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Cardiac complications from multisystem inflammatory syndrome associated with prior COVID-19 infection

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SUMMARY

Multisystem inflammatory syndrome in adults (MIS-A) is a systemic inflammatory condition that presents roughly 4–6 weeks after initial COVID-19 infection. Patients typically present with persistent fevers, widespread rash, abdominal pain, vomiting and diarrhoea, and new-onset neurological symptoms. Cardiac dysfunction is a prominent feature of COVID-19 sequelae due to the abundance of ACE2 receptors on cardiac tissue. Delayed diagnosis due to the novelty of MIS-A can lead to cardiac complications like heart failure and shock, which could result in chronic cardiac disease. Avoidance of complications and chronic illness is possible with prompt corticosteroid therapy. Despite patient recovery to baseline level of function, surveillance of cardiac function to screen for chronic cardiac disease in the follow-up period is recommended. We present a case of MIS-A in a young man, compare his presentation with other similar cases and discuss implications of delayed diagnosis.

BACKGROUND

With the increasing prevalence of COVID-19 infections and the ongoing evolution of variants of the SARS-CoV-2 virus, multisystem inflammatory syndrome in adults (MIS-A) associated with prior COVID-19 infection is being identified in more patients worldwide. MIS-A is a hyperinflammatory syndrome that afflicts patients in the postinfectious period, typically 4–6 weeks after initial COVID-19 infection. Despite the rising number of MIS-A cases, diagnosis and treatment are often delayed due to multiorgan involvement and the absence of universal diagnostic criteria. Additionally, the novelty of this COVID-19 sequela and consequent lack of clinician awareness may result in extensive evaluation for alternative etiologies, such as rare infectious pathogens and malignancies, which can ultimately delay the initiation of treatment. During this time, patients with MIS-A may experience rapid clinical deterioration with potential development of complications arising from the rampant inflammation that may be avoidable with earlier diagnosis. We present a case of MIS-A complicated by cardiovascular illness in a young adult, and discuss how clinicians can avoid consequences of delayed diagnosis and treatment.

CASE PRESENTATION

A man in his late 30s, with only known medical history of anxiety and depression, was admitted to a local community hospital for acute hypoxic respiratory failure 1 week after testing positive for COVID-19 by PCR test. During his 9-day hospital

course, the patient improved with high flow nasal cannula (NC) oxygen therapy, antibiotics and dexamethasone, and was discharged with a tapered prednisone course starting at 20 mg daily.

Four days later, he was readmitted there with recurring fever to 39.4°C and persistent generalised weakness. Blood tests during this admission were significant for a white cell count (WCC) of $33.2 \times 10^9/L$, neutrophilia (absolute neutrophil count $26.9 \times 10^9/L$), anaemia (haemoglobin 125 g/L) and lactic acidosis (lactate 3.4 mmol/L). Further admission laboratory assessment was notable for elevations in ferritin (74 564 ng/mL), C reactive protein (CRP) (20.3 mg/L), D-dimer (14.57 mg/L) and procalcitonin (14.3 ng/mL). COVID-19 PCR test on admission was negative. CT pulmonary angiogram was done due to elevated D-dimer, and showed multiple segmental pulmonary emboli and multifocal airspace opacities with mediastinal and hilar adenopathy. A transthoracic echocardiogram (TTE) was obtained for workup of pleuritic chest pain which revealed a newly reduced left ventricular ejection fraction (LVEF) of 38% (video 1) using the biplane Simpson's method. Despite the initiation of broad-spectrum antibiotics, the patient continued to have nightly fevers to 39°C. This led to an extensive workup for autoimmune and infectious processes that was ultimately negative (table 1).

A bone marrow biopsy was done and showed no findings of an underlying haematological malignancy. A second COVID-19 PCR test was negative. After 15 days there, he was transferred to our facility for further evaluation.

On presentation to our hospital, the patient reported persistent fevers despite improving leucocytosis and inflammatory markers (table 2, day 1).

On evaluation, the patient was febrile to 38.5°C, tachycardic to 105 beats/min and tachypneic to 18 breaths/min but remained normotensive (121/64 mm Hg) with normal oxygen saturation on room air. Physical examination revealed normal heart sounds, clear lung fields to auscultation and generalised abdominal tenderness to deep palpation.

INVESTIGATIONS

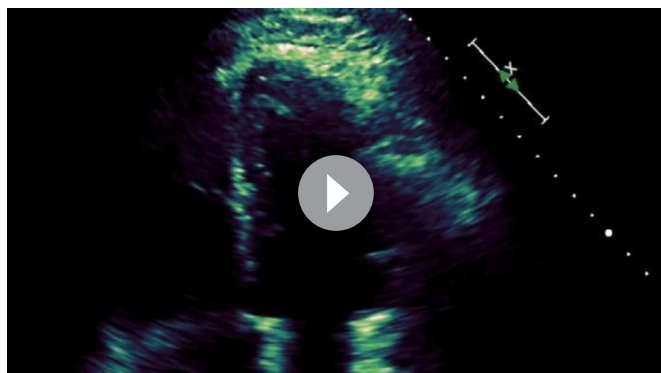
The extensive workup which was initiated at the outside hospital was continued and also broadened after arrival to our facility. Early on during his admission, ECG showed sinus tachycardia and left ventricular hypertrophy (figure 1). Repeat TTE showed recovered systolic function with LVEF of 60%–65%.

The patient was evaluated for bacterial and viral infections but also fungal, tickborne and parasitic infections; all were ultimately negative (table 1).



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Video 1 Transthoracic echocardiography showing reduced left ventricular ejection fraction.

Autoimmune evaluation was also pursued and unremarkable (table 1).

Given his negative infectious workup, recent COVID-19 infection, high fever, anaemia and elevated ferritin levels, he was evaluated for possible haemophagocytic lymphohistiocytosis.

NK cell quantitation was normal, but soluble interleukin-2 receptor (sIL-2R) level was elevated to 2733.1 pg/mL (reference range 175.3–858.2 pg/mL). His prior bone marrow biopsy

specimen was reviewed at our facility. It revealed normocellular marrow with trilineage haematopoiesis, with no significant dysplasia or increase in blasts. There were no findings of haemaphagocytosis.

On hospital day 3, the patient was febrile to 39.6°C, tachycardic to 136 beats/min and required 2L of oxygen via NC concerning for sepsis with subsequent chest radiograph showing bibasilar lung opacities and bilateral pleural effusions (figure 2). Follow-up CT scan of the chest revealed symmetric, hazy ground glass opacities in the lungs and bilateral pleural effusions (figure 3). Thoracentesis was performed with fluid analysis revealing exudative effusion by Light's criteria, which ultimately did not yield an infectious organism (table 3).

Bronchoscopy was attempted but aborted due to respiratory instability. During this time, his fevers persisted despite acetaminophen and ibuprofen.

On hospital day 5, the patient acutely developed pleuritic chest pain, significant tachycardia, hypotension, increased work of breathing, and need for supplemental oxygen. Urgent TTE demonstrated moderate to large pericardial effusion and late diastolic collapse of the right ventricle, concerning for cardiac tamponade (video 2, figure 4). The patient underwent emergent pericardiocentesis, with removal of 465 mL of serosanguinous fluid and placement of a pericardial drain. Postprocedure echocardiogram demonstrated minimal residual pericardial fluid. While WCC more than $25 \times 10^9/L$ were in the pericardial fluid, cultures were negative for both bacterial and fungal sources of infection, and pathology revealed significant inflammation (table 4).

The pericardial drain was removed 7 days after placement with follow-up echocardiograms demonstrating minimal pericardial fluid and no evidence of tamponade.

DIFFERENTIAL DIAGNOSIS

Due to the persistence of the patient's high fever and an array of systemic inflammatory symptoms, infectious and rheumatological processes were all considered but eventually excluded. HLH was ruled out given normal bone marrow biopsy results, lack of cytopenias and normal NK cell activity. Myocarditis from potential severe COVID-19 reinfection was felt to be unlikely due to multiple negative PCR tests. In addition to this, the significantly elevated leucocyte count with a predominance of segmented neutrophils far above the upper limits of normal for pericardial fluid studies,¹ in the context of sterile cultures pointed to a hyperinflammatory response that was confirmed by pathology. Therefore, with such broad workup being unremarkable but with persistent fevers in spite of broad-spectrum antibiotics, multiorgan involvement, and a COVID-19 infection that was, at this point about 5 weeks prior, MIS-A associated with prior COVID-19 infection became the working diagnosis.

TREATMENT

After MIS-A became the presumed diagnosis, the patient began corticosteroid therapy on hospital day 6, 1 day after pericardiocentesis. He was treated with intravenous methylprednisolone 80 mg daily for 4 days. Additionally, he was treated for pericarditis with a 5-day course of colchicine and non-steroidal anti-inflammatory drugs. Within 24 hours of treatment initiation, the patient's vitals stabilised. The patient remained afebrile for the remainder of his hospital course with marked improvement in pleuritic chest pain. Leucocytosis improved to a WCC of $12.98 \times 10^9/L$ on day of discharge. After 4 days of methylprednisolone, he was transitioned to

Table 1 Infectious and autoimmune evaluation

Laboratory test	Results
<i>Clostridium difficile</i> stool antigen	Negative
<i>Legionella</i> antigen	Negative
<i>Ehrlichia chaffeensis</i> antibodies	Negative
<i>Rickettsia rickettsii</i> antibody	Negative
<i>Anaplasma phagocytophilum</i> antibodies	Negative
<i>Francisella tularensis</i> antibodies	Negative
<i>Bartonella</i> antibodies	Negative
<i>Brucella</i> antibodies	Negative
Q fever antibody	Negative
Lyme disease antibody	Negative
Strongyloides IgG antibody	Negative
Babesia DNA PCR	Negative
Quantiferon-TB gold plus	Negative
Cryptococcal antigen	Negative
Coccidioides antibody	Negative
Blastomyces antibody	Negative
Histoplasma antibody	Negative
Aspergillus flavus antibody	Negative
Aspergillus niger antibody	Negative
Aspergillus fumigatus antibody	Negative
Influenza A PCR	Negative
Influenza B PCR	Negative
HIV-1 antigen, HIV-1/2 antibody	Negative
IgG antibody to EBV nuclear antigen	>8.0 (reference range: <0.8), positive
IgM antibody to EBV viral capsid antigen	Negative
IgG antibody to EBV viral capsid antigen	5.5 (reference range: <0.8), positive
CMV IgM antibody	Negative
Antinuclear antibody	Negative
Perinuclear antineutrophilic cytoplasmic antibody	Negative
Cytoplasmic antineutrophilic cytoplasmic antibody	Negative
Methicillin-resistant staphylococcus aureus nasal swab	Negative
Vancomycin-resistant enterococci perirectal swab	Negative
CMV, Cytomegalovirus; EBV, Epstein-Barr Virus; TB, Tuberculosis.	

Table 2 The patient's laboratory results during evaluation at our medical centre

Laboratory test	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7	Day 8	Day 9	Day 10	Day 11	Day 12	Day 13	Day 14
White cell count ($\times 10^9/L$)	16.4	16.8	20.0	17.0	22.7	27.5	16.0	17.3	16.7	15.5	17.1	30.3	15.9	13.0
Neutrophil count ($\times 10^9/L$)	13.7	16.1	18.4	14.6	13.6	22.3	14.9	13.6	11.5	11.5	15.0	28.6		
Platelets ($\times 10^9/L$)	480	514	498	473	621	487	442	489	559	501	564	554	535	494
Haemoglobin (g/L)	94	98	90	87	93	84	83	80	90	90	99	106	101	103
Ferritin (ng/mL)	40 000													
C reactive protein (mg/L)	17.1													

prednisone, with plans to taper over 3 weeks. Repeat TTE on discharge revealed LVEF of 60%–65% and trivial pericardial effusion. The patient continued treatment of systolic heart failure with metoprolol and losartan throughout his hospitalisation and at discharge. He was ultimately discharged on hospital day 14.

OUTCOME AND FOLLOW-UP

One week after discharge, the patient continued to have dizziness and chest pain. He was still taking prednisone, metoprolol and losartan. Approximately 1 month later, at his follow-up with his primary care physician, he reported symptomatic improvement and only mild dizziness with exertion along with lower extremity oedema for which he was started on torsemide. At outpatient follow-up with cardiology, the patient was diagnosed with American Heart Association Stage C chronic systolic heart failure with recovered LVEF with New York Heart Association Class III symptoms. During this visit, his metoprolol dose was decreased and torsemide was discontinued in light of euvoemia. He returned to work, but with limitations due to diminished exercise tolerance, and persistent exertional dyspnoea. The patient has ongoing follow-up with cardiology.

DISCUSSION

Rapid viral replication during COVID-19 infection causes massive endothelial cell death and vascular permeability that triggers an acute cytokine storm, resulting in a systemic inflammatory response and multiorgan damage. This systemic inflammation can cause a range of clinical manifestations with varying severities, which has led to the identification of multiple sequelae of COVID-19. Since the onset of the global pandemic, there are increasing number of reports of patients who experience persistent symptoms or organ dysfunction weeks after acute COVID-19 infection, characterised

as post-COVID-19 syndrome (colloquially known as 'long COVID-19') or MIS.² Since data on the wide constellation of illnesses are limited, continuous efforts are being made by organisations such as the Centers for Disease Control and Prevention (CDC) to create diagnostic criteria and management strategies for COVID-19 sequelae.

Since the identification of MIS in children (MIS-C) associated with COVID-19 infection in April 2020, the CDC has extended their definition to include adults over the age of 21, leading to the preliminary diagnostic criteria for MIS-A (box 1).³

Despite this preliminary definition, diagnosis and treatment of MIS-A is often delayed due to its novelty and since further evaluation of patients requires time.

We reviewed five case reports of adult cases of MIS-A with evidence of cardiac dysfunction,^{4–8} a case series of 16 patients published by the CDC,⁹ and a recently published systematic review of 221 patients, which represents the largest cohort of reported MIS-A cases.¹⁰

Based on the literature, symptom onset occurred 4–6 weeks after prior symptomatic illness from acute COVID-19 infection. Most patients present with high fever, hypotension, gastrointestinal distress, cardiac dysfunction and diffuse maculopapular rash. Despite the involvement of multiple systems, significant respiratory illness and hypoxia that characterises acute COVID-19 infection is typically not seen. Regarding laboratory testing, most patients present with elevated inflammatory markers (CRP, ferritin, procalcitonin) and elevated markers of coagulopathy (D-dimer). There is an increased occurrence of MIS-A among the following populations: younger age groups, males and minorities (Hispanic

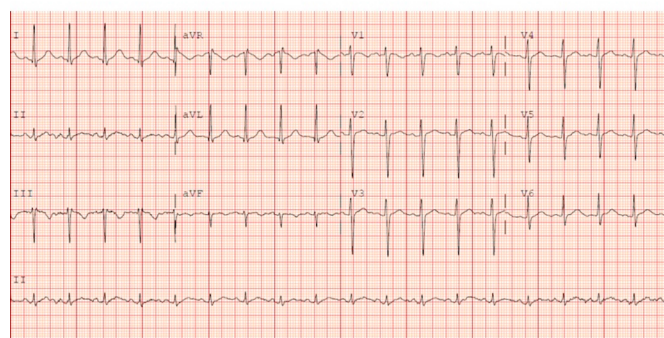


Figure 1 ECG consistent with sinus tachycardia and left ventricular hypertrophy. aVR, Augmented Vector Right; aVL, Augmented Vector Left; aVF, Augmented Vector Foot.

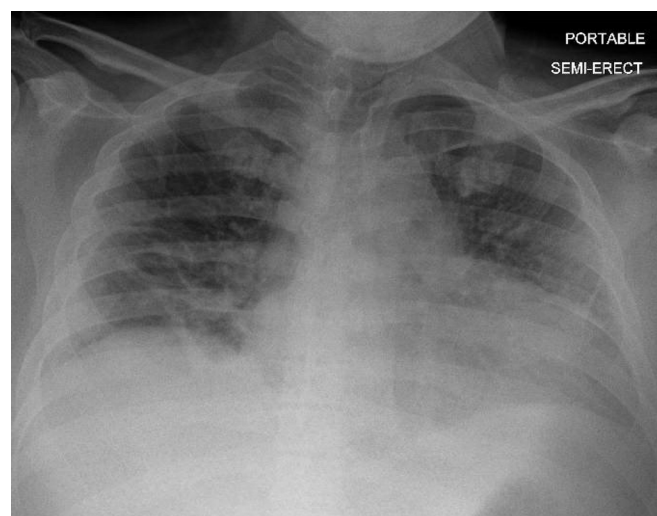


Figure 2 Chest X-ray showing bibasilar opacities and bilateral pleural effusions.

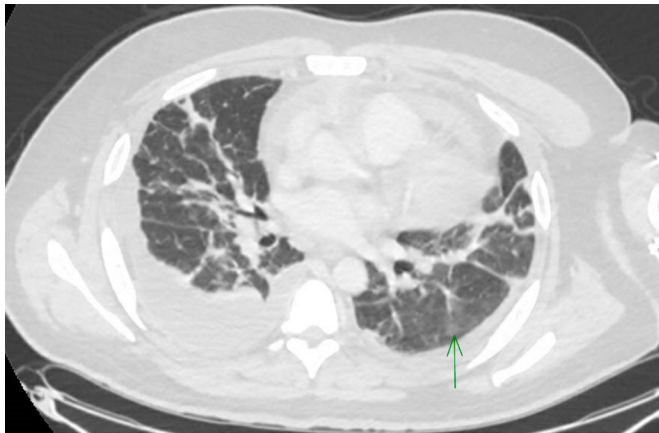
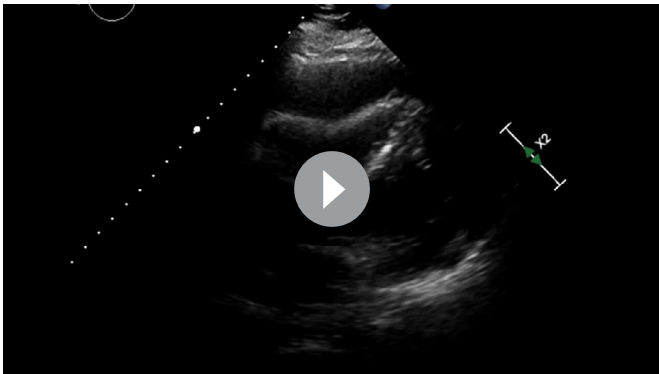


Figure 3 CT of the chest showing bilateral hazy ground glass opacities (arrow) and bilateral pleural effusions.

and non-Hispanic black patients).⁴⁻¹⁰ It is difficult to distinguish whether these populations have an increased inherent risk of developing MIS-A or if their increased risk is based on the health inequalities that minority communities face. Our patient meets all three of these population demographics.

In the systematic review of 221 MIS-A cases, only 32% of 192 patients tested positive on PCR and serological tests during hospitalisation for MIS-A. However, 98% of 211 patients had evidence of current or past COVID-19 infection via PCR and serological testing.¹⁰ Given the wide array of symptoms and non-specific findings in MIS-A, it is important to evaluate for recent COVID-19 infection and exposure on initial patient presentation. By assessing history of recent viral illness and obtaining laboratory evidence of current or past COVID-19 infection, diagnosis of MIS-A can be made in a timely fashion and treatment can be commenced. Delayed onset of treatment can lead to prolonged hospital stays and severe complications, as seen in our patient.

Most COVID-19 patients have evidence of cardiac dysfunction indicated by elevated troponin levels or abnormal echocardiographic findings. The high incidence of cardiac dysfunction in COVID-19 patients is thought to stem in part due to the abundant expression of ACE2 receptors on the myocardium that rivals the lungs. ACE2 is a surface enzyme expressed on multiple organs, but most notably the heart, and has been shown to play a role in hypertension, heart failure and myocardial infarction. With regard to COVID-19, it has been shown that the virus will enter cardiomyocytes via interaction between the SARS-CoV-2 spike protein and the N-terminal segment of the ACE2 receptor. Following entry of the virus into the cardiomyocyte, there is



Video 2 Transthoracic echocardiography demonstrating moderate-to-large pericardial effusion with late diastolic collapse of right ventricle, causing concern for cardiac tamponade.

inhibition of metabolic pathways which induces a cytotoxic response in cardiomyocytes leading to significant inflammation and fibrosis which ultimately result in cardiac injury and dysfunction.^{11 12} Prior COVID-19 infection can trigger a systemic proinflammatory state and a dysregulated immune response, leading to development of MIS-A. Factors such as preexisting comorbidities and inflammation due to ageing in adults can obscure and complicate MIS-A compared with children. In the systematic review of 221 MIS-A cases, a great proportion of patients with MIS-A presented with myocarditis and cardiac dysfunction compared with 3639 paediatric patients with MIS-C ($p<0.001$).¹⁰ In a case series of 33 paediatric patients with MIS-C, 73% of patients had abnormal cardiac testing (evident by ECG, echocardiogram and serum cardiac biomarkers). All patients in the MIS-C case series with evidence of cardiac dysfunction had normal cardiac testing on discharge.¹³ Given the prolonged inflammatory state present in MIS-A and evidence of cardiac dysfunction as a common finding in COVID-19 sequelae, patients diagnosed with MIS-A should undergo cardiac testing and imaging with ECG and echocardiogram on presentation and prior to discharge. In addition, throughout a patient's treatment course, there should also be low threshold for evaluation of disorders of the pericardium, and specifically the development of tamponade physiology, as without urgent intervention, patients can quickly progress to shock and death.

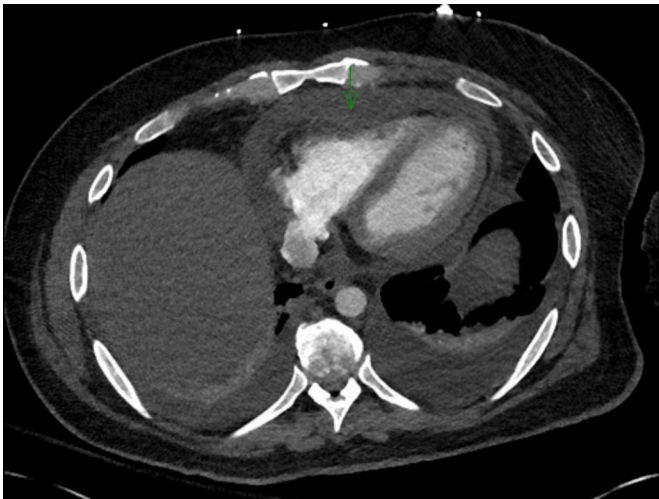


Figure 4 CT pulmonary angiogram demonstrating moderate to large pericardial effusion (arrow).

Table 3 Pleural fluid studies	
Laboratory test	Results
pH	7.3
Glucose	114 mg/dL
Protein	4.4 g/dL
Lactate dehydrogenase	741 U/L
White cell count	0.920×10 ⁹ cells/L
Segmented neutrophils	83%
Lymphocytes	10%
Monocyte/macrophage	7%
Culture	No growth
Gram stain	1+ white blood cells, no bacteria seen

Table 4 Pericardial fluid studies

Laboratory test	Results
White cell count	25.689×10 ⁹ cells/L
Segmented neutrophils	92%
Monocyte/macrophage	8%
Bacterial culture	No growth
Gram stain	Few white blood cells, no bacteria seen
Fungal culture	No growth
Pathology	Acute inflammation, no evidence of malignancy

MIS-A can be treated with corticosteroid therapy, intravenous Ig, or immune modulators like tocilizumab. All patients in the individual case reports had marked symptom improvement with some variation of treatments previously described.^{4–8} Of the 13 patients in a CDC case series that received some variation of 3 treatments, 1 did not survive to discharge.⁹

On review of the patient's clinical course, we believe the patient's initial presentation of MIS-A was likely on the day of readmission to the outside hospital, which was roughly 3 weeks after his initial positive COVID-19 PCR test. If this is the case, the patient ultimately started corticosteroid therapy 21 days after his initial presentation of MIS-A. During this delay in diagnosis and treatment, the patient developed acute systolic heart failure and obstructive shock secondary to acute pericardial tamponade. On reflection, both cardiac injuries might have been avoided with earlier initiation of corticosteroid therapy. Despite normalisation of our patient's cardiac function on discharge, he continued to experience symptoms of chronic systolic heart failure despite his recovered LVEF in the follow-up period. After discharge, patients with MIS-A who are diagnosed with cardiovascular complications during hospitalisation should follow-up with cardiology in the outpatient setting to evaluate for chronic cardiac illness and appropriate management.

Sequelae of COVID-19 remain a novelty to the medical community. Better defined classifications and diagnostic criteria are needed to prompt the timely diagnosis and management of

Box 1 Centers for Disease Control and Prevention case definition of multisystem inflammatory syndrome in adults

1. Documented fever ($\geq 38^{\circ}\text{C}$) for ≥ 24 hours prior to hospitalisation or within first 3 days of hospitalisation.
2. Meet at least three clinical criteria (atleast one must be a primary clinical criterion).
 - a. Primary clinical criteria
 1. Severe cardiac illness (ie, myocarditis, pericarditis, ejection fraction $<50\%$).
 2. Rash and non-purulent conjunctivitis.
 - b. Secondary clinical criteria
 1. New-onset neurological symptoms (ie, seizures, meningeal signs).
 2. Shock or hypotension.
 3. Abdominal pain, vomiting, diarrhoea.
 4. Thrombocytopenia (platelet count $<150 \times 10^9/\text{L}$).
3. Meet laboratory evidence criteria
 - a. Elevated levels of atleast two of the following: C reactive protein, ferritin, interleukin-6, erythrocyte sedimentation rate, procalcitonin.
 - b. A positive COVID-19 test via RT-PCR, serology or antigen detection.

Patient's perspective

I was anxious when first admitted, a feeling that was only exacerbated with my deterioration at the time of impending tamponade. Furthermore, there was the challenge of a potentially invasive procedure. The promptness of my team of doctors and nurses and their collaborative decision-making approach with involvement of my significant other every step of the way was immensely helpful in allaying my fears and ensuring recovery. I hope that sharing my case with the medical community at large will impart important lessons on early diagnosis and appropriate management.

Learning points

- ▶ Consider multisystem inflammatory syndrome in adults (MIS-A) in adults with recent COVID-19 infection presenting with persistent high fever and multiorgan symptoms or involvement.
- ▶ In patients with a suspected diagnosis of MIS-A and a history of asymptomatic acute COVID-19 infection without prior positive PCR test, conduct antibody testing to confirm previous COVID-19 infection.
- ▶ Promptly commence corticosteroid therapy for MIS-A as soon as other differential diagnoses with a high level of clinical suspicion are ruled out.
- ▶ Appropriately treat chronic medical conditions (ie, chronic systolic heart failure) associated with MIS-A and COVID-19 infection in the inpatient setting.
- ▶ Recommend appropriate outpatient follow-up for chronic conditions arising from MIS-A and COVID-19 infection.

MIS-A. Furthermore, greater efforts are required to understand the long-term effects of COVID-19 infection on physiology and its impact on development of chronic diseases after resolution of viral infection.

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Case reports provide a valuable learning resource for the scientific community and can indicate areas of interest for future research. They should not be used in isolation to guide treatment choices or public health policy.

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