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Commentary

Cardiogenic shock temporally associated with COVID-19 vaccination after prior COVID-19 infection: A case report

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ABSTRACT

The introduction of coronavirus 2019 (COVID-19) vaccination has been an integral force in stopping the spread of COVID-19 across the globe. While reported side effects of vaccination have predominantly been mild, in the last year reports have emerged of myocarditis following the BNT162b2 (Pfizer-BioNtech) and mRNA-1273 (Moderna) vaccinations. The adolescent and young adult population have been the population most reported, with over 1000 cases under review by the Centers for Disease Control (CDC) since April 2021. Here we report a case of a previously healthy 21-year-old male who developed Multisystem Inflammatory Syndrome in Adults (MIS-A) and following the second dose of the Pfizer-BioNtech vaccine. The young male initially presented with fever, leukocytosis with high neutrophil-lymphocyte ratio, severe cardiac illness, and positive COVID-19 nucleocapsid serology, consistent with MIS-A diagnosis. His case was complicated by cardiogenic shock, requiring brief venoarterial extracorporeal membrane oxygenation (VA-ECMO) support. While this report does not detract from the overwhelming benefit of vaccination from COVID-19, clinicians should be aware of this possible relationship in the future.

1. Introduction

Coronavirus disease-2019 (COVID-19) quickly spread worldwide in 2020, becoming a global pandemic causing significant morbidity and mortality. The severe, acute respiratory syndrome caused by COVID-19 has had devastating effects, with over 5 million deaths reported [1]. The disease burden has predominantly been in adults, with children and adolescents typically experiencing mild illness without hospital admission [2]. During early May 2020 however, reports emerged from the United Kingdom of children requiring admission to intensive care units due to an unexplained Kawasaki-like disease syndrome now known as Multisystem Inflammatory Syndrome in Children (MIS-C) [2,3]. A similar condition has also been reported as a rare complication of COVID-19 in adults, named Multisystem Inflammatory Syndrome in Adults (MIS-A) [2,22–23]. While the incidence of MIS-C/A remains low, the severity of the disease can be significant, with many patients requiring inotropic and ventilatory support [2].

The prompt development of several vaccines including the BNT162b2 (Pfizer-BioNtech) and mRNA-1273 (Moderna) vaccinations, have been key to preventing further severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2) infection. At the time of this report (January 2022), there have been over 9 billion doses of vaccines administered. Along with increasing rates of vaccination, since April 2021, there have been case reports and case series emerging of young adults (mostly male adolescents 16 years or older) with biomarker and cardiac magnetic resonance imaging (cMRI) confirmed myocarditis within 2–4 days of receiving either the BNT162b2 or mRNA-1273 vaccines [12–16,20,24]. The temporal association of COVID-19 vaccination with onset of myocarditis suggests an etiological link, but the pathological mechanism remains unclear. Of note, these reported cases are largely COVID-19 RT-PCR negative and nonreactive for nucleocapsid protein (anti N Ab Negative), indicating that this is a process separate from native infection [12–16,24].

Here we present an unusual case of a MIS-A like disease in a young adult male after a second dose of the BNT162b2 vaccine administered after an asymptomatic COVID-19 infection.

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2. Case report

2.1. Clinical presentation

A 21-year-old previously healthy African American male presented to the emergency department with intractable nausea and vomiting. He received his Pfizer- BioNtech vaccine on 05/11/2021, and developed fatigue, subjective fever, night sweats and myalgias the day after. Within three days, he developed a non-pruritic confluent red macular rash on his hands bilaterally that spread to his arms, legs then to his torso. He also reported nausea, with non-bilious, non-bloody emesis, and diarrhea. He had poor appetite, and minimal oral intake, and was reportedly unable to keep anything down, so he was brought to the emergency room by his family seven days after the onset of symptoms.

In the emergency room, the patient was found to be hypotensive (95/56 mmHg) and tachycardic (139 bpm) and developed fever with temperature of 38 °C. The patient's laboratory findings were notable for leukocytosis (white blood cell count 35,000) with neutrophilia (absolute neutrophil count 30.92) and lymphopenia (lymphocyte count 0.99), and thrombocytopenia (platelet count 118,000). Multiple inflammatory markers were elevated including C-reactive protein (28 mg/dL), erythrocyte sedimentation rate (85 mm/h), D-dimer (2053 ng/mL) and lactate dehydrogenase (528 U/L). Blood cultures were drawn, and the patient was started on empiric antibiotics and fluid resuscitation for presumed sepsis.

An electrocardiogram (ECG) showed sinus tachycardia with ST segment elevation in anterior leads V2, V3 and reciprocal depressions in the inferior leads II, III, and aVF (Fig. 1). Troponin I was elevated at 1.94, and BNP was elevated at 785. Given the patient's young age, and clinical presentation of viral prodrome, at this time it was suspected that the elevated troponins were attributable to demand ischemia, as opposed to coronary arterial disease. A bedside echocardiogram was performed revealing a reduced left ventricular (LV) ejection fraction at 35–40% as well as reduced right ventricular systolic function. Despite fluid resuscitation, the patient remained hemodynamically unstable and was transferred to the intensive care unit (ICU) for further management.

2.2. Initiation of venoarterial extracorporeal membrane oxygenation (VA-ECMO)

An expedited cardiac workup was completed within 24 h of ICU admission with a cardiac MRI revealing severe biventricular systolic dysfunction with global hypokinesis (LV ejection fraction 11%, RV ejection fraction 12%) and normal ventricular size. Cardiac MRI did not show elevation of myocardial T2 signal or hyperenhancement to suggest myocardial edema, inflammation, or fibrosis (Fig. 2). Cardiac MRI was also significant for no evidence of myocardial infarction, and left heart catheterization revealed normal coronary arteries. Right heart catheterization was done with evidence of severe biventricular failure, with right atrial and right end diastolic pressure of 13 mmHg, pulmonary capillary wedge pressure of 25 mmHg, and cardiac index 1.6 L/min/m2. During the right heart catheterization procedure, the patient had an episode of sustained ventricular tachycardia, requiring termination with synchronized cardioversion. Given severe cardiac dysfunction and continued hemodynamic instability evidenced by sinus tachycardia and elevated lactate despite appropriate fluid resuscitation, the patient was initiated on VA-ECMO for management of cardiogenic shock.

2.3. Recovery and discharge from ICU

After initiation of VA-ECMO, the patient continued to require blood pressure support with epinephrine and inotropic support with milrinone for several days. He was continued on broad spectrum antibiotics (meropenem 1 g q8 hr and vancomycin 1.5 g q12 hr) and started on methylprednisolone. Diagnostic workup was extensive for the patient's presentation including infectious, autoimmune, and toxicology studies (Table 1). Stool PCR resulted positive for Campylobacter and serum studies resulted positive for Coxsackie A IgG. As there was concern for MIS-C/A, the patient was tested for COVID-19 with a PCR test twice, each time resulting negative. He was then tested for SARS-CoV-2 Nucleocapsid IgG which resulted positive.

While on ECMO, the patient required pulmonary arterial catheter guided diuresis with IV loop diuretics. The patient had multiple episodes of atrial fibrillation with rapid ventricular response that converted to normal sinus rhythm after a brief amiodarone infusion. An echocardiogram was performed six days after initiation of ECMO, which revealed a LVEF of 40% and a normal right ventricular function. ECMO decannulation was successfully performed three days later, and patient was weaned off all vasopressor medications. A cardiac MRI was performed the day after transferring out of the ICU, which showed further improvement in cardiac function with a LVEF of 60% with normal left ventricular size. As before, there was no evidence of myocarditis on repeat cardiac MRI. After seven days total, meropenem, vancomycin, and high dose steroids were discontinued. The patient was discharged home the next day with scheduled follow up with outpatient Cardiology.

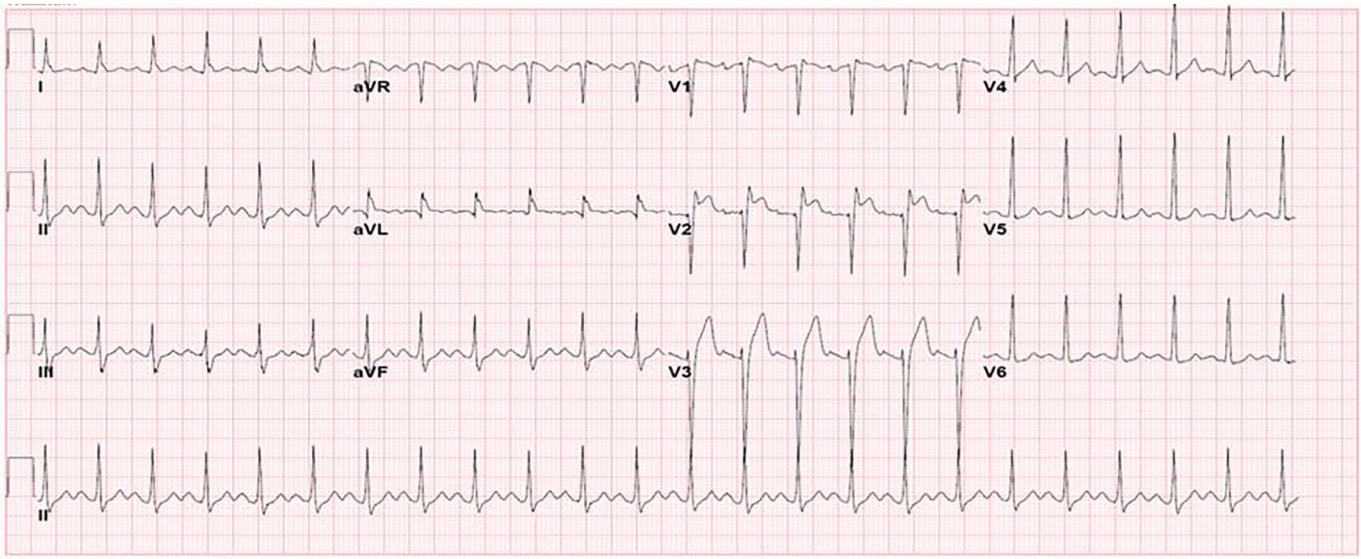

Fig. 1. Initial electrocardiogram. Sinus tachycardia. ST elevations in V2 and V3.

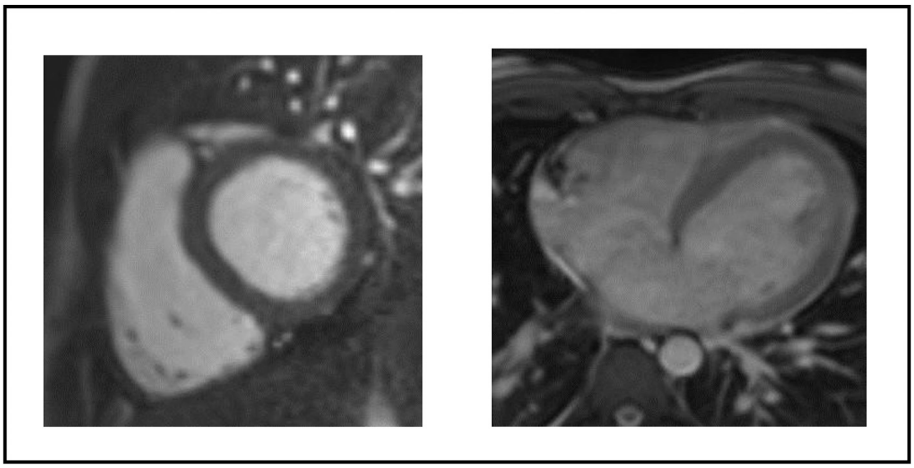

Fig. 2. Cardiac MRI on admission to ICU, without evidence of early gadolinium enhancement.

Table 1
Exploratory studies for diagnostic workup and investigation.

Laboratory data	
Variable	Results
Blood cultures on admission	No Growth
Syphilis Total antibody (Ab)	Negative
Coxsackie A Ab	Positive IgG
Coxsackie B Ab	Negative
Cytomegalovirus	Negative
Hepatitis C (Ab)	Negative
Legionella Antigen (Ag)	Negative
***Streptococcus pneumoniae* Ag**	Negative
HIV (Ag/Ab)	Negative
Stool PCR for Gastrointestinal pathogens	Positive for *Campylobacter* species
Salmonella species	
Vibrio species	
Shigella species	
Campylobacter species	
Yersinia enterocolitica	
Shiga toxin	
Rotavirus A	
Urine Toxicology Screen	Positive for Cannabinoid
Amphetamine	
Barbiturate	
Benzodiazepines	
Cannabinoid	
Cocaine	
Fentanyl	
Methadone	
Opiates	
Oxycodone	
Autoimmune Screen	Negative
Antinuclear antibody	
Rheumatoid factor	
Antineutrophil cytoplasmic antibodies	
TSH	1.95 μIU/mL
SARS-CoV-2 Nucleocapsid IgG Antibody	Positive
RT-PCR for SARS-CoV-2	
Day 1 of admission	Negative
Day 7 of admission	Negative

3. Discussion

Here, we present a case of cardiogenic shock temporally related to BNT162b2 vaccination in a patient with prior asymptomatic COVID-19 infection. Interestingly, the clinical syndrome on presentation for this patient resembles the presentation of MIS-C/A. The six criteria in the preliminary case definition for MIS-A include adult age (≥ 21 years), fever, laboratory markers of inflammation, shock/hypotension, severe cardiac illness, and lack of alternative diagnosis. The MIS-A criteria by the CDC is currently the only definition for MIS-A (Table 2).

Our patient presented with fever, thrombocytopenia, diffuse rash, hypotension, severe cardiac illness, elevated C-reactive protein level and erythrocyte sedimentation rate, and positive COVID-19 serology meeting criteria for a MIS-A diagnosis (Table 3). The chronology of the patient's symptoms could suggest a relation between his clinical presentation and receiving the BNT162b2 vaccine dose. Additionally, since our patient had positive serology for COVID-19 infection, the vaccination in combination with previous infection may have coalesced as trigger for the post-infectious cytokine-mediated hyper-inflammatory

Table 2
Case Definition of MIS-A, defined by clinical presentation, and laboratory evidence.

Case Definition for Multisystem Inflammatory Syndrome in Adults by the Center for Disease Control and Prevention
A patient aged ≥21 years hospitalized for ≥24 h, or with an illness resulting in death, who meets the following clinical and laboratory criteria.
Clinical Criteria Subjective fever or documented fever (≥38.0 C) for ≥24 h prior to hospitalization or within the first THREE days of hospitalization* and at least THREE of the following clinical criteria occurring prior to hospitalization or within the first THREE days of hospitalization*. At least ONE must be a primary clinical criterion.
1) Primary clinical criteria
➢ Severe cardiac illness Includes myocarditis, pericarditis, coronary artery dilatation/aneurysm, or new-onset right or left ventricular dysfunction (LVEF<50%), 2nd/3rd degree A-V block, or ventricular tachycardia. ➢ Rash AND non-purulent conjunctivitis
2) Secondary clinical criteria
➢ Shock or hypotension not attributable to medical therapy (e.g., sedation, renal replacement therapy) ➢ Abdominal pain, vomiting, or diarrhea ➢ Thrombocytopenia (platelet count <150,000/μl) ➢ New-onset neurologic signs and symptoms Includes encephalopathy in a patient without prior cognitive impairment, seizures, meningeal signs, or peripheral neuropathy (including Guillain-Barré syndrome)
Laboratory evidence The presence of laboratory evidence of inflammation AND SARS-CoV-2 infection.
➢ Elevated levels of at least TWO of the following: C-reactive protein, ferritin, IL-6, erythrocyte sedimentation rate, procalcitonin ➢ A positive SARS-CoV-2 test for current or recent infection by RT-PCR, serology, or antigen detection

Table 3
Patient meets criteria for MIS-A.

MIS-A criteria	Patient data
Clinical Criteria	Patient aged 21 years Hospitalized for >24 h Subjective fever for ≥24 h prior to hospitalization and documented fever (38 C) on first day of hospitalization.
Primary Clinical Criteria	Severe cardiac illness (new-onset right and left ventricular dysfunction (LVEF<30%))
Secondary Clinical Criteria	Shock (hypotension not attributable to medical therapy) Thrombocytopenia (platelet count 118,000/μl) Nausea, vomiting and diarrhea
Laboratory Evidence	Elevated levels of C-reactive protein, procalcitonin and erythrocyte sedimentation rate A positive SARS-CoV-2 test for recent infection by serology

process typically seen in MIS-A.

From a mechanistic standpoint, in contrast to the respiratory failure observed in acute COVID-19 infection in adults, cardiovascular dysfunction is the most frequently described physiological abnormality in MIS-C/A [2,4,5,23]. Among patients admitted to the intensive care unit for MIS-C, over 80% required cardiovascular support (fluid resuscitation and/or inotropic support), while only about 15% required mechanical ventilation [2,3]. It has been proposed that COVID-19 may directly cause myocardial damage in MIS-C/A, as cardiac troponin, and natriuretic peptides [N-terminal (NT)-proBNP or proBNP] were increased in most cases reported (73.6% and 86.8% respectively) [4]. Furthermore, in most cases an echocardiogram revealed reduced ejection fraction (<50%) within days of presentation [3,6–10,24]. These findings indicate that the post-viral systemic inflammatory response seen in MIS-C/A perhaps produces a myocyte injury similar to other viral etiologies of myopericarditis and heart failure. The mechanism of injury still requires further evaluation however, as MIS-C/A appears to be a distinct syndrome from viral myocarditis, with cardiac illness varying significantly in phenotype including coronary artery dilation, valvular disease, ventricular dysfunction and pericardial effusion [25].

Since April 2021, many cases have been reported of young adults with myocarditis within two to four days of receiving either the BNT162b2 or mRNA-1273 vaccine [12–17]. A review of more than 2.5 million vaccinated persons in Israel reported an incidence of only 2.3 per 100,000 persons, and of those reported with myocarditis only one was associated with cardiogenic shock [21]. A descriptive study of myocarditis cases reported to the CDC Vaccine Adverse Event Reporting System noted an overall rate of 8.3 per 100,000 persons [24]. The cases of myocarditis reported in literature differ from our case, as most reports are of patients who tested negative for SARS-CoV-2 Nucleocapsid IgG Antibodies, indicating no previous COVID-19 infection [12–17]. Moreover, these cases rarely resulted in severe illness, with only 1.8% requiring vasoactive medications and no cases (0 out of 1626) requiring VA-ECMO or ventricular assist devices [24].

While the case we report here had clear evidence of cardiac dysfunction (LVEF 11% on CMRI) and injury (troponin 1.94), there was no evidence of myocardial edema on imaging, failing to meet a myocarditis diagnosis by Lake Louise consensus criteria (Table 4). In the reported cases of MIS-C and MIS-A, the role of myocardial enhancement varies—with one case series reporting myocardial edema on CMRI in only 50% of cases [25]. This again distinguishes our case from previous reports of vaccine associated myocarditis, as the clinical picture for our patient was closer to that of MIS-A.

Fortunately, our patient had recovery of cardiac function, with his LV ejection fraction returning to normal within ten days of presentation to the hospital. It appears that prompt initiation of VA-ECMO, and inotropic support, as well the anti-inflammatory effect of corticosteroids, were key to this patient's recovery, as has been seen with MIS-C/A in the literature [2–4,11].

Several limitations to this report merit comment. Since MIS-A is a clinical diagnosis based on a constellation of symptoms, rather than a specific diagnostic marker, we cannot definitively confirm the proposed diagnosis here. Additionally, without an endomyocardial biopsy, we were unable to definitively rule out myocarditis given the positive serology for Coxsackie A and Campylobacter [18,19]. It is important to note however, in comparison to Coxsackie B viral myocarditis—which contributes to nearly 25% cases of myocarditis yearly—Coxsackie A and Campylobacter are rarely reported to cause myocarditis [26,27]. Furthermore, with lack of inflammatory myocardial findings in two cardiac MRIs days apart, it appears to be more likely that the patient had a diagnosis of MIS-A, and not multiple concomitant infections leading to myocarditis. Considering the clinical history, the short interval between the administration of vaccination and the presentation of cardiogenic shock, and the exclusion of other common causes, BNT162b2 vaccine-induced MIS-A is the proposed diagnosis in our patient.

Table 4
Consensus for CMRI myocarditis criteria.

Proposed diagnostic CMR criteria (Lake Louise Consensus Criteria) for myocarditis
In the setting of clinically suspected myocarditis, CMR findings are consistent with myocardial inflammation, if at least two of the following criteria are present:
1. Regional or global myocardial SI increase in T2-weighted images 2. Increased global myocardial early gadolinium enhancement ratio between myocardium and skeletal muscle in gadolinium-enhanced T1-weighted images 3. There is at least one focal lesion with non-ischemic regional distribution in IR-prepared gadolinium-enhanced T1-weighted images ("late gadolinium enhancement")[d]
A CMR study is consistent with myocyte injury and/or scar caused by myocardial inflammation, if - criterion 3 is present.
A repeat CMR study between 1 and 2 weeks after the initial CMR study is recommended, if - none of the criteria are present, but the onset of symptoms has been very recent and there is strong clinical evidence for myocardial inflammation. - one of the criteria is present.
The presence of LV dysfunction or pericardial effusion provides additional, supportive evidence for myocarditis.

As the global effort for COVID-19 vaccination continues, further surveillance will be needed as to whether MIS-C/A could be an adverse effect of mRNA-COVID-19 immunization. Both mRNA-COVID-19 vaccines have been shown to be relatively safe and effective. Any notion of a causal relationship between the mRNA-COVID-19 vaccine and MIS-A in this case remains speculative. This report does not detract from the overwhelming benefit of vaccination from COVID-19. If there are further reports of MIS-C/A after vaccination however, clinicians should be aware of this possible relationship in the future.

Informed consent

Patient provided written informed consent.

CRediT authorship contribution statement

Elizabeth M. Jean-Marie, MD, MSc: Writing, Review & Editing, Visualization, Conceptualization.
Aya Tabbalat, MD: Writing, Review & Editing.
Chad Raymond, DO: Review& Editing.
Meisam Moghbelli MD, MPH: Review& Editing.
Keith Armitage, MD: Review& Editing.
Ian J. Neeland, MD: Writing, Review& Editing, Supervision.

Uncited references

[11,17,19,20]

Declaration of competing interest

The authors declare that the research was conducted in the absence

of any commercial or financial relationships that could be construed as a potential conflict of interest.

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