Emerging Challenges: COVID-19-Related Cardiomyopathy and Intra-Cardiac Thrombosis in a Young Infant Patient: A Case Report

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ABSTRACT

This case report presents a rare and severe manifestation of COVID-19 infection in a young infant. The patient, a 9-month-old girl, presented with symptoms suggestive of respiratory distress and was subsequently diagnosed with COVID-19. During hospitalization, the patient developed supraventricular tachycardia (SVT), which responded to 2 doses of adenosine treatment. Echocardiography also identified cardiomyopathy with impaired cardiac function and intra-cardiac thrombosis of the left atrium, which posed significant clinical challenges. Prompt recognition and multidisciplinary management led to complete thrombus resolution and progressive recovery of cardiac function. This case highlights the importance of recognizing and managing cardiac complications in pediatric COVID-19 patients, including very young infants, and the need for early intervention to improve clinical outcomes.

Keywords: Coronavirus disease; Pediatric; supraventricular tachycardia; hypercoagulable state; heart failure; echocardiography

INTRODUCTION

Coronavirus disease (COVID-19) affects individuals of all ages to varying degrees of severity. While pediatric cases have generally been milder than adult cases, severe and atypical presentations can occur. COVID-19 in infants typically manifests as a mild respiratory or gastrointestinal infection[1]. The incidence of COVID-19 among children in various countries is estimated at 1–2% of all reported cases, and 20% of infected children are infants[1,2]. Most COVID-19-infected children and infants experience a mild infection; however, infants are more prone to a severe course of the disease than older children[3]. Numerous thromboembolic events associated with COVID-19 have been reported among adults and are associated with high mortality; fortunately, this complication is very rare among children and, particularly, infants[4,5]. Although cardiac involvement in pediatric COVID-19 has been described, reports of infants developing the combined triad of acute cardiomyopathy, supraventricular tachycardia, and intra-cardiac thrombosis are exceedingly rare. To our knowledge, no previously published reports describe these three complications in a previously healthy young infant. This case, therefore, fills an essential gap in the literature and underscores the importance of early cardiac evaluation in infants diagnosed with COVID-19.

CASE PRESENTATION

A previously healthy 9-month-old girl presented in 2024 to Al Qassimi Women's and Children's Hospital, United Arab Emirates (UAE), following a concerning illness that began 5 days before her admission. Her initial symptoms included an intermittent fever from 38–39°C, accompanied by reduced oral intake and diminished activity. The infant had been taken to a private hospital on 2 occasions, where she received symptomatic treatment. Her condition did not improve in response to these efforts.

On the fifth day of her illness, the child's parents observed that she began to experience frequent vomiting episodes, which occurred approximately 4 times. In addition, she appeared increasingly lethargic and had an elevated respiratory rate. Her parents were concerned about her deteriorating condition and decided to seek further medical attention, leading them to another private hospital, from which she was transferred to Al Qassimi Women's and Children's Hospital for further evaluation and care.

Birth, neonatal, developmental, immunization, and maternal histories were unremarkable. There was no family history of cardiac or hematological disorders, no known allergies, and no recent travel.

Course and hospital management: Upon arrival at the Pediatric Emergency Department of Al Qassimi Women's and Children's Hospital, the patient appeared alert but displayed signs of severe respiratory distress, characterized by head bobbing and chest wall retractions. Auscultation revealed equal bilateral air entry, with a slightly reduced right lower zone and no wheezing.

Vital signs: Heart rate (HR): 266 beats per minute (BPM); respiratory rate (RR): 66 breaths per minute; blood pressure (BP): 95/77 mmHg; oxygen saturation (SpO2): 97%; body weight: 9.1 kg; temperature (tympanic): 36.8°C.

Capillary refill time was measured at 2–3 seconds.

Upon connection to a 12-lead ECG, the patient exhibited narrow complex tachycardia suggestive of supraventricular tachycardia (SVT). This rhythm reverted to sinus rhythm after the administration of a second dose of adenosine at 0.2mg/kg. One fluid bolus of normal saline was also administered to address fluid-responsive shock.

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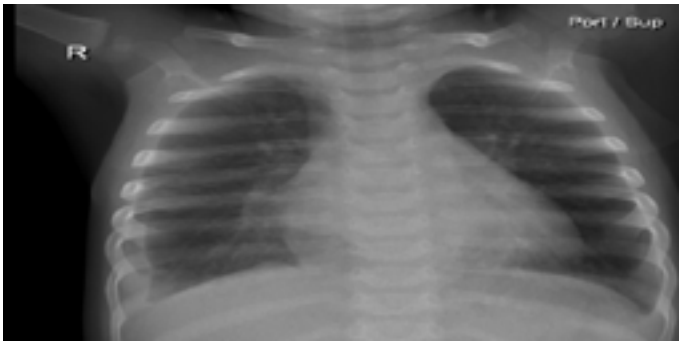

Figure 1. CXR at admission showed mild right pleural effusion.

Imaging findings: A chest X-ray (CXR) revealed mild right pleural effusion, Figure 1.

Nasopharyngeal swab: The patient's test for SARS-CoV-2 was positive, and the COVID-19 status check for both parents was positive for the mother and negative for the father.

Clinical impression: After stabilization in the emergency department, the patient was transferred to the Pediatric Intensive Care Unit (PICU) for further management. The clinical impression included suspected sepsis with fluid-responsive septic shock, possible myocarditis, SVT, and right pleural effusion.

Course in PICU: In the PICU, the patient remained alert, had no apparent dysmorphic features, was hemodynamically stable and well hydrated, had warm peripheries, had a HR of 140 BPM, had normal sinus rhythm, the peripheral and central pulses were palpable, capillary refill was <2 seconds, maintained a BP of 96/66 mmHg, and required no inotropic support. She exhibited respiratory distress on arrival with a RR of 35–40 breaths per minute on arrival, but settled down during her stay, maintaining a SpO2 of 95–99% on a 2L/min nasal cannula.

Cardiac evaluation: The patient's cardiac enzymes were elevated. Troponin I measured 49>>88>>160>>241 ng/L (Reference range [Ref]: 00–60 ng/L); pro-B-type natriuretic peptide: 15833 >20681 >24619 pg/mL (Ref: 0–125 pg/mL); total creatine kinase (CK): 253 IU/L (Ref: 26–192 IU/L); CK-MB: 6.5 µg/L (Ref: 0–3.6 µg/L); and lactate dehydrogenase: 1070 U/L (Ref: 81–234 U/L).

Bedside echocardiography was performed and revealed enlarged ventricles with decreased biventricular systolic function (ejection fraction: 40%) and notable dilation of the left atrium consistent with cardiomyopathy. In addition, a substantial thrombus-like mass measuring 20 x 9 mm was identified within the left atrium, attached to the left atrial appendage, Figure 2.

Valvular regurgitations: The patient had mild to moderate tricuspid regurgitation, mild to moderate mitral regurgitation, and trivial aortic regurgitation. A patent foramen ovale with left-to-right shunting was detected. The coronary arteries followed a normal route and calibration. Elevated pulmonary artery pressure raised concerns about the possibility of pulmonary hypertension.

Multiple organs exhibited symptoms reflecting an exaggerated inflammatory response (cytokine storm), which manifested as acute kidney injury that required conservative treatment. The patient's creatinine levels were 59.8 >> 28 µmol/L (Ref: 18–35 µmol/L) and urea was 11.7 > 5.5 mmol/L (Ref: 2.5–6.4 mmol/L).

Elevated liver enzymes: Aspartate transferase was 2340 > 1601 > 616 U/L (Ref: 15–37 U/L), and alanine transaminase was 1502 > 1241 > 836 U/L (Ref: 15–37 U/L).

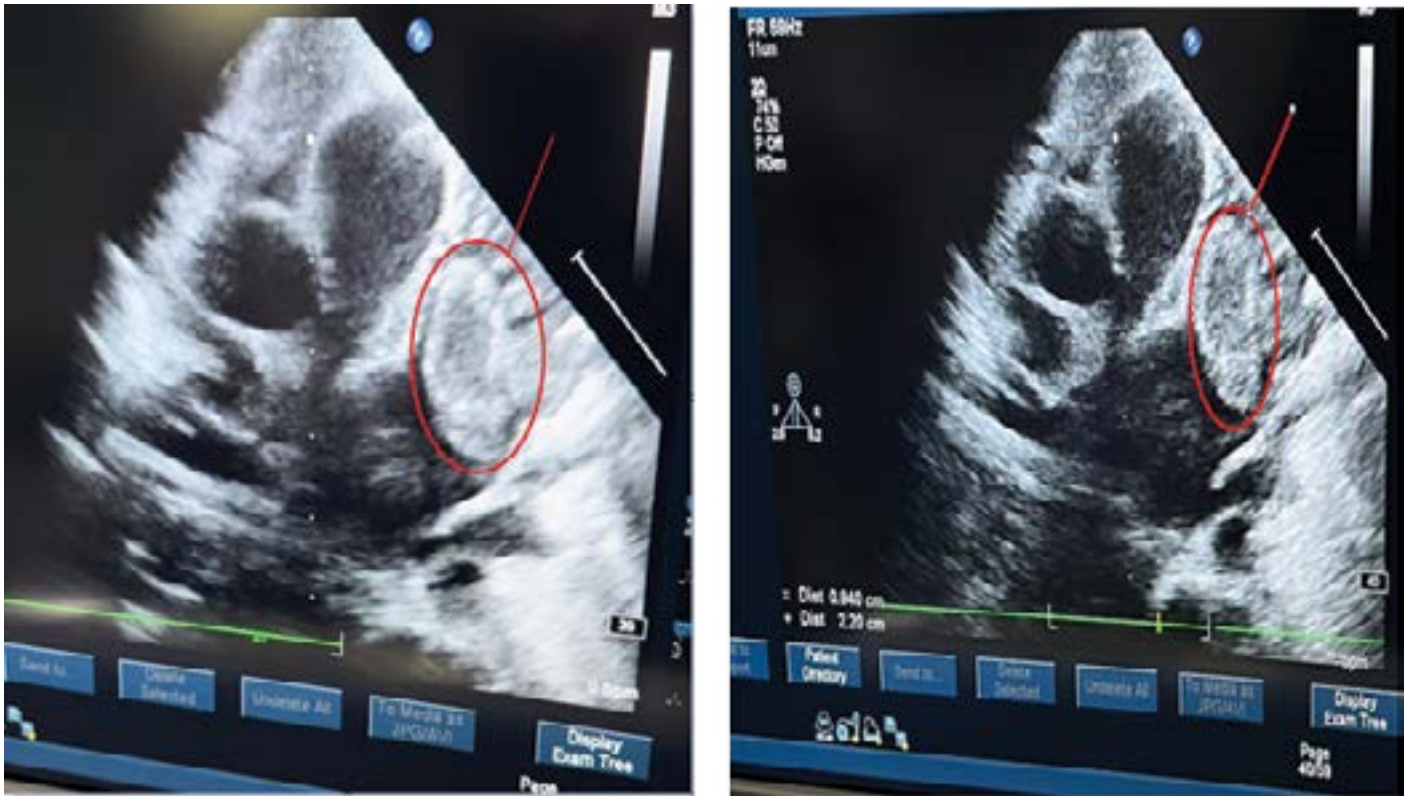

Figure 2. Echocardiograms showing the dilated left atrium and large (20 x 9 mm) thrombus-like mass in the left atrium, attached to the left atrial appendage (red cycles).

Table 1. Key laboratory parameters during hospitalization

Parameter	First measurement	Second measurement	Third measurement
Troponin I (ng/L)	49	88	160
Pro-BNP (pg/mL)	15,833	20,681	24,619
WBC (×10⁹/L)	20.22	7.99	13
Creatinine (µmol/L)	59.8	28	-
Urea (mmol/L)	11.7	5.5	-
AST (U/L)	2340	1601	616
ALT (U/L)	1502	1241	836
D-dimer (mg/L)	>8.17	-	-
Fibrinogen (g/L)	<0.89	-	-
INR	1.64	-	-
APTT (sec)	32.9	-	-
CK (IU/L)	253	-	-
CK-MB (µ/L)	6.5	-	-
LDH (U/L)	1070	-	-

Other testing revealed low hemoglobin (9.5 g/dL; Ref: 11.1–13.1 g/dL), low fibrinogen (<0.89 g/L; Ref: 1.8–3.6 g/L), high D-dimer (>8.17 mg/L; Ref: 0–0.5 mg/L), high INR (1.64 seconds; Ref: 0.8–1.29 seconds), a high prothrombin time (>100: Ref: 12–15 seconds); and a normal activated partial thromboplastin time (APTT; 32.9 seconds; Ref: 25–35 seconds).

The patient remained afebrile from admission through her stay, with no skin rash; however, her labs showed high inflammatory markers, including leucocytes (20.22 >> 7.99 >> 13), lymphocytes (52%), procalcitonin (0.3 >> 0.29 ng/mL; Ref: 0–0.1 ng/mL), IL-6 (7.2 pg/mL; Ref: ≤4.4 pg/mL). Therefore, samples of blood and urine were collected and sent for culture while the patient was started on an empirical dose of the antibiotic ceftriaxone, which was stopped after 23 days when the culture results were negative. Screening for other viruses, including adenovirus PCR, Coxsackie virus IgM, herpes 6 PCR, and herpes simplex virus PCR, was negative.

A thrombophilia evaluation was performed to rule out underlying prothrombotic conditions. Protein C, protein S, and antithrombin III levels were within age-appropriate ranges. The antiphospholipid antibody panel was negative Table 1.

Assessment: COVID-19 virus infection involving multiple organs, reflecting an exaggerated inflammatory response (cytokine storm). Otherwise, hemodynamically stable, requiring conventional oxygen by nasal cannula without increasing demand.

PICU management: The patient was admitted to the PICU for close monitoring and management by a multidisciplinary team (MDT), including:

- COVID-19 management: Supportive care, including oxygen supplementation and fluids, was initiated. Antiviral therapy with remdesivir was administered according to the *UAE National Guidelines for COVID-19 Management Version 9*.
- Cardiomyopathy management: The patient received inotropic support and was started on a milrinone infusion to optimize cardiac function, which was replaced by anti-heart failure medications (captopril and furosemide) after 2 days. As the patient's cardiac enzymes were high, which suggests carditis, the cardiologist advised intravenous immunoglobulin, of which the patient received a total of 2 g/kg.
- Anticoagulation: Anticoagulation therapy with unfractionated heparin was initiated to manage the intra-cardiac thrombosis, starting with a loading dose of 50 IU/kg and continuing with a maintenance dose of 28 IU/kg/h and a follow-up test of APTT according to the heparin infusion protocol (target APTT: 60–80 seconds). Anticoagulation in infants presents significant challenges due to developmental differences in hemostasis, narrow therapeutic ranges, and increased bleeding risk, particularly in the setting of hepatic dysfunction. Close monitoring was maintained throughout, and no bleeding complications occurred. A follow-up echocardiogram after 3 days of treatment revealed the complete resolution of the left atrium thrombus, Figure 3.

Therefore, the heparin infusion was stopped, and the patient started on an antiplatelet dose of aspirin.

- Serial follow-up echocardiograms were performed to assess the patient's cardiac function, which showed minimal improvement with no further rhythm disturbance.

Outcome: Despite the severity of the presentation, the patient showed full resolution of the left atrium thrombus and gradual cardiac function improvement with treatment. Serial echocardiograms demonstrated

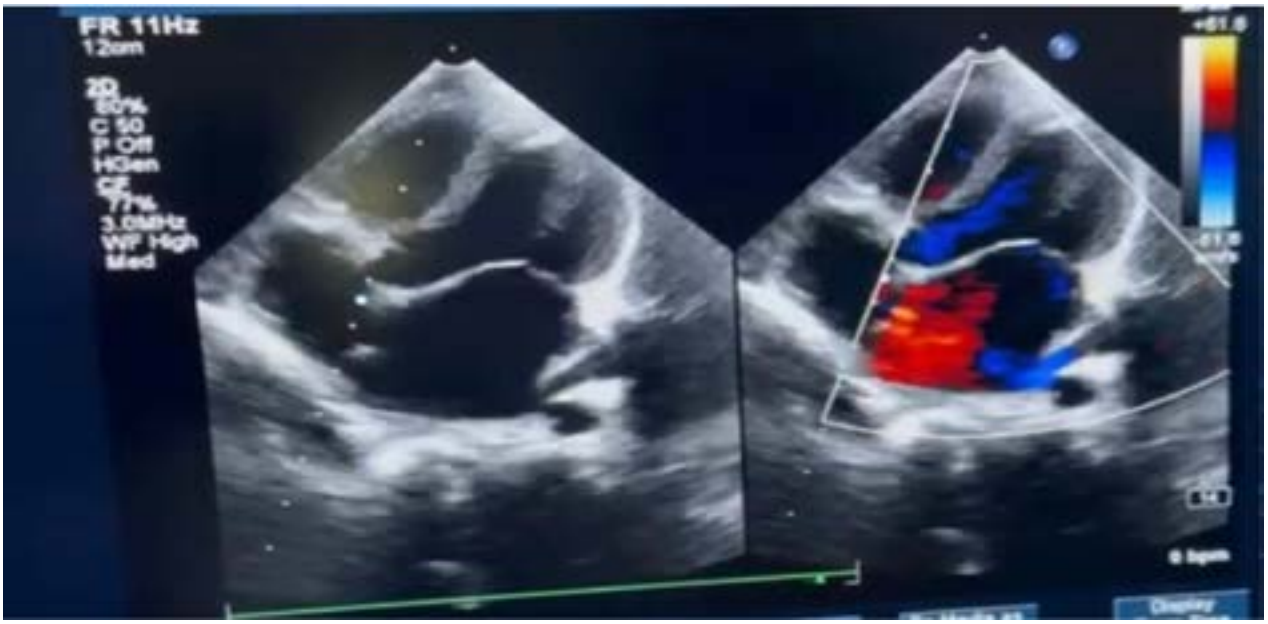

Figure 3. Repeat echocardiograms showed resolution of the left atrial thrombus 72 hours after starting the therapeutic dose of heparin infusion.

an increasing ejection fraction and no recurrence of intra-cardiac thrombus. The patient was weaned off inotropic support and discharged with appropriate medical therapy for cardiomyopathy. Follow-up appointments were scheduled to monitor her cardiac function and thrombus recurrence.

Patient consent was obtained from the guardian for publication purposes. This study protocol was reviewed and approved by the Dubai Research Ethics Committee at the Ministry of Health and Prevention, United Arab Emirates (MOHAP/DXB-REC / D.D.D /No.216/ 2024).

DISCUSSION

In this report, we describe a case of COVID-19 infection in a young infant who developed cardiomyopathy, SVT, and intra-cardiac thrombosis. Cardiovascular disease is the most common comorbidity in adult COVID-19 patients, and it is related to the disease's severity, comorbidities, and the existence of a hypercoagulable state[6-8]. However, cardiovascular involvement in children is uncommon.[9] COVID-19 induces direct and indirect damage to the heart, resulting in arrhythmias and myocarditis with a high inflammatory burden. In addition, in children, COVID-19 induces cardiac damage that leads to myocarditis, coagulation abnormalities, venous thromboembolism, Takotsubo cardiomyopathy, Kawasaki-like disease, and multisystem inflammatory syndrome[6]. This myocardial injury is explained by an imbalance of oxygen supply/demand, direct viral myocardial invasion, cytokine release, the rupture of coronary plaque, endothelial dysfunction, microvascular thrombosis, and adrenergic stress. The thromboembolic events are related to a hypercoagulability state that is distinctive of COVID-19[4,10,11] and seen in children presenting with multisystem inflammatory syndrome. In the literature, cases of adult COVID-19 patients who developed intracardiac thrombosis are reported, as in Samia B. et al., who reported a case of a 38-year-old male who developed a right atrial thrombosis associated with a pulmonary embolism after COVID-19 infection[10] and Philip et al., who identified adult COVID-19 patients with left ventricular thrombi, although most of these had comorbidities and risk factors for cardiac diseases or thrombosis, so these complications could be related to a post-COVID condition or a primary cause[12]. The authors highlighted these adult cases to provide insight into COVID-19-related thrombosis, given the limited pediatric-specific literature, especially in infants, making this case particularly informative. Children are at a very low risk of severe disease and death from COVID-19[13] and the authors found no published case of COVID-19-related cardiomyopathy, SVT, or intra-cardiac thrombosis in young infants. So, in rare instances like this, where patients develop these complications without comorbidities or risk factors, heart involvement should be expected and an MDT should examine the patient carefully to improve patient outcomes. To mitigate the risk of intra-cardiac thrombi and subsequent embolic events, anticoagulation measures, such as heparin, enoxaparin, warfarin, and/or antiplatelet agents are recommended[14]. Early recognition and intervention are crucial to optimize outcomes when evaluating and managing pediatric COVID-19 patients.

CONCLUSION

The authors present a rare case of COVID-19 infection associated with cardiomyopathy, SVT, and intra-cardiac thrombosis in a young infant. This case underscores the importance of vigilance to recognize and manage cardiac complications in pediatric COVID-19 patients, even very young infants, and the need for early intervention to improve clinical outcomes. Early cardiac evaluation, including echocardiography, should be considered for infants presenting with moderate or severe COVID-19 symptoms. Serial monitoring of cardiac enzymes and coagulation markers enables early detection of evolving cardiomyopathy or thrombus formation. Prompt multidisciplinary management is recommended to monitor cardiac recovery and prevent recurrence. Further studies are warranted to better understand the pathophysiology and optimal management of such cases.

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Potential Conflicts of Interest: None

Competing Interest: None

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