JACC PATIENT CARE PATHWAYS

Fulminant Myocarditis Following SARS-CoV-2 Infection

JACC Patient Care Pathways

Saurabh Rajpal, MD,[a] Rami Kahwash, MD,[a] Matthew S. Tong, DO,[a] Kelly Paschke, DO,[a] Anjali A. Satoskar, MD,[b] Beth Foreman, MD,[a] Larry A. Allen, MD, MHS,[c] Nicole M. Bhave, MD,[d] Ty J. Gluckman, MD, MHA,[e] Valentin Fuster, MD, PHD[f,g]

ABSTRACT

A 60-year-old woman with a past medical history of asthma presented with fulminant myocarditis 9 days after testing positive for SARS-CoV-2 and 16 days after developing symptoms consistent with COVID-19. Her hospital course was complicated by the need for veno-arterial extracorporeal membrane oxygenation, ventricular arrhythmias, and pseudomonas bacteremia. She ultimately recovered and was discharged to home with normal left ventricular systolic function. Thereafter, she developed symptomatic ventricular tachycardia, for which she received an implantable cardioverter-defibrillator and antiarrhythmic drug therapy. (J Am Coll Cardiol Case Rep 2022;4:567–575) © 2022 by the American College of Cardiology Foundation.

CASE PRESENTATION

A 60-year-old woman with a past medical history of asthma presented to the emergency department (ED) during the summer of 2020 with extreme fatigue, shortness of breath, and palpitations.

Nine days prior, she was seen by her primary care physician to evaluate a week of symptoms, including fever, decreased sense of smell and taste, body aches, sore throat, and congestion. These symptoms began several days after close contact with someone who was COVID-19 positive. She was diagnosed with SARS-CoV-2 infection by a PCR test and was started on supportive treatment with antipyretics. Although her symptoms improved initially, she came to the ED because of worsening fatigue, shortness of breath, and a heart rate of approximately 140 beats/min on a home monitor.

Initial vital signs included a heart rate of 141 beats/min, blood pressure of 138/63 mm Hg, temperature of 97.3 °F, respiratory rate of 16 breaths/min, and oxygen saturation by pulse oximetry of 98% on room air. Upon arrival, she was not in respiratory distress, her

lungs were clear to auscultation, cardiac auscultation was unremarkable except for tachycardia, and neither jugular venous distention nor pedal edema was present.

Her initial electrocardiogram demonstrated sinus tachycardia, ST-segment elevation in leads V_1, V_2, and aVR, and ST-segment depression in multiple other leads (Figure 1). Her chest X-ray showed a normal heart size; normal pulmonary vasculature; and absence of focal consolidation, pleural effusion, or pneumothorax (Figure 2). Laboratory testing at presentation revealed a significantly elevated high-sensitivity troponin T (hs-TnT) of 1,737 ng/L (normal 0-14 ng/L) (Table 1). Cardiology consultation was requested.

Out of concern for an acute coronary syndrome, she was treated with aspirin, intravenous heparin, intravenous nitroglycerin, metoprolol, and atorvastatin. A repeat hs-TnT, 4 hours after the initial troponin was 2,382 ng/L. She was taken for urgent coronary angiography, which demonstrated normal coronary arteries. Her left ventricular end-diastolic pressure was 17 mm Hg with an arterial blood pressure of 96/65 mm Hg. Left ventriculography demonstrated mild global hypokinesis. An echocardiogram done around the same time showed moderately increased left ventricular wall thicknesses, mild global left ventricular systolic dysfunction (left ventricular ejection fraction [LVEF] 49%, Simpsons), grade II diastolic dysfunction, and a trivial to small pericardial effusion. Based on these findings, a presumptive diagnosis of viral myocarditis was made.

Within a few hours of her cardiac catheterization, she developed worsening hypotension that was treated with a fluid bolus. A computed tomography scan of the abdomen and pelvis found no evidence of a retroperitoneal bleed. Over the next 12-24 hours, her clinical status deteriorated, with markedly increased work of breathing. Examination showed jugular venous distension and diffuse crackles on lung examination, and she was started on noninvasive positive pressure ventilation. She also showed signs of a low-output state, with cold, clammy extremities and drowsiness. A repeat echocardiogram showed worsening left ventricular systolic function with an ejection fraction of 30%-35%, moderately increased left ventricular wall thicknesses with hyperechoic myocardial echotexture, and a small pericardial effusion (Videos 1 and 2). For worsening hypoxemia despite noninvasive positive-pressure ventilation, invasive mechanical ventilation was initiated. Pertinent laboratory values at that time included pH 6.96, pCO2 20.7 mEq (normal 35-45 mEq), bicarbonate 4.7 mEq (normal 22-26 mEq), pO2 247 mm Hg (normal 80-100 mm Hg), oxygen saturation 98% by arterial blood gas, lactate 14.9 mmol/L

TAKE HOME MESSAGES

- COVID-19 is associated with widely ranging cardiovascular complications that include, but are not limited to, myocarditis, myocardial injury, stress cardiomyopathy, thromboembolism, and post-acute sequelae of SARS-CoV-2 infection.

- Fulminant myocarditis following SARS-CoV-2 infection is rare. When present, it is characterized by severe myocardial inflammation with myocardial edema and myocyte necrosis. Cardiogenic shock, life-threatening arrhythmias, and multi-organ failure are defining features. Early institution of MCS can be lifesaving.

- Echocardiography and coronary angiography to exclude obstructive coronary artery disease are usually part of the initial evaluation of patients with suspected myocarditis. Echocardiographic features of myocarditis include ventricular wall motion abnormalities often in a noncoronary distribution, increased left ventricular wall thickness, abnormal ventricular strain, and reduced left ventricular systolic function.

- CMR is the preferred noninvasive test for evaluation of myocarditis and can also be used as a supplemental strategy for ongoing assessment and risk stratification. Persistence of LGE is an independent risk factor for mortality, heart failure, need for heart transplantation, and arrhythmias including sudden cardiac death.

- EMB is considered the gold standard for diagnosis of myocarditis, and despite its low yield, can be useful in confirming the diagnosis and evaluating for specific etiologies, like giant cell or eosinophilic myocarditis, that may require specific therapies.

- Arrhythmias represent important short- and long-term risks with myocarditis.

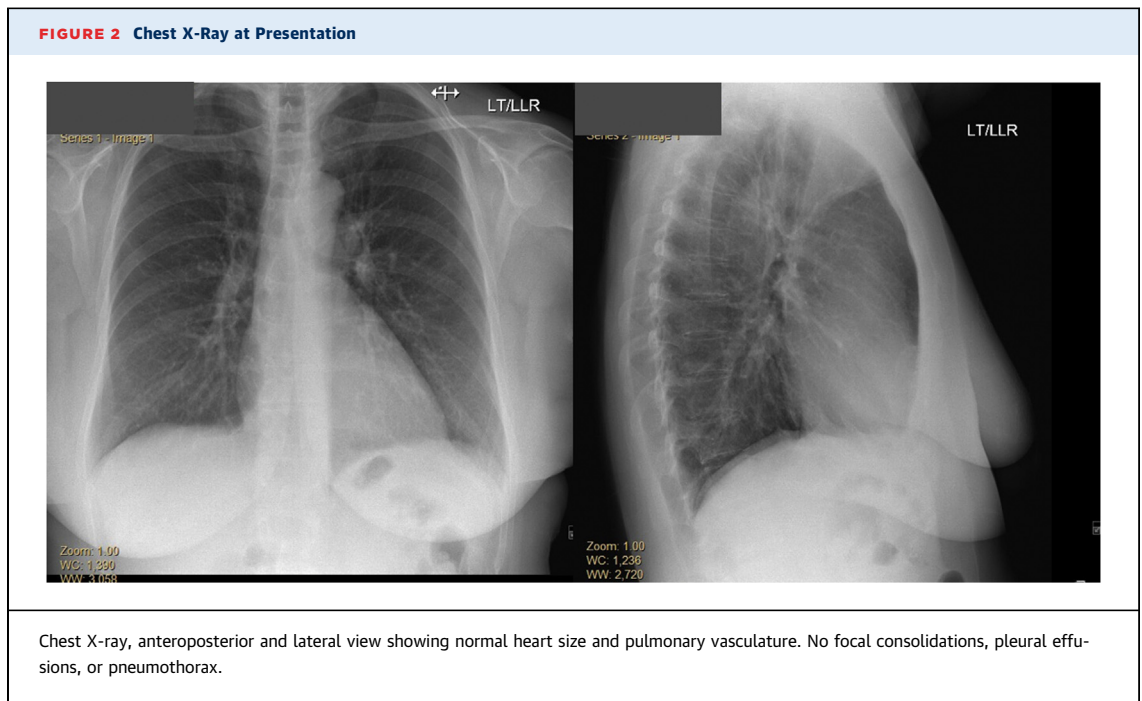**FIGURE 1** Electrocardiogram at Presentation
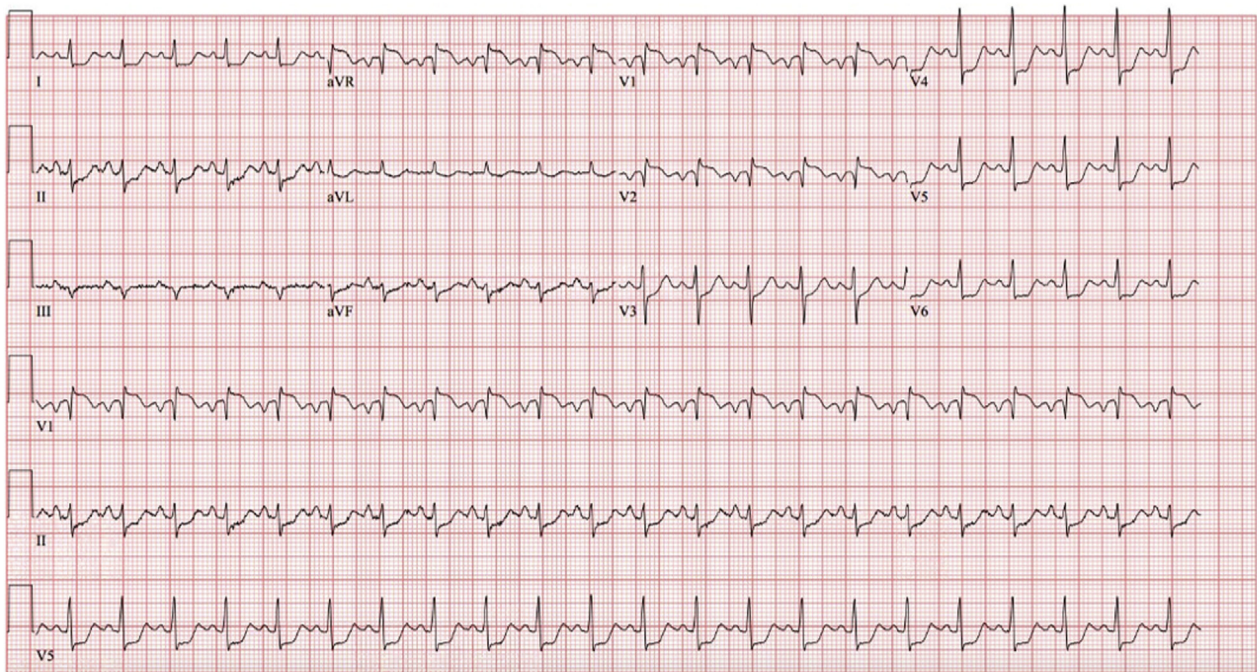
A 12-lead electrocardiogram showing sinus tachycardia; ST-segment elevation in avR, V₁, and V₂; and ST-segment depression in multiple other leads.

(normal 0.5-1.6 mmol/L), sodium 132 mEq/L, creatinine 1.4 mg/dL, N-terminal pro-B-type natriuretic peptide 19,054 pg/mL (normal <300 pg/mL), interleukin-6 2,169 pg/mL (normal <6 pg/mL) and hs-TnT 5,810 ng/L. She was started on intravenous dobutamine and administered a dose of intravenous furosemide. Her clinical status continued to deteriorate, with persistent hypotension requiring escalating doses of norepinephrine, vasopressin, epinephrine, and phenylephrine. Within the next few hours, she had 2 episodes of sustained ventricular tachycardia (VT), for which she underwent synchronized cardioversions twice and was started on intravenous amiodarone. An arterial blood gas showed severe metabolic acidosis, for which she was started on intravenous bicarbonate.

On hospital day 3, she remained hypotensive with acidemia and was started on veno-arterial extracorporeal membrane oxygenation (VA ECMO), cannulating the right femoral artery and vein. Her rapid hemodynamic deterioration suggested fulminant myocarditis. Several forms of percutaneous temporary mechanical support were considered by the shock team, informed by our center's experience. Primary bridge-to-decision VA ECMO was favored because of her rapid hemodynamic compromise, need for full circulatory support, risk of worsening right ventricular failure in the setting of myocarditis, and ability of ECMO to provide adequate oxygenation should COVID-19 acute respiratory distress syndrome evolve.

While on VA ECMO, continuous renal replacement therapy (CRRT) was initiated for worsening renal function. A bedside echocardiogram on hospital day 6 demonstrated severely reduced left ventricular systolic function with an ejection fraction <15%, along with increased left ventricular wall thicknesses and a small cavity size. Based on her presumed diagnosis of fulminant COVID-19 myocarditis, she was treated with 3 days of intravenous methylprednisolone (1 g/d), followed by a prednisone taper (60 mg daily initially, weaning 10 mg every 3 days). Concurrent with this, she experienced multiple episodes of nonsustained VT, for which she was continued on intravenous amiodarone and started on lidocaine. Over the next few days, her inotropic support was weaned off. On ECMO day 9, flow was weaned to 1.3 L/min, without worsening oxygenation, and a bedside echocardiogram demonstrated severely reduced but improving left ventricular systolic function with an ejection fraction of 25%-30%. On ECMO day 10, she was taken to the operating room, ECMO was weaned, and she was decannulated

FIGURE 2 Chest X-Ray at Presentation

Chest X-ray, anteroposterior and lateral view showing normal heart size and pulmonary vasculature. No focal consolidations, pleural effusions, or pneumothorax.

successfully. She required a brief course of vasopressin, epinephrine, and milrinone following decannulation. CRRT was discontinued on hospital day 20, transitioning to intermittent hemodialysis. Her urine output increased with intravenous furosemide within 6 days after CRRT discontinuation, and no further dialysis was required thereafter.

On hospital day 24, a repeat echocardiogram demonstrated improvement in her left ventricular systolic function with an ejection fraction of 50%-55%, normal left ventricular size, and reduced left ventricular wall thickness. Given stable and acceptable hemodynamics, she was started on metoprolol succinate, isosorbide dinitrate, and hydralazine. Cardiac magnetic resonance (CMR) was ordered to confirm the diagnosis of myocarditis and to assess more fully left and right ventricular systolic function. Diffuse hyperintensity on T_2 mapping (up to 67 milliseconds, normal <52 milliseconds) suggestive of myocardial edema or inflammation was observed (**Figure 3,** Video 3). The myocardial extracellular volume fraction was significantly elevated at 39% (normal <29%), indicative of diffuse interstitial expansion. LVEF and right ventricular ejection fraction were 45% and 33%, respectively, with normal biventricular size. Right heart catheterization and endomyocardial biopsy (EMB) were also performed to exclude granulomatous myocarditis, given the arrhythmia burden. The biopsy showed scattered perivascular and interstitial inflammatory cells

consisting of CD3-positive T-lymphocytes, CD20 positive B-lymphocytes, and histiocytes, along with interstitial and myocyte injury, suggestive of acute viral myocarditis. Viral immunostains for herpes simplex, adenovirus, and cytomegalovirus were negative. Congo red and iron stains were negative. There was no evidence of multinucleated giant cells, eosinophils, or granulomas (**Figure 4**).

Her hospital course was further complicated by pseudomonas bacteremia from infection at the ECMO cannula site, for which she completed a course of antibiotics. She was discharged to home on hospital day 40 with improved ventricular function by echocardiography (Videos 4 and 5). Before discharge, she underwent placement of an implantable loop recorder (ILR) and an external defibrillator vest and guideline-directed medical therapy for heart failure (metoprolol succinate, lisinopril, amiodarone, and furosemide) was continued. An implantable cardioverter-defibrillator was considered; however, preference was given to a wearable defibrillator, as the LVEF was >45%, and her myocarditis was considered to be an acute and potentially reversible insult. The ILR was felt to be an important adjunct, given her significant arrhythmia burden during hospitalization and its ability to help guide her need for an implantable cardioverter-defibrillator in follow-up.

At her 3-month cardiology follow-up, she underwent repeat CMR (**Figure 3,** Video 6) that demonstrated normal left ventricular systolic function with

TABLE 1 Laboratory Testing at Different Time Points of the Hospitalization

Laboratory Test	Presentation Day 1	Day 2	ECMO Day 1	ECMO Day 2	Discharge	Normal Range
High-sensitivity troponin T, ng/L	1,737	2,018				0-14
NT-proBNP, pg/mL	250	19,054				0-125
C-reactive protein, mg/L			44.79	88.61		<10
Sodium, mEq/L	140	132	149	141	146	135-148
Potassium, mEq/L	4.0	5.4	3.6	5.1	4.7	3.4-5.3
Chloride, mEq/L	101	95	101	101	111	96-110
Carbon dioxide, mEq/L	23	5	21	26	24	19-32
Glucose, mg/dL	108	217	189		77	72-99
BUN, mg/dL	10	23	33	28	26	3-29
Creatinine, mg/dL	0.6	1.4	1.86	2.28	1.2	0.5-1.2
Calcium, mg/dL	9.4	9.2				8.5-10.5
Lactate, mmol/L		14.9	17.5	1.56		0.5-1.6
Anion gap	16	32	31	19		5-15
Bilirubin, mg/dL		0.4		1.0	0.2	0-1.5
AST, U/L		199	4,393	6,200	18	14-40
ALT, U/L		118	3,240	2,480	24	9-42
ALP, U/L		178	98	116		32-126
WBC, K/mm^3	10.3	11.2	26.9		12.3	3.8-10.8
Hemoglobin, g/dL	14.5	12.8	9.6		8.6	12-15.6
Platelet count	275	253	157		264	130-400
PT		17.4				Seconds
INR		1.4				Seconds

ALP = alkaline phosphatase; ALT = alanine transaminase; AST = aspartate transaminase; BUN = blood urea nitrogen; ECMO = extracorporeal membrane oxygenation; INR = international normalized ratio; NT-proBNP = N-terminal pro–B-type natriuretic peptide; PT = prothrombin time; WBC = white blood cell.
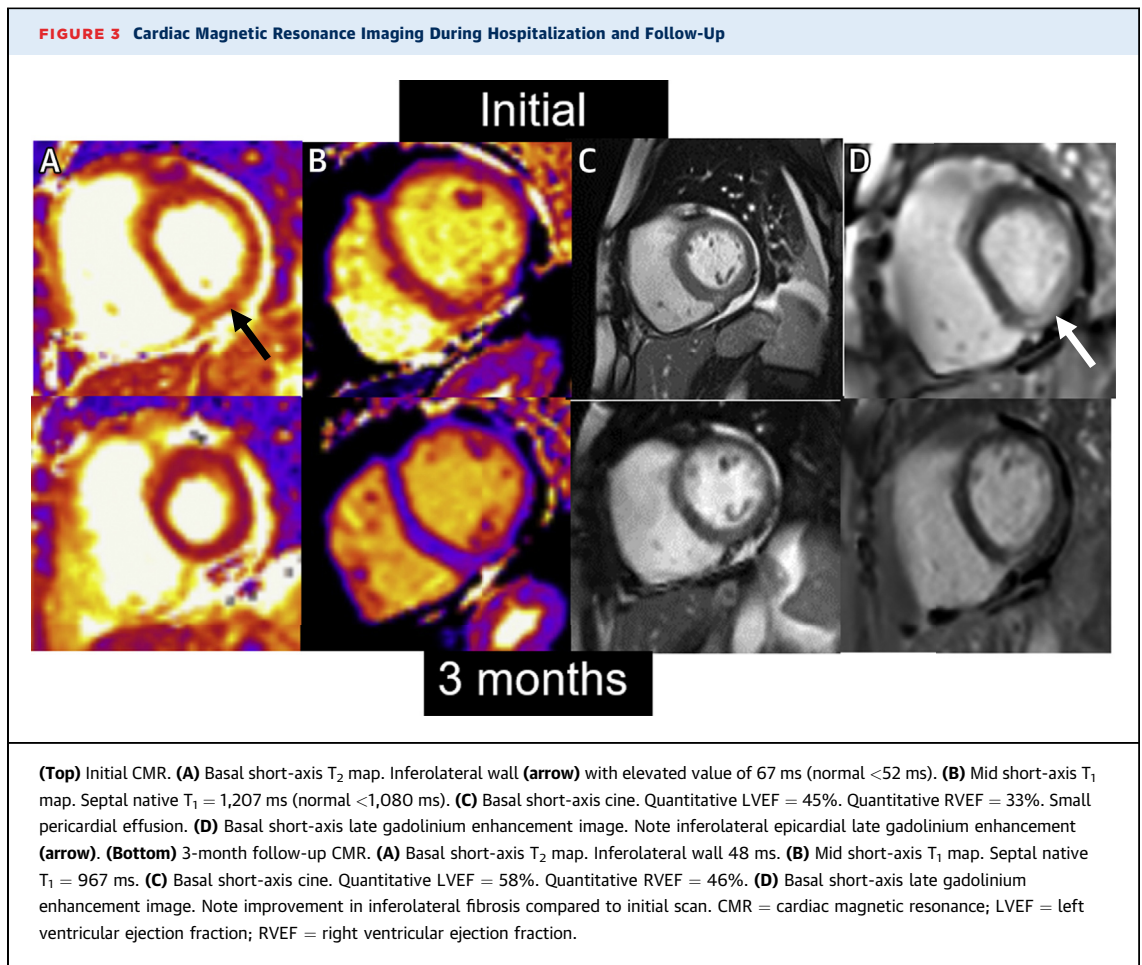
an ejection fraction of 58%, a low-normal right ventricular ejection fraction of 46%, and residual linear epicardial nonischemic fibrosis involving the basal to mid inferolateral and lateral left ventricular walls, suggestive of prior myocarditis (Figure 3). Interval improvement was noted in right and left ventricular systolic function, along with resolution of myocardial edema/inflammation and diffuse interstitial expansion. As her left ventricular systolic function had improved and no significant arrhythmias were noted with interrogation of her ILR, implantation of a cardioverter-defibrillator was deferred. However, 6 months later (9 months after her initial presentation) she presented to the ED with lightheadedness and was found to be in a wide complex tachycardia (confirmed to be VT) at a rate of 176 beats/min. Interrogation of her ILR demonstrated persistence of this rhythm for 2 hours and 30 minutes. Thereafter, she underwent an electrophysiology study that demonstrated multiple VT morphologies. For this, a cardioverter-defibrillator was implanted, and she was started on antiarrhythmic drug therapy with mexiletine. She has done well in follow-up, with preserved left ventricular systolic function, improving functional capacity, and no further arrhythmias.

OVERVIEW OF FULMINANT MYOCARDITIS

EPIDEMIOLOGY, PRESENTATION, EVALUATION, AND MANAGEMENT. Fulminant myocarditis is a life-threatening condition characterized by severe myocardial inflammation with myocardial edema and myocyte necrosis. Defining features include a rapidly progressive course, with cardiogenic shock, life-threatening cardiac arrhythmias, and multiorgan failure.

The incidence of viral myocarditis is estimated to be approximately 10 to 20 cases per 100,000 per year.[1] Although the incidence of fulminant myocarditis is less well-defined, it is considered quite rare. Classically, fulminant myocarditis has a lymphocytic infiltrate on EMB, with further categorization by etiology (eg, infectious, autoimmune, toxic).[2]

Most commonly, patients with fulminant myocarditis have symptoms and signs of heart failure with impending cardiogenic shock. Electrocardiographic findings may include low voltage in the setting of myocardial edema, significant ST-segment changes, and both tachyarrhythmias and bradyarrhythmias.[3] Laboratory abnormalities reflect compromised perfusion of various organs, with elevation of cardiac

FIGURE 3   Cardiac Magnetic Resonance Imaging During Hospitalization and Follow-Up

(Top) Initial CMR. **(A)** Basal short-axis T_2 map. Inferolateral wall **(arrow)** with elevated value of 67 ms (normal <52 ms). **(B)** Mid short-axis T_1 map. Septal native T_1 = 1,207 ms (normal <1,080 ms). **(C)** Basal short-axis cine. Quantitative LVEF = 45%. Quantitative RVEF = 33%. Small pericardial effusion. **(D)** Basal short-axis late gadolinium enhancement image. Note inferolateral epicardial late gadolinium enhancement **(arrow)**. **(Bottom)** 3-month follow-up CMR. **(A)** Basal short-axis T_2 map. Inferolateral wall 48 ms. **(B)** Mid short-axis T_1 map. Septal native T_1 = 967 ms. **(C)** Basal short-axis cine. Quantitative LVEF = 58%. Quantitative RVEF = 46%. **(D)** Basal short-axis late gadolinium enhancement image. Note improvement in inferolateral fibrosis compared to initial scan. CMR = cardiac magnetic resonance; LVEF = left ventricular ejection fraction; RVEF = right ventricular ejection fraction.

troponin being ubiquitous. A key feature of fulminant myocarditis is its rapid progression, with cardiogenic shock that requires vasoactive medications, temporary mechanical circulatory support (MCS), and/or extracorporeal life support (ECLS). Invasive evaluation with coronary angiography should be performed to assess for an ischemic etiology, along with hemodynamic monitoring to identify and address cardiogenic shock.[3,4]

Use of immunosuppressive therapy in fulminant viral myocarditis is not well supported by the literature and generally is reserved for patients with nonviral etiologies (eg, giant cell myocarditis, necrotizing eosinophilic myocarditis, and immune checkpoint inhibitor myocarditis).[3] Despite this, patients with viral myocarditis are frequently treated with corticosteroids.

MCS, IMAGING, EMB, AND ARRHYTHMIAS IN FULMINANT MYOCARDITIS

MECHANICAL CIRCULATORY SUPPORT. Hemodynamic and respiratory support represent early goals

of care for patients with fulminant myocarditis, with close monitoring of end-organ perfusion. Early transfer to an advanced heart failure center is recommended if fulminant myocarditis is suspected. Temporary MCS devices should be initiated in cases of refractory shock. Because biventricular failure is common, preference may instead be given to ECLS.[3]

The use of hemodynamic support devices in this population carries significant risks, including bleeding, coagulopathy, thrombosis, acute limb ischemia, infection, and neurological events. In most circumstances, however, the potential benefits of MCS and ECLS in fulminant myocarditis outweigh the risks.[5,6]

Liberating patients from MCS depends upon hemodynamics, cardiac output, and response to trials involving a reduction in circulatory support. Close monitoring for hemostasis and short-term complications is recommended thereafter.

CHOICE OF IMAGING. Echocardiography is the preferred imaging modality for initial evaluation of patients with suspected fulminant myocarditis, as it

FIGURE 4 Endomyocardial Biopsy
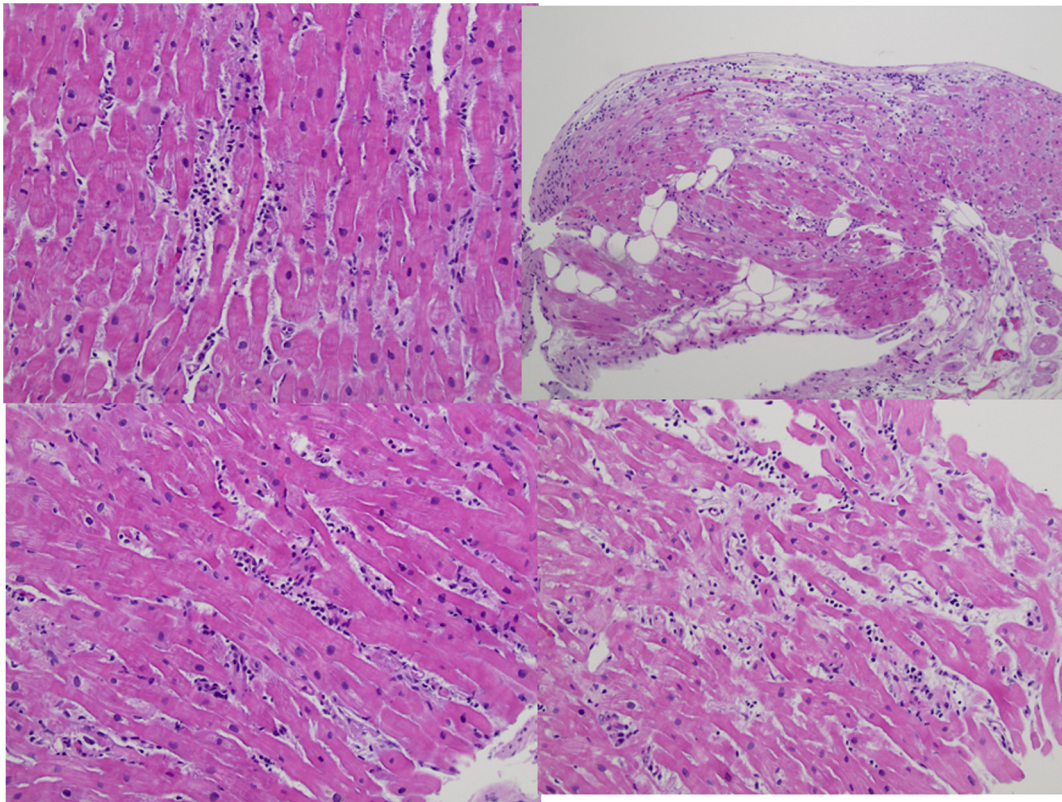
Endomyocardial biopsy showing scattered perivascular and interstitial inflammatory cell infiltrates, interstitial and myocyte injury, suggestive of acute myocarditis. Predominant CD3 T-lymphocytes fewer CD20 B-lymphocytes and CD68 histiocytes. Viral immunostains for herpes simplex virus, adenovirus, and cytomegalovirus were negative.

is widely available and can be performed at the bedside in rapidly deteriorating patients.[3] Increased myocardial thickness caused by myocardial edema with regional wall motion abnormalities in a non-coronary distribution and normal left ventricular size represent findings consistent with myocarditis; in contrast, a dilated ventricle is more often suggestive of an alternative process.[7] Presence of regional wall motion abnormalities may introduce challenges in discerning this from an acute coronary syndrome.

In all cases of myocarditis, exclusion of obstructive coronary artery disease is important, most often with invasive coronary angiography. This is particularly important in the setting of COVID-19, in which ischemic heart disease may be found at a higher rate.[8]

CMR can be used to confirm myocarditis and is the preferred noninvasive test for its evaluation.[3,9-11] However, in fulminant cases, patient instability may

prohibit its use, at least initially. Updated Lake Louise Criteria, which incorporate T_1 abnormalities (elevated native T_1 and/or late gadolinium enhancement [LGE]) and elevated native T_2 signals (in a focal or global distribution representative of myocardial edema or inflammation), along with additional supportive criteria (eg, regional or global dysfunction, pericardial effusion) (**Figure 3**), are highly sensitive and specific for those with suspected myocarditis.[12,13] T_1 and T_2 mapping improves diagnostic yield with CMR, and patterns of abnormalities can be used to identify specific types of myocarditis.[14] CMR also has prognostic utility, with persistence and extent of LGE predicting adverse outcomes including mortality and arrhythmias.[15] Finally, CMR can also be used to guide sites for endomyocardial biopsy (EMB).[9]

ROLE OF BIOPSY. Traditionally, EMB has been considered the gold standard for diagnosis in patients

with acute myocarditis. In cases of fulminant myocarditis, EMB can help discern the specific etiology and provide additional assistance in guiding therapy.[9] In less severe cases of myocarditis, EMB has a lower sensitivity for diagnosis than CMR and is also less likely to inform management.[2,16] EMB is also limited by low yield because of sampling error and lack of consensus on standard criteria for interpretation.[10,11,16] In our case, EMB helped confirm the diagnosis of post-viral lymphocytic myocarditis, while ruling out alternative etiologies.

ARRHYTHMIAS. Atrial and ventricular arrhythmias are common in fulminant myocarditis and may have multiple underlying mechanisms (eg, myocardial inflammation or edema, scar).[3,4,17] Exercise can trigger arrhythmias in acute myocarditis, and as such, current guidance cautions against exercise for 3-6 months to ensure resolution of myocardial inflammation.[18] In our patient, ventricular arrhythmias were observed at 2 different time points. During her hospitalization, polymorphic VT was felt to be the result of severe myocardial inflammation. This arrhythmia continued in the early post-ECMO period and then abated as ventricular function improved later in her course. As the acute phase resolved, no further episodes of VT were detected on her ILR. Nine months after discharge, however, she presented with symptomatic VT. CMR done at that time showed persistent LGE, and her VT was likely driven by presence of myocardial scar or persistent inflammation. Importantly, persistent LGE on CMR is an independent predictor of arrhythmias independent of ventricular function.[15,17]

COVID-19 AND MYOCARDITIS

In a recent study that included ∼154,000 patients with COVID-19, the HR for myocarditis was 5.4 compared with uninfected contemporary and historical cohorts.[8] Despite this, fulminant myocarditis with COVID-19 remains rare,[19,20] with most reported cases being mild in severity. Autopsy studies have further

suggested that although nonspecific cardiac inflammation is common with COVID-19, myocarditis meeting strict pathological criteria is rare.[21] Multiple reports have also noted myocardial involvement by CMR among asymptomatic and minimally symptomatic individuals.[22,23] Collectively, this has made estimation of the prevalence and incidence of myocarditis with COVID-19 quite challenging.

In addition to myocarditis, other forms of cardiovascular disease may manifest after SARS-CoV-2 infection, including pericarditis, myocardial ischemia, microvascular disease, nonischemic cardiomyopathy, stress cardiomyopathy, thromboembolism, and arrhythmia. For others, cardiovascular symptoms (eg, chest pain, dyspnea, palpitations) may occur or persist, with no specific cardiac etiology identified after testing. The 2022 ACC Expert Consensus Decision Pathway on Cardiovascular Sequelae of COVID-19 in Adults provides a framework by which to evaluate and manage these individuals.[24]

CONCLUSIONS

This case of fulminant myocarditis following SARS-CoV-2 infection highlights many of the severe clinical manifestations that may occur with this condition. Early recognition and implementation of supportive care (eg, ECLS) in cases such as this is critically important. Close monitoring of these patients after discharge is also paramount, as additional risks (eg, arrhythmic) may persist.

ADDRESS FOR CORRESPONDENCE: Dr Saurabh Rajpal, The Ohio State University Division of Cardiovascular Medicine, Davis Heart and Lung Research Institute, 473 West 12th Avenue, Suite 200, Columbus, Ohio 43210, USA. E-mail: Saurabh.Rajpal@osumc.edu.

REFERENCES

1. Tschöpe C, Ammirati E, Bozkurt B, et al. Myocarditis and inflammatory cardiomyopathy: current evidence and future directions. *Nat Rev Cardiol.* 2021;18(3):169–193. https://doi.org/10.1038/s41569-020-00435-x

2. Trachtenberg BH, Hare JM. Inflammatory cardiomyopathic syndromes. *Circ Res.* 2017;121(7):803–818. https://doi.org/10.1161/CIRCRESAHA.117.310221

3. Kociol RD, Cooper LT, Fang JC, et al. Recognition and initial management of fulminant myocarditis: a scientific statement from the American Heart Association. *Circulation.* 2020;141(6):e69–e92. https://doi.org/10.1161/CIR.0000000000000745

4. Ammirati E, Cipriani M, Moro C, et al. Clinical presentation and outcome in a contemporary cohort of patients with acute myocarditis:

Multicenter Lombardy Registry. *Circulation.* 2018;138(11):1088–1099. https://doi.org/10.1161/CIRCULATIONAHA.118.035319

5. Subramaniam AV, Barsness GW, Vallabhajosyula S. Complications of temporary percutaneous mechanical circulatory support for cardiogenic shock: an appraisal of contemporary literature. *Cardiol Ther.* 2019;8(2):211–228. https://doi.org/10.1007/s40119-019-00152-8

6. van Diepen S, Katz JN, Albert NM, et al. Contemporary management of cardiogenic shock: a scientific statement from the American Heart Association. *Circulation*. 2017;136(16):e232–e268. https://doi.org/10.1161/CIR.0000000000000525

7. Skouri HN, Dec GW, Friedrich MG, Cooper LT. Noninvasive imaging in myocarditis. *J Am Coll Cardiol*. 2006;48(10):2085–2093. https://doi.org/10.1016/j.jacc.2006.08.017

8. Xie Y, Xu E, Bowe B, Al-Aly Z. Long-term cardiovascular outcomes of COVID-19. *Nat Med*. 2022. https://doi.org/10.1038/s41591-022-01689-3

9. McDonagh TA, Metra M, Adamo M, et al. 2021 ESC guidelines for the diagnosis and treatment of acute and chronic heart failure: developed by the Task Force for the Diagnosis and Treatment of Acute and Chronic Heart Failure of the European Society of Cardiology (ESC). *Eur J Heart Fail*. 2022;24(1):4–131. https://doi.org/10.1002/ejhf.2333

10. Tschöpe C, Cooper LT, Torre-Amione G, Van Linthout S. Management of myocarditis-related cardiomyopathy in adults. *Circ Res*. 2019;124(11): 1568–1583. https://doi.org/10.1161/CIRCRESAHA.118.313578

11. Ammirati E, Frigerio M, Adler ED, et al. Management of acute myocarditis and chronic inflammatory cardiomyopathy: an expert consensus document. *Circ Heart Fail*. 2020;13(11):e007405. https://doi.org/10.1161/CIRCHEARTFAILURE.120.007405

12. Ferreira VM, Schulz-Menger J, Holmvang G, et al. Cardiovascular magnetic resonance in non-ischemic myocardial inflammation: expert recommendations. *J Am Coll Cardiol*. 2018;72(24):3158–3176. https://doi.org/10.1016/j.jacc.2018.09.072

13. Messroghli DR, Moon JC, Ferreira VM, et al. Clinical recommendations for cardiovascular magnetic resonance mapping of T1, T2, T2* and extracellular volume: a consensus statement by the Society for Cardiovascular Magnetic Resonance (SCMR) endorsed by the European Association for Cardiovascular Imaging (EACVI). *J Cardiovasc Magn Reson*. 2017;19(1):75. https://doi.org/10.1186/s12968-017-0389-8

14. Friedrich MG, Sechtem U, Schulz-Menger J, et al. Cardiovascular magnetic resonance in myocarditis: a JACC white paper. *J Am Coll Cardiol*. 2009;53(17):1475–1487. https://doi.org/10.1016/j.jacc.2009.02.007

15. Yang F, Wang J, Li W, et al. The prognostic value of late gadolinium enhancement in myocarditis and clinically suspected myocarditis: systematic review and meta-analysis. *Eur Radiol*. 2020;30(5):2616–2626. https://doi.org/10.1007/s00330-019-06643-5

16. Baughman KL. Diagnosis of myocarditis: death of Dallas criteria. *Circulation*. 2006;113(4):593–595. https://doi.org/10.1161/CIRCULATIONAHA.105.589663

17. Peretto G, Sala S, Rizzo S, et al. Arrhythmias in myocarditis: state of the art. *Heart Rhythm*. 2019;16(5):793–801. https://doi.org/10.1016/j.hrthm.2018.11.024

18. Maron BJ, Udelson JE, Bonow RO, et al. Eligibility and disqualification recommendations for competitive athletes with cardiovascular abnormalities: task force 3: hypertrophic cardiomyopathy, arrhythmogenic right ventricular cardiomyopathy and other cardiomyopathies, and myocarditis: a scientific statement from the American Heart Association and American College of Cardiology. *Circulation*. 2015;132(22):e273–e280. https://doi.org/10.1161/CIR.0000000000000239

19. Naneishvili T, Khalil A, O'Leary R, Prasad N. Fulminant myocarditis as an early presentation of SARS-CoV-2. *BMJ Case Rep*. 2020;13(9): e237553. https://doi.org/10.1136/bcr-2020-237553

20. Guzik TJ, Mohiddin SA, Dimarco A, et al. COVID-19 and the cardiovascular system: implications for risk assessment, diagnosis, and treatment options. *Cardiovasc Res*. 2020;116(10): 1666–1687. https://doi.org/10.1093/cvr/cvaa106

21. Halushka MK, Vander Heide RS. Myocarditis is rare in COVID-19 autopsies: cardiovascular findings across 277 postmortem examinations. *Cardiovasc Pathol*. 2021;50:107300. https://doi.org/10.1016/j.carpath.2020.107300

22. Rink LD, Daniels CJ, Boersma D, et al. Competitive sports, the coronavirus disease 2019 pandemic, and big ten athletics. *Circ Cardiovasc Qual Outcomes*. 2020;13(12):e007608. https://doi.org/10.1161/CIRCOUTCOMES.120.007608

23. Clark DE, Parikh A, Dendy JM, et al. COVID-19 Myocardial Pathology Evaluation in AthleTEs with Cardiac Magnetic Resonance (COMPETE CMR). *Circulation*. 2021;143(6):609–612. https://doi.org/10.1161/CIRCULATIONAHA.120.052573

24. Gluckman TJ, Bhave NM, Allen LA, et al. 2022 ACC expert consensus decision pathway on cardiovascular sequelae of COVID-19 in adults: myocarditis and other myocardial involvement, post-acute sequelae of SARS-CoV-2 infection, and return to play. *J Am Coll Cardiol*. 2022;79(17): 1717–1756.

KEY WORDS COVID-19, fulminant myocarditis, ventricular arrhythmias

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