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MINI-FOCUS ISSUE: CORONARY INTERVENTIONS

INTERMEDIATE

CASE REPORT: CLINICAL CASE

Successful Intervention for a Thrombosed Giant Coronary Artery Aneurysm in Multisystem Inflammatory Syndrome in Children

Timothy Stidham, MD,[a] James McKee, MD,[b] Joshua Vogt, MD,[a] Austine Kuder Siomos, MD[c]

ABSTRACT

We report the case of a 13-year-old who presented with an ST-segment elevation myocardial infarction caused by a thrombotic occlusion of an aneurysmal left anterior descending coronary artery. Our patient was diagnosed and treated for multisystem inflammatory syndrome in children and underwent successful balloon angioplasty and aspiration thrombectomy.(**Level of Difficulty: Intermediate.**) (J Am Coll Cardiol Case Rep 2022;4:945–949) © 2022 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

HISTORY OF PRESENTATION

A 13-year-old boy presented to a critical access hospital with 1 week of upper respiratory symptoms and 1 day of fever, nausea, and vomiting. He had been seen 4 days previously at his primary care provider's office and had positive results on an antigen test for severe acute respiratory syndrome-coronavirus-2 (SARS-CoV-2).

LEARNING OBJECTIVES

- To identify a myocardial infarction in a pediatric patient.
- To discuss treatment of myocardial infarction in the pediatric population.
- To promote communication between specialties to care for a patient in a rare acute emergency.

PAST MEDICAL HISTORY

The patient had a history of obesity and gingivitis. He was eligible for COVID-19 vaccination but was not vaccinated.

DIFFERENTIAL DIAGNOSIS

The differential included acute COVID-19, acute gastrointestinal viral illness, multisystem inflammatory syndrome in children (MIS-C), pulmonary embolism, and myocardial infarction (MI).

INVESTIGATIONS

On initial presentation the patient's weight was 113 kg, height: 178 cm, and body mass index: 35.9 kg/m^2. The patient's temperature was 36.4 °C, heart rate 78 beats/min, respiratory rate 16 breaths/min, oxygen saturation 99%, blood pressure 115/77 mm Hg. He had

From the [a]Logan Health, Kalispell, Montana, USA; [b]Northwest Imaging PC, Kalispell, Montana, USA; and [c]Logan Health Heart & Lung, Kalispell, Montana, USA.

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ABBREVIATIONS AND ACRONYMS

CT = computed tomography

ECG = electrocardiogram

LAD = left anterior descending coronary artery

MIS-C = multisystem inflammatory syndrome in children

MI = myocardial infarction

RCA = right coronary artery

STEMI = ST-segment elevation myocardial infarction

TIMI = Thrombolysis In Myocardial Infarction

moderate tenderness to palpation diffusely over his abdomen. His cardiac examination results were unremarkable. He had diminished breath sounds on auscultation at the bilateral lower lung fields. Initial abnormal laboratory results included troponin I ultrasensitive (0.36 ng/mL; normal range: <0.034 ng/mL), B-type natriuretic peptide (337 pg/mL; normal range: <100 pg/mL), D-dimer (721 ng/mL; normal range: <600 ng/mL), erythrocyte sedimentation rate (19 mm per hour; normal range: <15 mm/h).

During transfer, the patient developed chest pain. His troponin rapidly increased to a peak of 35.7 ng/mL, 1,000 × the upper limit of normal and 300 × the acute MI threshold of 0.12 ng/mL (**Figure 1**).

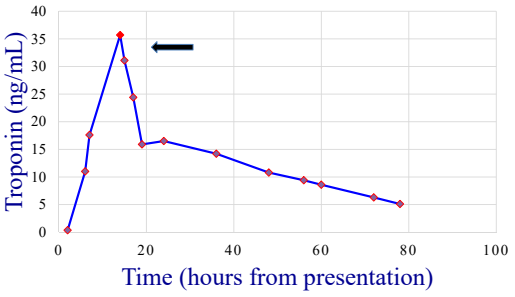
The initial electrocardiogram (ECG) demonstrated ST-segment elevation in leads I and aVL and ST-segment depression in II, III, aVF, V_3, and V_4 (**Figure 2**).

Initial echocardiogram demonstrated regional wall abnormalities with hypokinesis of the mid-septum and the apex and an estimated ejection fraction of 35% (Videos 1 and 2). A computed tomography (CT) angiogram demonstrated severe right coronary artery (RCA) dilation and occlusion of the mid-left anterior descending (LAD) coronary artery (**Figure 3**).

MANAGEMENT

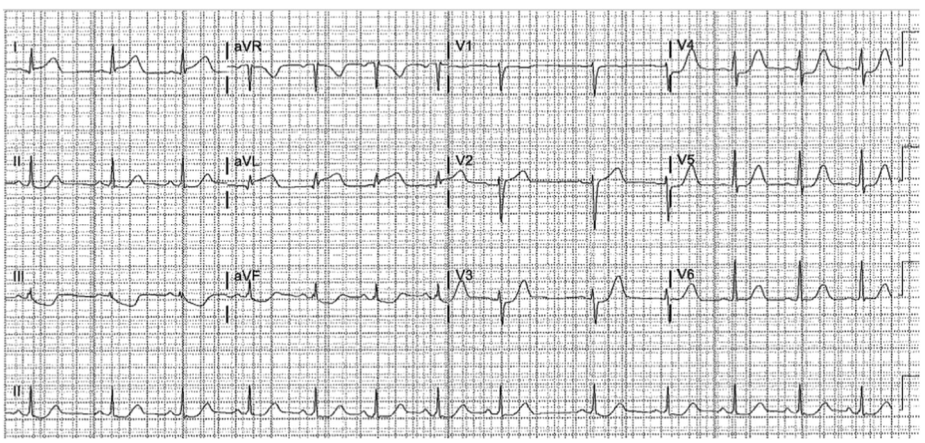
Intravenous cangrelor and heparin were started, and the patient was taken for emergent cardiac catheterization (**Figure 3,** Videos 3 to 6). Predilation was performed with a 2.5-mm x 12-mm semicompliant balloon at 6 atm and 8 atm throughout the proximal and mid-LAD, restoring flow. Aspiration thrombectomy was then performed with 2 passes of a Pronto (Teleflex) catheter. Thrombotic material was aspirated. Intracatheter nitroprusside was given. Balloon angioplasty was performed with a 3.0-mm × 15-mm semicompliant balloon throughout to 8 atm. Given the massive thrombus burden in the aneurysmal segment, post-dilation was performed with a 5.0 × 15-mm semicompliant balloon throughout the proximal and mid-LAD to 6 atm and 10 atm, serially. Final angiography demonstrated TIMI

FIGURE 1 Troponin Trend



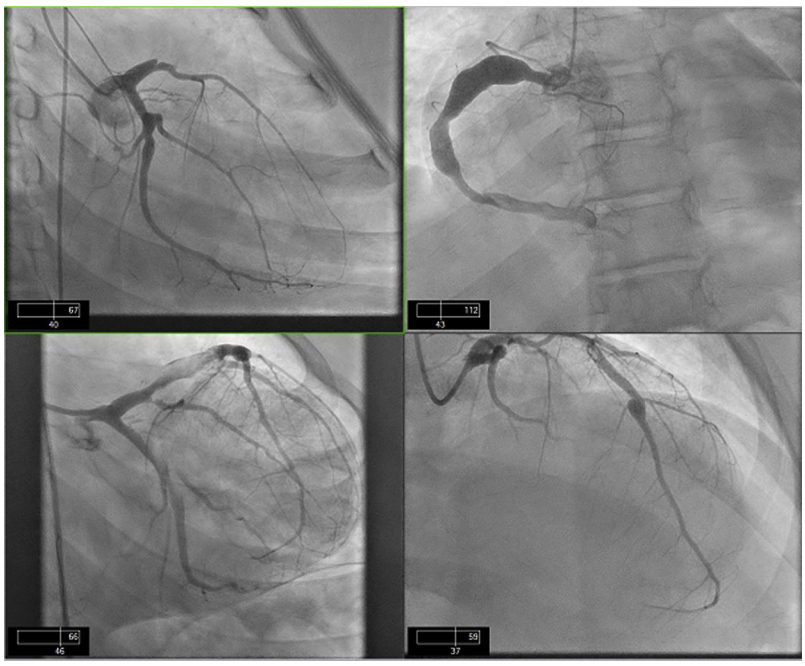
Troponin values from presentation until the patient stabilized. The time on the x axis is in hours. The troponin on the y axis is in nanograms per milliliter. The upper limit of normal for troponin using this scale is 0.012 ng/mL. The **arrow** delineates the time at which patient underwent catheter intervention.

FIGURE 2 Presenting Electrocardiogram



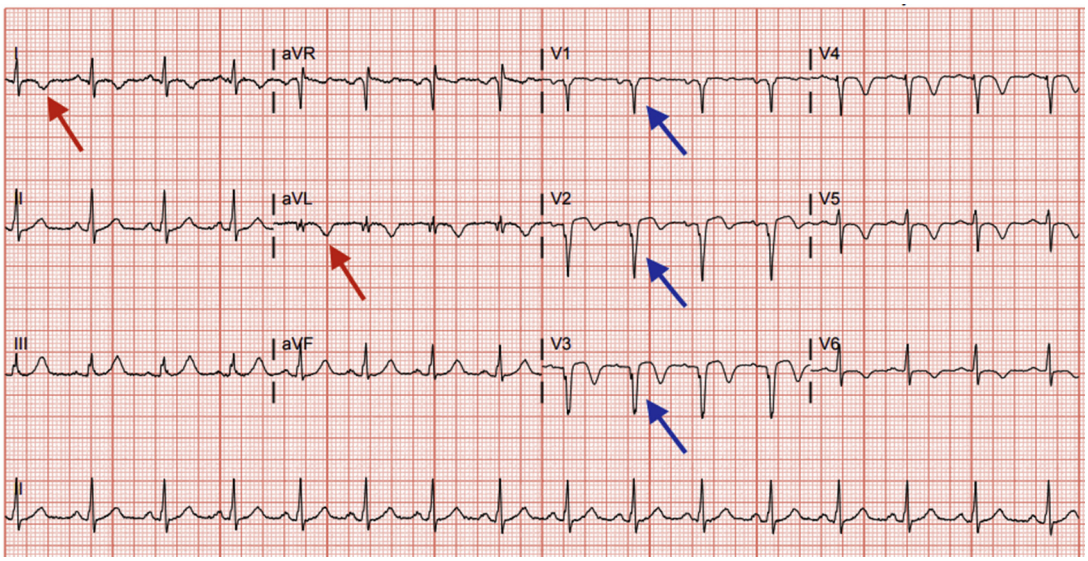
Electrocardiogram on admission to the pediatric intensive care unit demonstrating ST-segment elevation in leads I and aVL and ST-segment depression in II, III, aVF, V_3, and V_4.

FIGURE 3 Coronary Angiograms



Selective left coronary artery angiogram using a 6-F catheter in the caudal projection **(top left)**, demonstrating acute occlusion of the proximal left anterior descending coronary artery; selective right coronary artery angiogram using a JR4 5-F catheter **(top right)**. Post-balloon angioplasty and aspiration thrombectomy left coronary artery in the caudal projection with a 6-F catheter **(bottom right)** and cranial projection **(bottom left)**. There is dilation of the left main coronary artery, proximal, and mid-left anterior descending coronary artery with early aneurysm formation in the mid-left circumflex coronary artery.

FIGURE 4 Electrocardiogram Demonstrating Final Infarct Pattern



Electrocardiogram on discharge demonstrating sinus rhythm with T-wave inversion in I and aVL **(orange arrows)**, Q waves in V_1 to V_3 **(blue arrows)**, consistent with an anteroseptal infarct.

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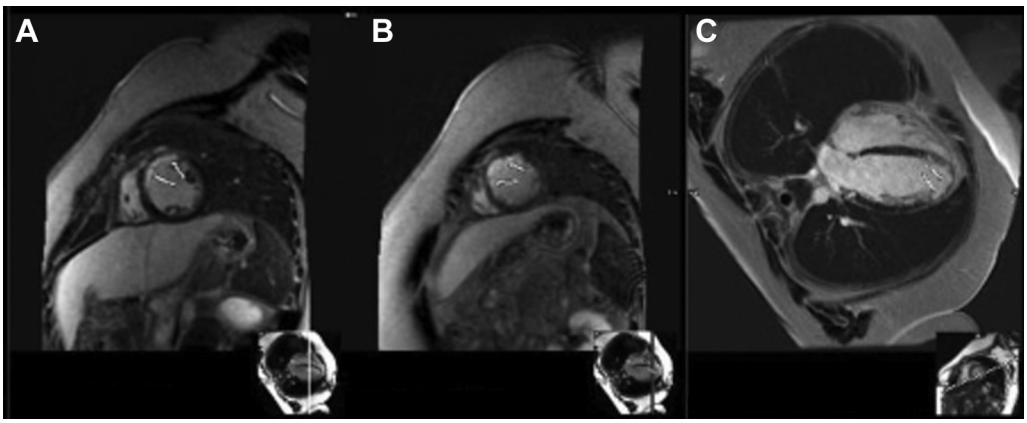
FIGURE 5 Cardiac Magnetic Resonance Image



Magnetic resonance image 4 months from myocardial infarction without and with intravenous gadolinium. **(A)** Short axis left ventricle at the level of the papillary muscles. **(B)** Apical short axis of the left ventricle. **(C)** Four-chamber; delayed subendocardial enhancement involving the anterior wall and anterior septum of the left ventricle, spanning basal to apical segments.

(Thrombolysis In Myocardial Infarction) flow grade 3 throughout (Video 3). The time from initial ST-segment elevation ECG to reperfusion was 12 hours. Myocardial salvage was achieved based on resolution of ST-segment elevation, resolution of chest pain, restoration of TIMI coronary flow grade 3, and post-hospitalization cardiac magnetic resonance imaging (MRI) demonstrating nontransmural infarction, as described in follow-up.

Post-procedure antithrombotic therapy was continued with aspirin, cangrelor, and intravenous unfractionated heparin. He was transitioned to clopidogrel and aspirin for antiplatelet coverage. Management of MIS-C included intravenous immunoglobulin, infliximab, and methylprednisolone. The patient had clinically significant epistaxis and required operative management with nasal endoscopy, cauterization, and silicone splints. Aspirin was stopped, and he was continued on clopidogrel and enoxaparin.

He was started on medical therapy for ischemic cardiomyopathy including carvedilol, lisinopril, and spironolactone. Repeat CT angiogram demonstrated good perfusion through the LAD and persistence of giant aneurysms of the RCA and LAD (**Figure 3**). Electrocardiogram on discharge demonstrated a final infarct pattern of T-wave inversion in I and aVL, Q waves in V_1 to V_3, consistent with an anteroseptal infarct (**Figure 4**). Because of an ejection fraction below 35%, the patient was discharged with a wearable defibrillator.

DISCUSSION

We describe the first reported case of successful aspiration thrombectomy to treat thrombosis of an aneurysmal coronary artery in a pediatric patient with MIS-C. MIS-C was first recognized when a cluster of Kawasaki-like cases was described in Northern Italy and Southeast England.[1] MIS-C is associated with myocardial dysfunction and coronary artery changes.[2] Our patient met the Centers for Disease Control and Prevention (CDC) criteria for MIS-C.[3] His presenting troponin could be consistent with a primary diagnosis of MIS-C, in which case treatment would have been medical with immunomodulation.[4] The rapid rate of rise of troponin in our case prompted a change in the differential diagnosis. Importantly, our patient's troponin peaked well above the average troponin level for pediatric patients with MIS-C who are admitted to intensive care units.[5] Disconcertingly, this initial diagnosis of MIS-C and subsequent thrombotic MI could have been missed in our patient, who presented without chest pain.

Giant coronary artery aneurysms have been described in MIS-C.[6] Kawasaki disease studies have demonstrated that at least 1 MI occurs in 23% to 26%

of patients with giant aneurysms by 20 years after the onset of Kawasaki disease.[7]

The standard of care for adult MI is a door-to-intervention time of <90 minutes. Guidelines for management of pediatric myocardial infarction were first published in 2020.[8] If STEMI is evident on ECG and percutaneous coronary intervention is not possible or available, tissue plasminogen activator and unfractionated heparin should be started. This may also be done if expected time to the catheterization laboratory is more than 90 minutes.

FOLLOW-UP

Four months from presentation, the patient underwent cardiac MRI, demonstrating delayed subendocardial enhancement involving the anterior wall and anterior septum of the left ventricle, spanning basal to apical segments (**Figure 5**). His ejection fraction by MRI was 42%. His echocardiogram demonstrated global mildly decreased ventricular systolic function with an ejection fraction of 45% (Videos 7 and 8). The patient was active in cardiac rehabilitation and was attending school.

CONCLUSIONS

Our patient had MIS-C and giant coronary artery aneurysms after being infected with SARS-CoV-2. A prothrombotic inflammatory state resulted in acute coronary artery thrombosis. We propose a high index of suspicion for acute MI in the case of a patient with MIS-C or severe COVID and rapidly increasing troponin. In any case of suspected MIS-C a screening ECG should be performed to exclude an acute coronary syndrome. This rare case demonstrates the necessity of early multidisciplinary collaboration to salvage myocardium in a young patient.

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The authors have reported that they have no relationships relevant to the contents of this paper to disclose.

ADDRESS FOR CORRESPONDENCE: Dr Austine Kuder Siomos, Logan Health Heart & Lung, 350 Heritage Way, Suite 2100, Kalispell, Montana 59901, USA. E-mail: austine.kuder@gmail.com.

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KEY WORDS acute heart failure, anticoagulation, antiplatelet, myocardial infarction, percutaneous coronary intervention, thrombosis

APPENDIX For supplemental videos, please see the online version of this paper.