood, and other children whose ventricular function has not fully recovered may fail due to arrhythmias. (Godhiwala et al., 2021)

Conclusion

Cases of DCM associated with COVID-19 infection are rare. The diagnosis and treatment of DCM depends on the results of adequate examinations and requires subspecialist treatment. In the era after the COVID-19 pandemic, consideration needs to be given to COVID-19 infection in children with symptoms of respiratory tract infection.

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