

## CARDIOVASCULAR CASE SERIES

# The Variety of Cardiovascular Presentations of COVID-19

Information about a real patient is presented in stages (boldface type) to expert clinicians (Drs Uriel and Sayer), who respond to the information and share their reasoning with the reader (regular type). A discussion by the authors follows.

**T**he global pandemic caused by coronavirus disease 2019 (COVID-19) has affected more than 880 000 people in over 180 countries or regions worldwide.<sup>1</sup> COVID-19 is the clinical manifestation of infection with severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2) and most frequently presents with respiratory symptoms that can progress to pneumonia and, in severe cases, acute respiratory distress syndrome and shock. However, there is increasing awareness of the cardiovascular manifestations of COVID-19 disease and the adverse impact that cardiovascular involvement has on prognosis.<sup>2</sup> Discriminating between a cardiac or respiratory etiology of symptoms can be challenging since each may present predominantly with dyspnea. It is also critical to recognize when cardiac and pulmonary involvement coexist. In this paper, we present 4 cases that illustrate a variety of cardiovascular presentations of COVID-19 infection. In addition to discussing the basic clinical physiology, we also discuss clinical decision making in the current environment, while considering resource allocation and the welfare of healthcare professionals.

### CASE 1: CHEST PAIN AND ST ELEVATION

#### Patient Presentation

**A 64-year-old woman with a history of hypertension and hyperlipidemia and no known exposure to SARS-CoV-2 presented with persistent chest pressure for 2 days. She denied dyspnea, cough, fever, chills, diarrhea, recent travel, or sick contacts. On admission, she was afebrile (37.1°C), her blood pressure was 130/80 mmHg, heart rate 98 bpm, and oxygen saturation 100% on 2 L of oxygen. The initial ECG showed sinus tachycardia at 102 bpm, low voltage QRS complexes in the limb leads, ST segment elevations in leads I, II, aVL, V2–V6, and PR elevation and ST depressions in aVR (Figure 1A). Troponin I on admission was 7.9 ng/mL. She was brought to the cardiac catheterization laboratory where angiography demonstrated nonobstructive coronary artery disease (Figure 1B). During the procedure the patient's blood pressure fell to 72/43 mmHg. Right heart catheterization was performed revealing a right atrial pressure of 10 mmHg, pulmonary artery pressure of 30/20 mmHg, pulmonary capillary wedge pressure of 21 mmHg, and a Fick cardiac index of 1.0 L·min<sup>-1</sup>·m<sup>-2</sup>, confirming the diagnosis of cardiogenic shock. An intraaortic balloon pump (IABP) was inserted and dobutamine infusion was started with rapid normalization of blood pressure.**

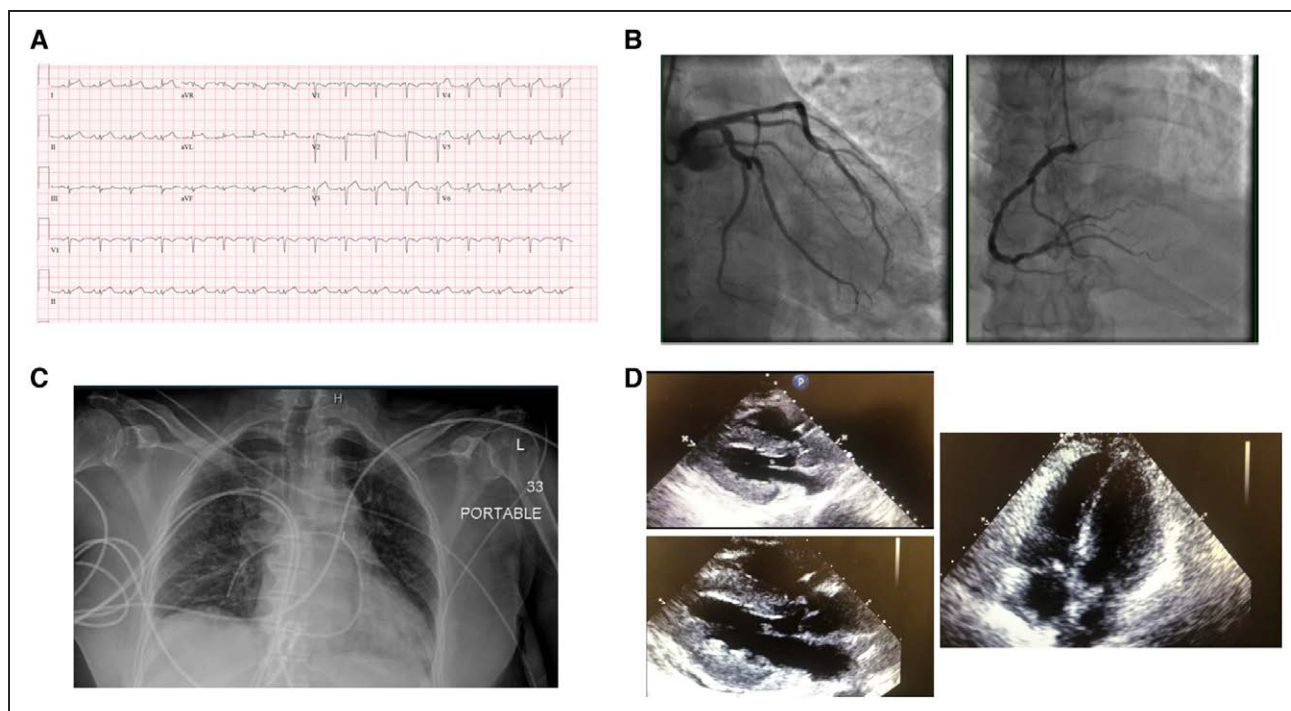
**Justin A. Fried, MD  
Kumudha Ramasubbu, MD  
Reema Bhatt, MD  
Veli K. Topkara, MD, MSc  
Kevin J. Clerkin, MD, MSc  
Evelyn Horn, MD  
LeRoy Rabbani, MD  
Daniel Brodie, MD  
Sneha S. Jain, MD, MBA  
Ajay J. Kirtane, MD, SM  
Amirali Masoumi, MD  
Koji Takeda, MD, PhD  
Deepa Kumaraiah, MD, MBA  
Daniel Burkhoff, MD, PhD  
Martin Leon, MD  
Allan Schwartz, MD  
Nir Uriel, MD, MSc\*  
Gabriel Sayer, MD\***

\*Drs Uriel and Sayer are co-senior authors.

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**Figure 1. Chest pain and ST elevation.**

Initial ECG showed sinus tachycardia, low-voltage QRS complexes in the limb leads, and diffuse ST elevation in leads I, II, aVL, and leads V2–V6 (A). Coronary angiogram demonstrated mild disease in the left anterior descending artery and left circumflex artery and 40% stenosis in the mid-right coronary artery (B). Chest radiography demonstrated clear lungs (C). Transthoracic echocardiogram with severe increased left ventricular wall thickness and left ventricular ejection fraction approximately 30% with trace circumferential pericardial effusion (D).

Chest radiography demonstrated normal heart size and clear lungs (Figure 1C). Transthoracic echocardiography (TTE) demonstrated a small left ventricular end-diastolic dimension of 2.9 cm, severe concentric left ventricular hypertrophy, and left ventricular ejection fraction (LVEF) of 30%, with a dilated, severely hypokinetic right ventricle. No significant valve lesions or dysfunction were noted. A small circumferential pericardial effusion was noted (Figure 1D). Laboratory analysis revealed an arterial lactate of 5, ferritin of 967  $\mu\text{g/L}$ , C-reactive protein of 54  $\text{ng/mL}$ , and D-dimer 166  $\text{ng/mL}$  (normal). There was also a small increase in M protein on serum electrophoresis, and the ratio of kappa to lambda free light chains was 2.15.

The differential diagnosis included myopericarditis and cardiac amyloidosis. SARS-CoV-2 testing was positive. She was started on oral hydroxychloroquine 600 mg every 12 hours for 1 day followed by 400 mg daily for 4 additional days. On IABP and dobutamine infusion, her cardiac index and lactate normalized and her end-organ function remained stable. The troponin-I peaked at 18.6  $\text{ng/mL}$  and subsequently trended down to 0.4  $\text{ng/mL}$ . The IABP was weaned after 7 days and the patient remained hemodynamically stable off IABP and inotropes. On repeat echocardiography

on hospital day 10, LVEF improved to 50% and wall thickness was reduced.

## Discussion

*Dr Uriel:* The predominant presenting symptoms of this patient were cardiac in nature without symptoms suggestive of infection. Thus, although the initial ECG findings of diffuse ST elevations and elevated cardiac enzymes raised the possibility of myopericarditis, the typical diagnostic algorithm for acute coronary syndrome was followed, since this remains a more common cause of this presentation.

As experience with the COVID-19 pandemic grows, the treatment pathway for cardiac presentations may also evolve rapidly. One area of uncertainty is whether to proceed to coronary angiography in response to ECG changes and positive troponin, which risks exposing additional healthcare personnel to infection.<sup>3</sup> Likewise, the role of endomyocardial biopsy to detect myocarditis is uncertain. In this case, hemodynamic assessment played a crucial role in the detection of profound cardiogenic shock, guiding appropriate management to achieve a successful outcome. To reduce the potential contamination associated with patient transport to the catheterization suite, bedside placement of a pulmonary artery catheter and IABP may be considered when coronary angiography is not required. Other potential

indicators of the hemodynamic status may include sampling the central venous saturation or hemodynamic assessment via echocardiography. Whether the increased wall thickness present in this patient will be characteristic of myocarditis-like presentations with COVID-19 will need further study. A low threshold to assess for shock in acute systolic heart failure associated with COVID is important. Likewise, there should be a low threshold for SARS-CoV-2 testing in patients presenting with signs of myopericarditis even in the absence of fever and respiratory symptoms. Although the patient in this case improved in the short term, the long-term effects of the myocardial injury are still to be determined.

## CASE 2: CARDIOGENIC SHOCK RESCUED BY VENO-ARTERIAL-VENOUS EXTRACORPOREAL MEMBRANE OXYGENATION

### Patient Presentation

A 38-year-old man with a history of type 2 diabetes mellitus presented with 1 week of cough, pleuritic chest pain, and progressive shortness of breath to an outside hospital. On presentation, he was tachypneic with an oxygen saturation of 93% on room air. Initial labs were notable for a white blood cell count of 9000 per  $\mu\text{L}$  with 11% band forms, 11% lymphocytes, a venous lactate of 1.7 mmol/L, and normal renal and liver function. The chest radiograph showed bilateral pulmonary opacities. Testing for SARS-CoV-2 was positive.

Over the course of the next 6 hours, his respiratory status rapidly deteriorated, requiring intubation for hypoxemic respiratory failure. TTE demonstrated normal left ventricular function. He developed a supraventricular tachycardia with a rate  $>200$  bpm and was successfully cardioverted. Despite deep sedation and paralysis, the arterial blood gas revealed pH 7.26,  $\text{Pco}_2$  40 mmHg, and  $\text{Po}_2$  56 mmHg on 100% fraction of inspired oxygen and positive end-expiratory pressure of 16 mmHg.

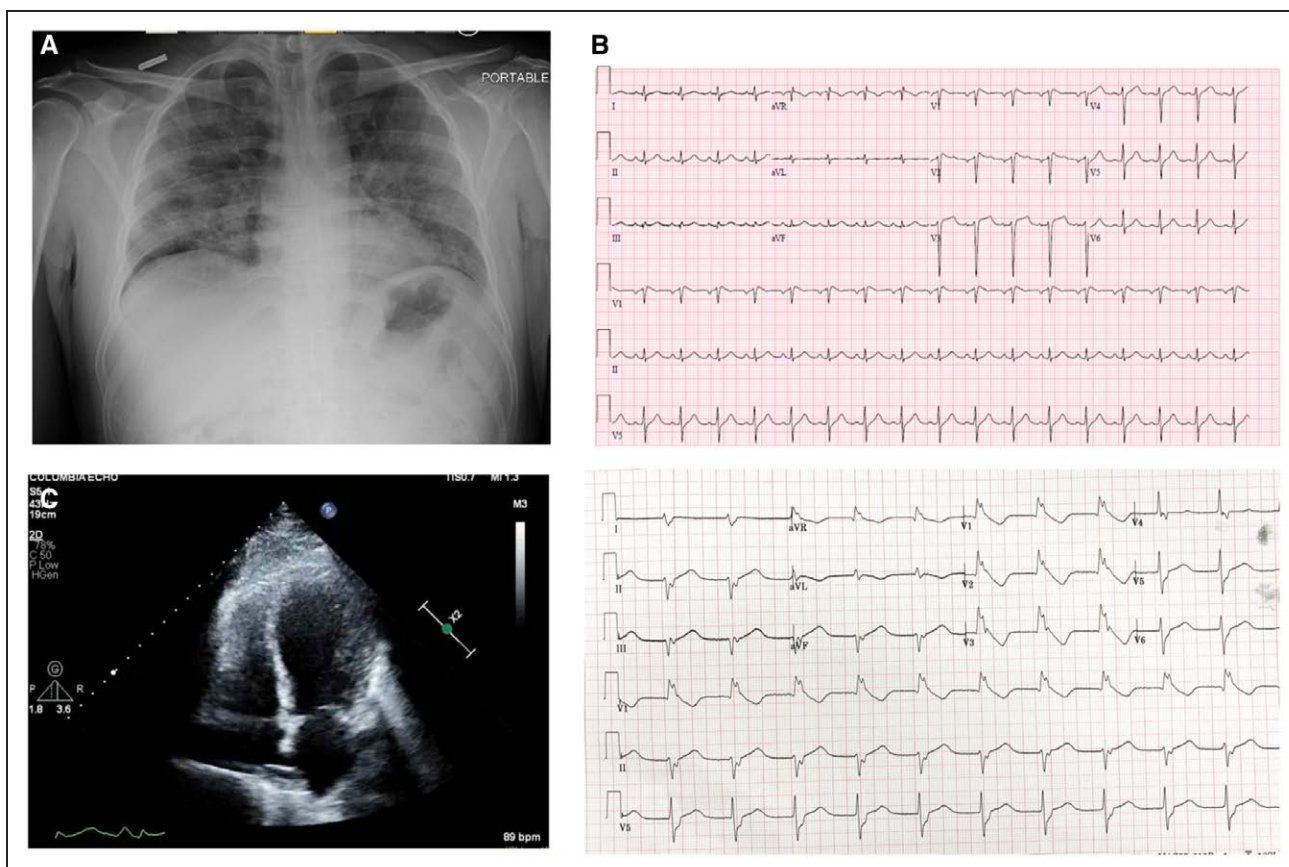
Our mobile extracorporeal membrane oxygenation (ECMO) team was rapidly dispatched. Before ECMO cannulation, the patient had a bradycardic arrest lasting 6 minutes. He was cannulated for venovenous (VV) ECMO with a left femoral 25F drainage cannula and left internal jugular 20F reinfusion cannula. After initiation of VV ECMO, his oxygenation was acceptable but he became hypotensive, requiring vasopressors. On arrival at our institution, his blood pressure was 85/70 mmHg and laboratory analysis showed an arterial lactate of 5.1 mmol/L, C-reactive protein  $>300$  mg/L, ferritin 4420 ng/mL, and interleukin-6 of 331.8 pg/mL.

The chest radiograph showed diffuse ill-defined airspace opacities bilaterally (Figure 2A). He was started on hydroxychloroquine (same dosing protocol as Case 1).

Over the next 12 hours, the patient had a persistently elevated lactate, ongoing pressor requirement and declining urine output. ECG showed low limb lead voltage, sinus tachycardia and he was noted to have runs of an accelerated idioventricular rhythm (Figure 2B). TTE showed left ventricular end-diastolic dimension of 4.5 cm, LVEF 20% to 25%, with akinesis of the mid-left ventricular segments, and normal right ventricular size with mildly reduced function (Figure 2C). High sensitivity troponin T was 1341 ng/L. After a multidisciplinary discussion, a 15F arterial limb was added via the right femoral artery to convert the circuit to veno-arterial-venous ECMO, achieving 2 L of arterial flow. Systemic heparin was maintained with a goal activated partial thromboplastin time of 45 to 60 seconds. Over the next 24 hours, the pressor requirement decreased and the lactate normalized. The patient was decannulated from ECMO after 7 days and is hemodynamically stable, although he remains on mechanical ventilation.

### Discussion

*Dr Sayer:* The initial presentation of this case was more characteristic of severe COVID-19 disease than Case 1. Acute respiratory distress syndrome with profound hypoxia necessitated treatment with VV ECMO. The cardiac involvement only became evident after the initiation of VV ECMO. The etiology of cardiac dysfunction in this case may be multifactorial. Direct cardiac injury may occur as the result of viral invasion, while the cytokine storm induced by COVID-19 may also have toxic effects on the myocardium.<sup>4</sup> In this case, myocardial stunning after cardiac arrest or stress cardiomyopathy are other potential explanations for the severe left ventricular dysfunction. This case highlights the need for a multidisciplinary approach to these patients with frequent reassessment of response to mechanical circulatory support. Reports of ECMO use in COVID-19 patients are limited, and the outcomes are unknown. Even if ECMO shows clinical utility, it will play a limited role overall in the current pandemic because of the extensive resources required to provide the therapy and the limited availability of those resources.<sup>5</sup> In this case, 2 L/min of blood flow support was sufficient to maintain hemodynamic stability until the inflammatory response subsided. It will be of interest to see if such relatively low levels of hemodynamic support are sufficient in other cases with COVID-19 related cardiogenic shock. A benefit of supporting patients with relatively



**Figure 2.** Cardiogenic shock rescued by veno-arterial-venous extracorporeal membrane oxygenation.

Chest radiograph showed diffuse ill-defined airspace opacities bilaterally (A). Initial ECG (top) demonstrated sinus tachycardia with incomplete right bundle-branch block. Repeat ECG (bottom) demonstrated accelerated idioventricular rhythm (B). Transthoracic echocardiogram demonstrated left ventricular end-diastolic diameter of 4.5 cm, left ventricular ejection fraction 20% to 25%, with akinesis of mid-left ventricular segments (C).

low arterial flow rates is a decreased likelihood of left ventricular distention with its associated consequences.

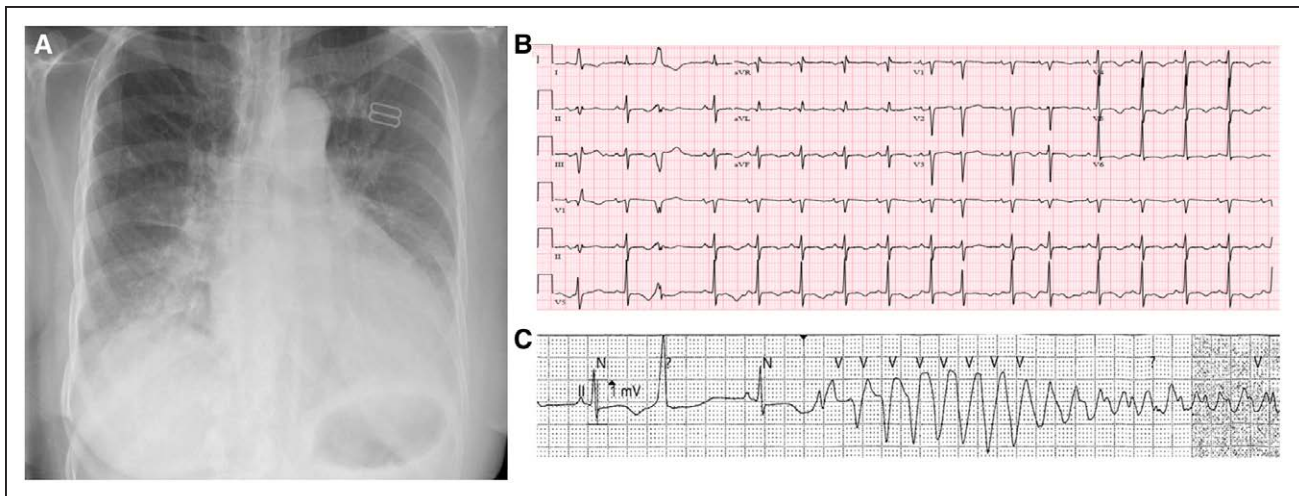
### CASE 3: DECOMPENSATED HEART FAILURE

#### Patient Presentation

A 64-year-old woman with a nonischemic cardiomyopathy (recent normalization of LVEF), atrial fibrillation, hypertension, and diabetes mellitus presented with a nonproductive cough and shortness of breath for 2 days. On arrival, she was afebrile, with blood pressure 153/120 mmHg, heart rate 100 bpm, and oxygen saturation 88%. Chest radiography revealed bibasilar predominant patchy airspace opacities, pulmonary vascular congestion, and small bilateral pleural effusions (Figure 3A). ECG showed sinus rhythm, an isolated premature ventricular complex, premature atrial complexes, lateral T wave inversions, and QTc 528 ms (Figure 3B). Laboratory analysis revealed white blood cell 6100 per  $\mu\text{L}$ , serum creatinine 1.11 mg/dL, aspartate aminotransferase 527 U/L, alanine aminotransferase 232 U/L,

N-terminal pro B-type natriuretic peptide 6137 pg/mL, high-sensitivity troponin T 42 ng/mL, C-reactive protein 19.4 mg/L, ferritin 639 ng/mL, and interleukin-6 12 pg/mL. Testing for SARS-CoV-2 was positive.

She was started on broad spectrum antibiotics for management of pneumonia, including intravenous azithromycin, but hydroxychloroquine was withheld because of the prolonged QT. Her heart failure was treated with intravenous furosemide and intravenous nitroglycerine. Her respiratory status worsened rapidly, requiring intubation. She developed hypotension and was started on vasopressors. An arterial lactate was 6.7 mmol/L. A bedside TTE showed severely reduced left ventricular function. She was ventilated with a low tidal volume strategy. Bedside pulmonary artery catheterization revealed a right atrial pressure of 10 mmHg, pulmonary artery pressure 45/20 mmHg, with a Fick cardiac index of  $1.7 \text{ L}\cdot\text{min}^{-1}\cdot\text{m}^{-2}$ . Dobutamine was started but was discontinued when she developed polymorphic ventricular tachycardia requiring cardioversion (Figure 3C). IABP was considered but deferred because of improvement in the arterial lactate and blood pressure. Troponin levels remained relatively stable



**Figure 3.** Decompensated heart failure.

Chest radiography shows pulmonary vascular congestion, patchy airspace opacities at bases, and bilateral pleural effusions (A). ECG shows sinus rhythm with premature atrial and ventricular complexes, lateral T-wave inversions, and a prolonged QT interval (B). Telemetry strip shows prolonged QT interval and torsades de pointes after R-on-T phenomenon (C).

throughout (peak 214 ng/mL). She remains intubated on day 9 of her hospitalization because of agitation with ventilator weaning attempts.

## Discussion

*Dr Uriel:* In this case, a patient with underlying cardiac disease developed profound decompensation in the context of COVID-19 infection, characterized by a recurrence of a reduced LVEF accompanied by cardiogenic shock and proclivity for tachyarrhythmias. Neither myocarditis nor cytokine storm were probable mediators of the recurrence of her depressed cardiac function, given the relatively low biomarker levels. Whether patients with recovered systolic function will be at similar risk for this recrudescence of reduced LVEF awaits further study. Because of tachyarrhythmias, management of the cardiogenic component of her shock with inotropic agents was not feasible. However, early intubation and reversal of her respiratory failure led to improvements in hemodynamics in the absence of direct cardiac support. This case also highlights the need for rapid correction of hypoxemia in patients with an underlying heart failure, and the usefulness of pulmonary artery catheterization when shock is thought to be multifactorial. In patients with a coexisting cardiomyopathy, baseline QT prolongation may impact consideration of use of hydroxychloroquine and azithromycin.

## CASE 4: HEART TRANSPLANT RECIPIENT

### Patient Presentation

A 51-year-old man with a history of heart transplantation in 2007 and renal transplantation in 2010 presented with intermittent fever, dry cough,

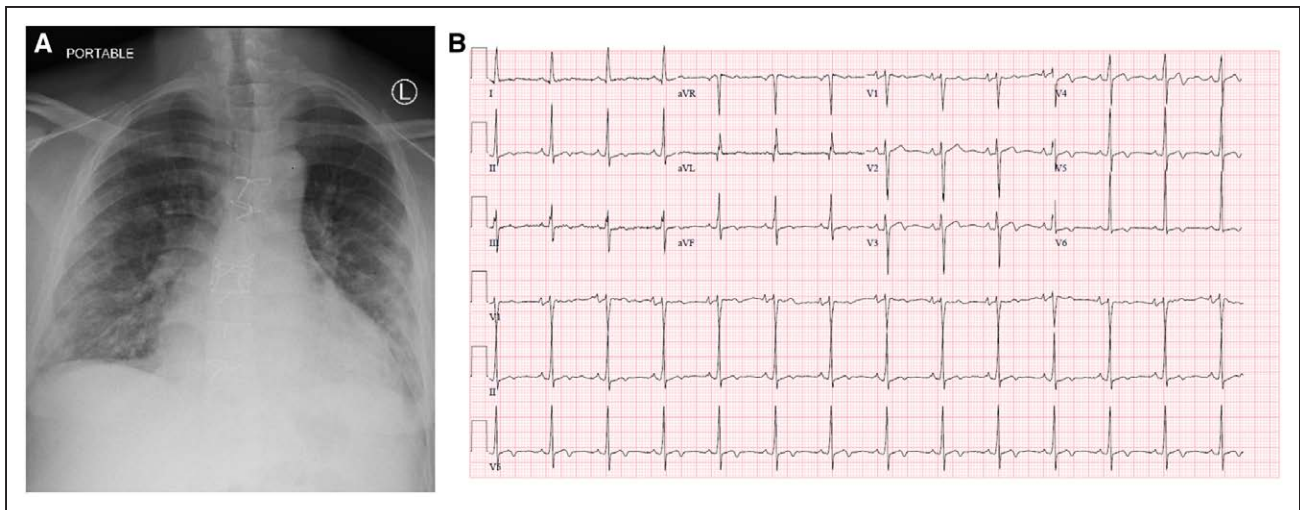
and shortness of breath for 9 days. He denied any recent travel or sick contacts. His outpatient immunosuppression included tacrolimus 5 mg twice daily, mycophenolate mofetil 250 mg twice daily, and prednisone 5 mg daily.

He was afebrile with a heart rate of 84 bpm, blood pressure of 137/84 mmHg, and oxygen saturation of 97% on room air. His chest radiograph was notable for multifocal bilateral patchy airspace opacities (Figure 4A). The ECG demonstrated normal sinus rhythm with new nonspecific T-wave inversions in the inferior and lateral leads (Figure 4B). Laboratory analysis revealed white blood cell 2750 per  $\mu\text{L}$  with lymphopenia and a serum creatinine 4.3 mg/dL. Other notable labs included ferritin 1514 ng/mL, C-reactive protein 129.27 mg/L, interleukin-6 120 pg/mL, D-dimer 1.03  $\mu\text{g}/\text{mL}$ , N-terminal pro B-type natriuretic peptide 3212 pg/mL, and high-sensitivity troponin T 16 ng/L. Testing for SARS-COV-2 was positive.

Following admission, the mycophenolate mofetil was discontinued. The patient was started on hydroxychloroquine (same dose as previous cases) and azithromycin 500 mg daily for treatment of COVID-19. He was also started on ceftriaxone for empirical treatment of pneumonia. TTE showed normal cardiac allograft function. Through the first 5 days of the hospitalization, the patient was intermittently febrile and his inflammatory markers remained persistently elevated, though he remained clinically stable. He was discharged home after 7 days in the hospital.

### Discussion

*Dr Sayer:* The COVID-19 pandemic presents a unique challenge for solid organ transplant recipients. The



**Figure 4. Heart transplant recipient.** Chest radiography with multifocal bilateral patchy airspace opacities (A). ECG with normal sinus rhythm with new nonspecific ST changes and T wave inversions in inferior and lateral leads (B).

clinical symptoms of the patient described in this case were similar to what has been described in nonimmunosuppressed patients with COVID-19. Of note, the patient in this case was more than 2 years posttransplant. At this point, immunosuppression levels are generally much lower than those used in the first months after a transplant. Data are limited about how to adjust

immunosuppression during COVID-19 infection. In this case, we stopped the mycophenolate mofetil during the infection, with a plan to resume it after full recovery. In addition to considerations for transplant recipients, the COVID-19 pandemic has had an additional impact on how heart transplant programs manage their patients on the waitlist. Programs must balance the risks

<p><b><u>ST Segment Elevation</u></b></p> <ul style="list-style-type: none"> <li>• Myopericarditis should be strongly considered in patient with chest pain, ECG changes, and biomarker elevation. Maintain a low threshold to assess for cardiogenic shock in this setting</li> <li>• Use bedside TTE and possibly CCTA to triage cases prior to cardiac catheterization. Consider a conservative strategy in appropriately selected cases</li> <li>• Consider bedside pulmonary artery catheterization and bedside IABP placement. IABP may be preferred device for cardiogenic shock due to lower management requirements</li> <li>• Even if clinical presentation is dominated by cardiac manifestations and there is no fever, COVID-19 should be in differential</li> </ul>	<p><b><u>Cardiogenic Shock</u></b></p> <ul style="list-style-type: none"> <li>• Myocardial dysfunction may be caused by direct injury by virus or secondary to cytokine storm</li> <li>• ECMO provides circulatory (VA) and respiratory support (VV). Low flows on VA ECMO may be sufficient</li> <li>• Stabilization and recovery of profound cardiac dysfunction related to COVID-19 is possible with temporary mechanical circulatory support</li> <li>• ECMO requires high resource utilization and should be used judiciously during the COVID-19 pandemic</li> </ul>
<p><b><u>Decompensated Heart Failure</u></b></p> <ul style="list-style-type: none"> <li>• Preexisting cardiac conditions (congestive heart failure, atrial fibrillation, hypertension) may be exacerbated by COVID-19</li> <li>• Invasive hemodynamic monitoring may be beneficial in select cases to manage both cardiac and respiratory failure</li> <li>• The use of QT-prolonging agents (azithromycin, hydroxychloroquine) should be closely monitored in patients with underlying cardiomyopathies</li> </ul>	<p><b><u>Heart Transplant Recipient</u></b></p> <ul style="list-style-type: none"> <li>• Heart transplant recipients exhibit similar symptoms of COVID-19 infection as non-transplant population</li> <li>• Consider holding anti-metabolite (mycophenolate mofetil or azathioprine) in patients requiring hospitalization for COVID-19 infection</li> <li>• COVID-19 pandemic imposes challenging decisions for heart transplant programs, including maintaining safety of heart failure patients on waitlist and safety of post-transplant patients</li> </ul>

**Figure 5. Important messages from each cardiovascular presentation of COVID-19.** CCTA indicates cardiac computed tomographic angiography; COVID-19, corona virus disease 2019; ECMO, extracorporeal membrane oxygenation; IABP, intra-aortic balloon pump; TTE, transthoracic echocardiogram; VA, veno-arterial; and VV, veno-venous.

of new transplant recipients contracting COVID-19 during their hospitalization with the risks of waitlist mortality if transplantation is postponed. Furthermore, COVID-19 may also impact the donor pool because of diversion of crucial resources and fear of disease transmission from donors.

## TAKE-HOME MESSAGE

These cases illustrate the variable presentation of COVID-19 involvement of the cardiovascular system and highlight evolving considerations for treatment pathways across the spectrum of patients with preexisting cardiovascular diseases (Figure 5). In patients presenting with what appears to be a typical cardiac syndrome, COVID-19 infection should be in the differential during the current pandemic, even in the absence of fever or cough. One should have a low threshold to assess for cardiogenic shock in the setting of acute systolic heart failure related to COVID-19. If inotropic support fails in these patients, we consider IABP as the first line mechanical circulatory support device because it requires the least maintenance from medical support staff. When patients on VV ECMO for respiratory support develop superimposed cardiogenic shock, the addition of an arterial conduit at relatively low blood flow rates may provide the necessary circulatory support without inducing left ventricular distension. Our experience confirms that rescue of patients even with profound cardiogenic or mixed shock may be possible with temporary hemodynamic support at centers with availability of such devices.

COVID-19 infection can cause decompensation of underlying heart failure, and may lead to mixed shock. Invasive hemodynamic monitoring, if feasible, may be helpful to manage the cardiac component of shock in such cases. Medications that prolong the QT interval are being considered for COVID-19 patients and may require closer monitoring in patients with underlying structural heart disease. Our heart transplant recipient exhibited similar symptoms of COVID-19 infection as compared to the general population. For those transplant patients requiring hospitalization, how to alter the

antimetabolite and immunosuppression regimens remains uncertain. Furthermore, the COVID-19 pandemic creates a challenge for the management of heart failure patients on the heart transplant waitlist, forcing physicians to balance the risks of delaying transplant with the risks of donor infection and uncertainty regarding the impact of posttransplant immunosuppression protocols.

## ARTICLE INFORMATION

### Correspondence

Nir Uriel, MD, MSc, FACC, Columbia University Irving Medical Center, Weill Cornell Medicine, 622 West 168th Street, PH4-129, New York, NY 10032. Email nu2126@cumc.columbia.edu

### Affiliations

Department of Medicine, Division of Cardiology (J.A.F., V.K.T., K.J.C., L.R., S.S.J., A.K., A.M., D.K., D.B., M.L., A.S., N.U., G.S.); Division of Pulmonary, Allergy and Critical Care Medicine (D.B.); and Department of Surgery, Division of Cardiac Surgery (K.T.), Columbia University Vagelos College of Physicians and Surgeons, New York. NewYork-Presbyterian Brooklyn Methodist Hospital, Brooklyn (K.R.). Department of Medicine, Division of Cardiology, Weill Cornell Medicine, New York (R.B.).

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Dr Brodie reports receiving grants from Alung Technologies, and serving on the medical advisory boards for Alung Technologies, Xenios, Breathe, Baxter, and Hemovent. The other authors report no conflicts.

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