

Acute Right Ventricular Dysfunction in a Critically Ill Patient with COVID-19



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INTRODUCTION

The full spectrum of coronavirus disease 2019 (COVID-19) caused by severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2) is still emerging. Although acute respiratory distress syndrome (ARDS) is the most common complication of patients with COVID-19, recent studies have suggested involvement of other organs. Cardiac complications from COVID-19 may manifest as myocarditis, myocardial infarction, arrhythmias, or stress cardiomyopathy.¹⁻³ In this report, we present a case of acute right ventricular (RV) failure as a contributing factor for hemodynamic collapse in a patient with COVID-19.

CASE PRESENTATION

A 67-year-old man presented to the hospital for a 4-day history of malaise, anorexia, dry cough, and dyspnea. He was found to be in severe respiratory distress at the time of arrival to the emergency department, with oxygen saturation of 79%. Chest radiography showed bilateral interstitial infiltrates with right lower lobe consolidation. He was in shock, with an elevated lactic acid of 7.8 mmol/L. He required endotracheal intubation, mechanical ventilation, fluid resuscitation, antibiotics, and vasopressor support for septic shock secondary to presumed community-acquired pneumonia.

The patient had a significant history of malignancies, including hepatocellular carcinoma, rectal adenocarcinoma, and non-small-cell lung cancer, for which he underwent surgical resections and chemotherapy. He was found to be in remission during an office visit to his oncologist 1 month before the present hospitalization. He also had a history of chronic obstructive lung disease, chronic kidney disease (stage 3), and diabetes mellitus.

Because of his risk factors, the patient was screened for SARS-CoV-2 infection, and the nasopharyngeal swab specimen for real-time polymerase chain reaction result for SARS-CoV-2 was positive. Because of ongoing shock, echocardiography was performed and showed preserved left ventricular ejection fraction but a severely dilated right ventricle with reduced RV function, flattening of the interventricular septum, severe tricuspid regurgitation, and an estimated systolic

pulmonary artery pressure of 56 mm Hg (Figures 1 and 2, Videos 1 and 2, Table 1).

An echocardiogram obtained 4 years previously showed normal RV function. D-dimer was elevated at >4.0 mg/L (reference value, <0.42 mg/L). High-sensitivity troponin at baseline was 275 ng/L (reference value, <15 ng/L), and the subsequent 6-hour value was 255 ng/L. An electrocardiogram obtained at admission showed normal sinus rhythm without significant ST-T-wave changes. The patient was started on therapeutic anticoagulation because of high clinical suspicion of venous thromboembolism risk associated with COVID-19 and elevated D-dimer level. Venous duplex studies showed acute deep vein thrombosis in the left posterior tibial vein. Computed tomographic angiography of the chest showed subsegmental right upper lobe pulmonary embolism.

The patient required neuromuscular blockade and prone-position ventilation to support his respiratory failure from ARDS due to COVID-19 (mechanical ventilatory support included tidal volumes between 5 and 7 ml/kg of ideal body weight, positive end-expiratory pressure between 10 and 14 cm H₂O, and plateau pressure between 26 and 32 cm H₂O). He required vasopressor support with norepinephrine and vasopressin. His hospital course was complicated by acute kidney injury requiring continuous renal replacement therapy, shock liver, gastrointestinal hemorrhage, *Escherichia coli* pneumonia, and bacteremia. Because of the patient's poor prognosis, his family opted for comfort measures and withdrawal of life-support interventions, and he passed away after 22 days in the intensive care unit (ICU).

DISCUSSION

COVID-19 has posed numerous challenges to clinicians worldwide because of its varied clinical presentations, increased incidence of acute organ dysfunction, and lack of effective therapeutic options. Because of the risks of patient transport and aerosolized viral particle exposure associated with ventilator circuit disconnection during patient transport, bedside echocardiography has emerged as a valuable tool in the early diagnosis and management of critically ill patients with COVID-19.

The majority of patients with SARS-CoV-2 infection who were admitted to the ICU had ARDS and required mechanical ventilation support.⁴ Patients requiring mechanical ventilator support had very high mortality.⁵ The proinflammatory and hypercoagulable state caused by SARS-CoV-2 infection has been implicated in the increased incidence of thrombotic complications in these patients, which may manifest as pulmonary embolism, myocardial infarction, and stroke.⁶⁻⁸

Acute RV dysfunction could develop in critically ill patients for a multitude of reasons, of which acute pulmonary embolism and acute cor pulmonale (ACP) are important etiologies. ACP is defined as RV dysfunction due to an acute increase in RV afterload, and ARDS is one of the most common causes of ACP in the ICU. The diagnosis of ACP relies on echocardiographic findings of septal dyskinesia with RV

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VIDEO HIGHLIGHTS

Video 1: Flattened interventricular septum suggestive of RV volume overload (parasternal short-axis view).

Video 2: Apical four-chamber view showing dilated right ventricle. Left pleural effusion and lung consolidation are also seen in the image (top left portion of the image).

[View the video content online at www.cvcasejournal.com.](http://www.cvcasejournal.com)

dilation (end-diastolic RV/left ventricular area ratio > 0.6) in the appropriate clinical setting.^{9,10} The incidence of ACP is estimated to be about 20% to 25% in patients with ARDS who are ventilated with lung-protective settings.¹¹ Although ACP is associated with deleterious impact on hemodynamics, its association with adverse clinical outcomes has not been consistent.¹² In contrast, the presence of RV dysfunction has been associated with increased mortality in patients with acute pulmonary embolism, including those who are hemodynamically stable.¹³

Our patient had acute RV dysfunction, likely due to increased RV afterload from a combination of ARDS and pulmonary thromboembolism. The amount of clot burden, as noticed on his imaging studies, is thought to be insufficient to explain the severity of RV dysfunction. Patients with SARS-CoV-2 infection are thought to be at a lower risk for ACP because of blunted hypoxic pulmonary vasoconstriction and higher lung compliance.¹⁴ However, a recent case series reported the presence of ACP in critically patients with COVID-19 with increased risk for cardiac arrest and mortality.¹⁵ It is plausible that the combination of thromboembolism, viral myocardial injury, and high transpulmonary pressure from severe ARDS could place a subgroup of patients with SARS-CoV-2 infection at an increased risk for RV dysfunction; however, the true prevalence of ACP in hospitalized patients with SARS-CoV-2 infection is unknown. Our patient had high plateau pressures and hypercapnia during his ICU course, which are associated with the development of ACP.¹² Direct myocardial injury with the presence of SARS-CoV-2 viral particles in the myocardial interstitial cells has recently been reported in the literature.¹⁶ We did not pursue endomyocardial biopsy, as the RV dysfunction was thought to be most likely secondary to ACP, and it would not have altered the management of the patient. Moreover, endomyocardial biopsy would increase the risk for disease transmission during transportation from designated COVID-19 unit and increases the direct contamination of the cardiac catheterization laboratory. Although direct RV injury due to the virus cannot be excluded, it does not appear to be the primary mechanism on the basis of the overall clinical picture. Thrombolytic use has been suggested as a therapeutic option in patients with COVID-19 with acute RV dysfunction, especially because of increased incidence of elevated D-dimer and venous thromboembolism in these patients.¹⁵ Our patient did not receive thrombolysis, because of low clot burden as noted in the imaging studies, and increased risk for bleeding complications due to severe thrombocytopenia and multiorgan dysfunction. He was also excluded from being considered for extracorporeal support because of his comorbidities and multiorgan dysfunction.

Literature on the role of echocardiography in the management of patients with COVID-19 is just starting to emerge.^{17,18} In a prospective, systematic, echocardiographic evaluation of consecutive

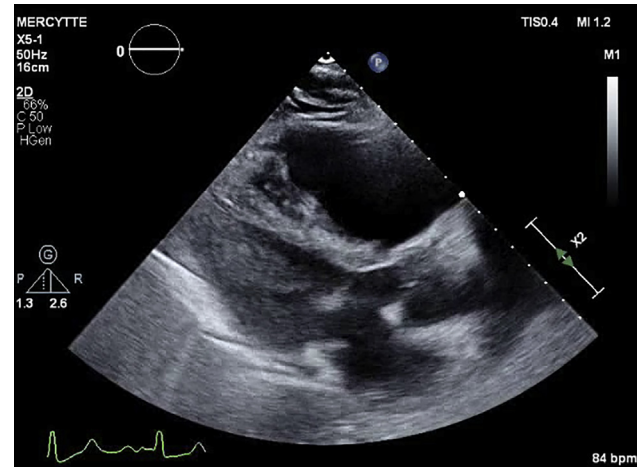


Figure 1 Parasternal long-axis view showing dilated right ventricle.

patients hospitalized with COVID-19, RV dilatation or dysfunction was the most common echocardiographic abnormality.¹⁹ RV dysfunction was also the predominant cause for clinical deterioration in this study. Thus, echocardiography seems to be a valuable tool in diagnosing the reasons for clinical deterioration. The common indications for performing transthoracic echocardiography are hemodynamic assessment and concern for major cardiovascular involvement (e.g., increased cardiac biomarkers, acute coronary syndrome, heart failure, myocarditis).²⁰ It is our practice to incorporate ultrasound examination in the management of any patient with COVID-19 admitted to the ICU with shock requiring vasoactive medications or respiratory failure requiring invasive mechanical ventilatory support. Most of these are point-of-care ultrasound (POCUS) examinations done by critical care physicians in accordance with the American Society of Echocardiography POCUS protocol.²¹ Transthoracic echocardiography would be requested if image acquisition were difficult, or to confirm an abnormality found on POCUS. An on-call cardiologist assessed the clinical appropriateness of transthoracic echocardiography after reviewing the case and discussing it with the ICU team.

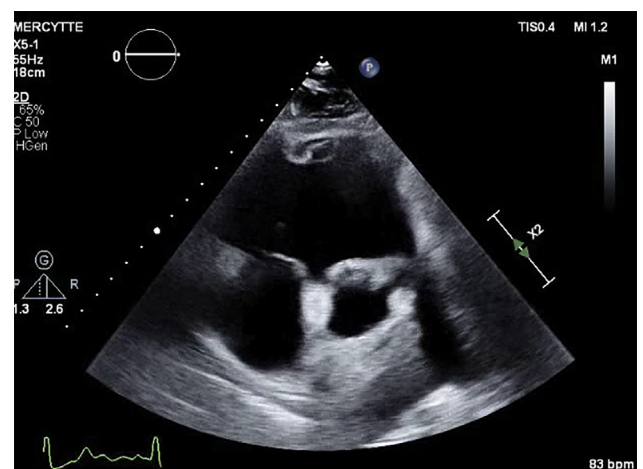


Figure 2 Parasternal short-axis view showing dilated right ventricle.

Table 1 Transthoracic echocardiographic findings

Variable	Patient's value	Reference value or range
RV diameter, base, cm	4.71	<4.2
RV diameter, mid cavity, cm	3.99	<3.5
Right ventricle fractional area change, %	3.21	>35
Tricuspid annular plane systolic excursion, cm	1.46	>1.6
Tricuspid regurgitant jet velocity, m/sec	3.41	≤2.8
Inferior vena cava diameter, cm	2.63	<2.1
Inferior vena cava collapsibility (patient mechanically ventilated), %	<50	

Sonographers used airborne precautions (N-95 masks, face shields, head caps, isolation gowns, shoe covers, and hand gloves) when performing echocardiography on patients with COVID-19. Only one echocardiography machine (iE33; Philips Medical Systems, Andover, MA) was used to scan patients with COVID-19. It was stored in a designated room when not in use. The entire echocardiographic machine was cleaned with disinfectant wipes twice after scanning a COVID-19 patient: once inside the patient's room and immediately after exiting the patient's room. Sonographers were asked to follow institutional protocol for personal protective equipment use and were trained in proper donning and doffing of personal protective equipment. POCUS examinations were done with a CX50 ultrasound machine (Philips Medical Systems), and similar disinfecting protocols were followed after each examination.²¹

CONCLUSION

This case describes one of the severe hemodynamic complications of COVID-19 and highlights the importance of early echocardiography in the diagnosis and management of these patients. Awareness of the potential association between COVID-19 and venous thromboembolic disease is vital for clinicians. Acute RV dysfunction due to increased RV afterload should be included in the differential diagnosis of hemodynamically unstable patients with COVID-19.

SUPPLEMENTARY DATA

Supplementary data related to this article can be found at <https://doi.org/10.1016/j.case.2020.08.007>.

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