Texas Heart Institute Journal

Case Reports

Dilated Cardiomyopathy With Multiple Left Ventricular Thrombi and Embolic Stroke After Mild COVID-19

Radhapriya Yalamanchi, DNB[1]; Madhan Kumar Murugan, MD[2]; Krishnaswamy Chandrasekharan, MD[1]; Refai Showkathali, MRCP[1]

[1]Department of Cardiology, Apollo Main Hospital, Chennai, India
[2]Department of Radiology, Apollo Main Hospital, Chennai, India

COVID-19 is a novel disease with multisystem involvement, but most patients have pulmonary and cardiovascular involvement in the acute stages. The cardiovascular impact of acute COVID-19 is well recognized and ranges from myocarditis, arrhythmias, and thrombotic occlusion of coronary arteries to spontaneous coronary artery dissection and microthrombi in small coronary vessels on autopsy. We report a case of a 37-year-old man who recovered from mild COVID-19 only to present a few weeks later with devastating cardiovascular involvement that included severe left ventricular impairment resulting from nonischemic cardiomyopathy, multiple left ventricular thrombi, and embolic stroke. ***(Tex Heart Inst J. 2022;49(6):e207488)***

Citation: Yalamanchi R, Murugan MK, Chandrasekharan K, Showkathali R. Dilated cardiomyopathy with multiple left ventricular thrombi and embolic stroke after mild COVID-19. *Tex Heart Inst J.* 2022;49(6):e207488. doi:10.14503/THIJ-20-7488

Keywords: COVID-19; SARS-CoV-2; DCM; cardiomyopathy, dilated; coronary thrombosis; stroke; embolic stroke; coronavirus; heart failure; ventricular dysfunction, left

Corresponding author: Refai Showkathali, MRCP, Adjunct Professor, Apollo Hospitals Educational and Research Foundation, Greams Road, Chennai 600 006, India

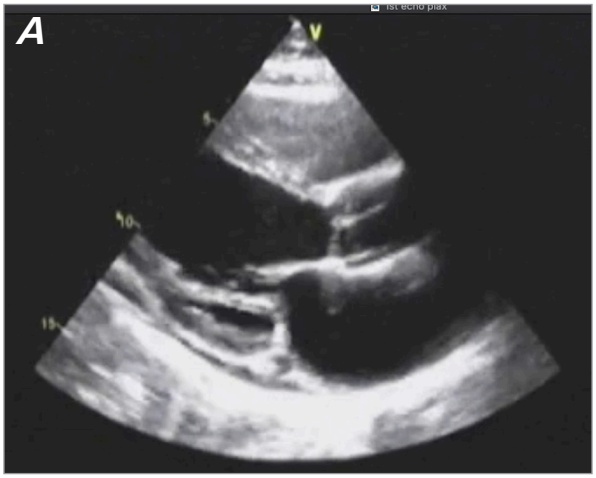
E-mail: refais@gmail.com

Though the acute cardiovascular impact of acute COVID-19 illness was well recognized, the subacute and late cardiac sequelae of COVID-19 were not well recognized globally during the initial COVID-19 wave. We report a case of a 37-year-old man who recovered from mild COVID-19 in June 2020 only to present a few weeks later with significant cardiovascular involvement.

Case Report

A 37-year-old man presented with breathlessness and orthopnea that had been worsening for 2 weeks. He had had symptoms (fever, dry cough, and sore throat) suggestive of COVID-19 a few weeks previously but did not get tested. He self-quarantined and self-medicated with paracetamol. His symptoms improved after a few days, and he was asymptomatic until 2 weeks ago, when he started to develop breathlessness. On examination, his pulse rate was 104/min and sinus, his blood pressure was 110/70 mm Hg, and his oxygen saturation was 97% on room air. Jugular venous pressure was elevated, with pedal edema and mild crepitations in the bases of both lungs. His kidney function was normal (creatinine level, 0.9 mg/dL), and his N-terminal pro–brain natriuretic peptide (NT-proBNP) level was 3,165 pg/mL. His COVID-19 reverse transcriptase–polymerase chain reaction result was negative. The electrocardiogram showed sinus tachycardia with nonspecific Q waves in lead III; an echocardiogram showed a dilated left ventricle (7.1/6.0 cm) and severe global hypokinesia of the left ventricle, with a left ventricular (LV) ejection fraction of 20% by the modified Simpson method but no thrombus (Fig. 1A and 1B). Right ventricular function was also impaired, as indicated by a tricuspid annular plane systolic excursion of 1.1 cm with moderate pulmonary hypertension (60 mm Hg). Because he had no episodes of chest pain and no risk factors for coronary artery disease, a provisional diagnosis of dilated cardiomyopathy (probable viral) was made. The immunoglobulin G antibody testing result with chemiluminescent immunoassay for COVID-19 was negative at this time.

The patient was admitted for treatment of heart failure (HF). Furosemide infusion was given for 48 hours with good clinical response. On repeat testing, his NT-



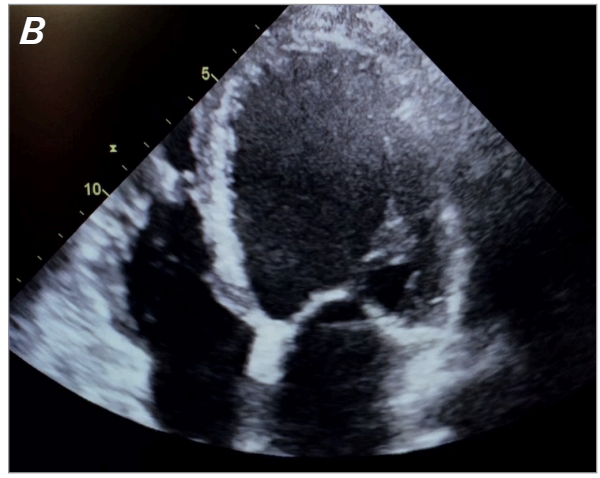


Fig. 1 *Transthoracic echocardiograms in the* ***A****) parasternal long-axis view and* ***B****) apical view show dilated left ventricle with no thrombus.*

Supplemental motion image is available for Figure 1A.

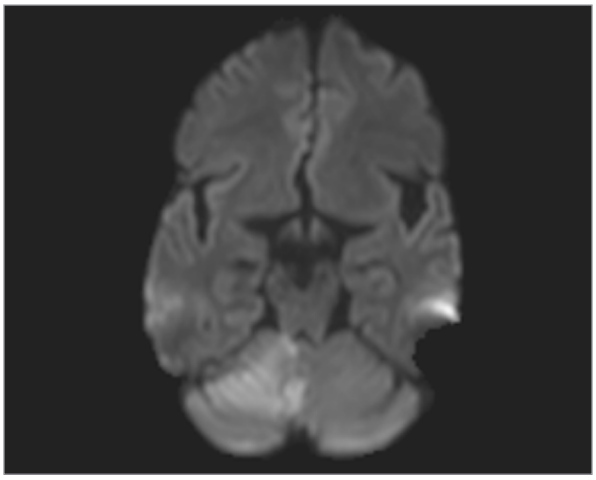


Fig. 2 *Axial, diffusion-weighted magnetic resonance image (b value = 1,000 s/mm^2) shows restricted diffusion in the right cerebellum, indicating acute infarction.*

proBNP level dropped to 1,151 pg/mL within 48 hours of admission. He was started on other HF medications, such as an angiotensin receptor–neprilysin inhibitor, β-blockers, and ivabradine, during his hospital stay. Because of the low index of suspicion for coronary artery disease, he was not given antiplatelet agents or prophylactic anticoagulation because he was ambulatory in the ward. His symptoms improved, and he was to be discharged on day 4 of admission. On the morning of day 4, however, he had an episode of vomiting with a drop in the Glasgow Coma Scale score to 13 (scores range from 3 [worst] to 15 [best]). Urgent magnetic resonance imaging (MRI) of the brain showed an acute infarct in the right superior cerebellar artery involving the right cerebellum and the vermis (Fig. 2). A corresponding magnetic resonance angiogram showed attenuation of the right superior cerebellar artery. An echocardiogram indicated multiple thrombi (1 mobile) of various sizes in the left ventricle, the largest of which was 3.1 × 1.3 cm (Fig. 3A and 3B). Dimerized plasmin fragment D was elevated at 1,024 ng/mL. The patient was started immediately on an infusion of unfractionated heparin in keeping with the protocol for patients with a high activated partial thromboplastin time. On day 5, he had problems with speech, and a plain computed tomography scan of his brain showed a new hypodense lesion in the right frontoparietal region suggesting an acute infarct that was not seen in the previous MRI (Fig. 4). Serial echocardiograms over the next few days showed resolution of the thrombus, with its complete disappearance on day 7. The patient was started on an oral anticoagulant (warfarin). There were no residual neurologic issues other than dysarthria. Cardiac MRI revealed a dilated, dysfunctional left ventricle with no T2-weighted short-τ inversion recovery myocardial edema or delayed gadolinium enhancement (Fig. 5A and 5B). A computed tomography coronary angiogram showed no significant coronary artery stenosis. He was discharged home for follow-up care in our outpatient clinic. His repeat COVID-19 immunoglobulin G antibody test result at his 2-week follow-up was positive,

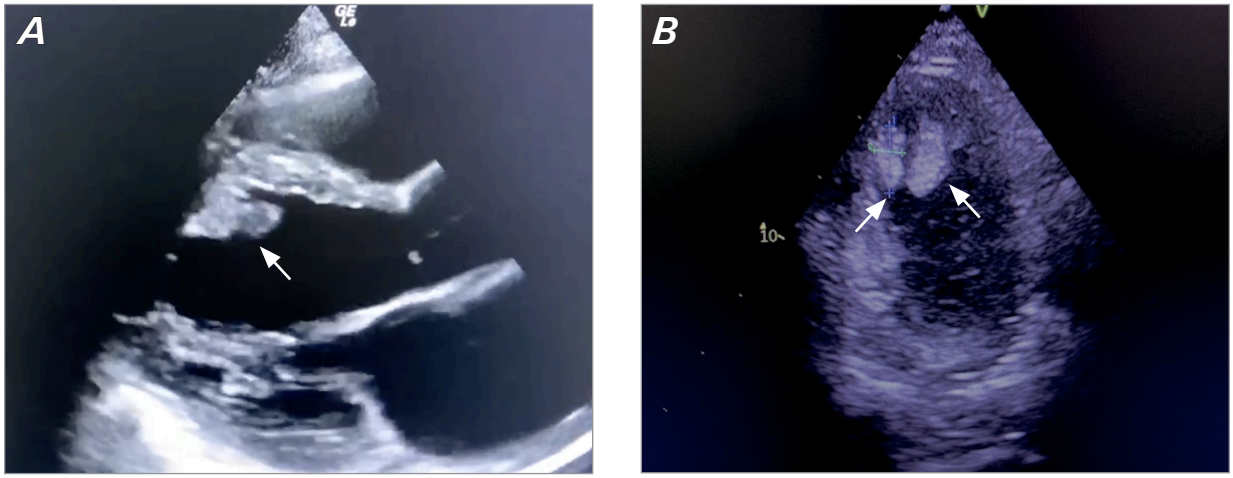


Fig. 3 *Transthoracic echocardiograms in the* ***A****) parasternal long-axis view and* ***B****) apical view show multiple thrombi in the left ventricle (arrows).*

Supplemental motion image is available for Figure 3A.

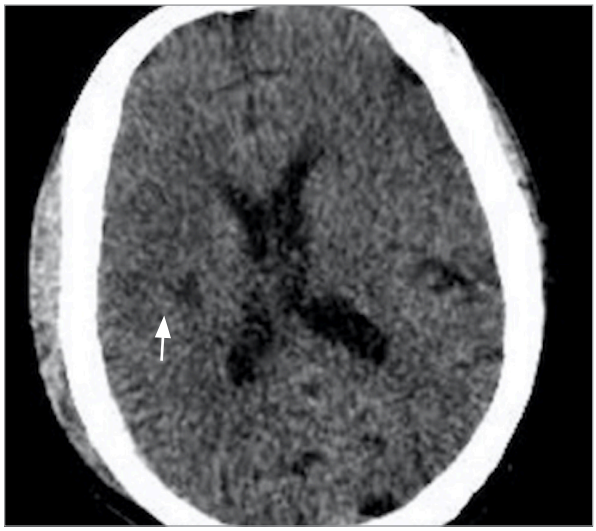


Fig. 4 *Axial computed tomographic brain scan shows a new hypodense lesion (arrow) in the right frontoparietal region, which had not been seen previously on magnetic resonance imaging, suggesting new infarction.*

and an echocardiogram at 1 month showed improved LV function with no thrombus (Fig. 6A and 6B).

Discussion

The most likely cause of HF presentation in this patient was subacute nonischemic cardiomyopathy with severely impaired LV function caused by COVID-19, even though his initial immunoglobulin G antibody test result was negative. The diagnostic accuracy of antibody testing for COVID-19 varies, particularly with the kits available in the United Kingdom and India.[1] The spectrum of cardiac damage COVID-19 causes in the acute stage includes myocarditis leading to acute HF,[2] ventricular arrhythmia,[3] thrombotic occlusion of coronary arteries presenting with myocardial infarction,[4,5] spontaneous coronary artery dissection,[6,7] and the presence of microthrombi in small coronary vessels on autopsy.[8] Recent studies suggest that late-onset cardiac involvement should be considered in patients with COVID-19. In a recent study, cardiac MRI performed on patients who were a mean of 71 days after recovery from mild to moderate COVID-19 (67% home recovery) showed cardiac involvement in 78% of patients, with features of active myocardial inflammation in 60%.[9] Another recently published autopsy study highlights similar findings in 39 patients who had had COVID-19 (35 of whom died of clinical pneumonia); viral involvement in the heart was seen in 61.5% of patients with no histopathologic evidence of acute myocarditis.[10] This finding suggests that COVID-19 is associated with some form of myocardial injury as a result of direct viral infection of the heart.

The patient in this case did not have severe clinical symptoms of COVID-19 that required medical attention, and he recovered completely from the acute illness with conservative management at home. This outcome suggests that he did not have acute myocarditis caused

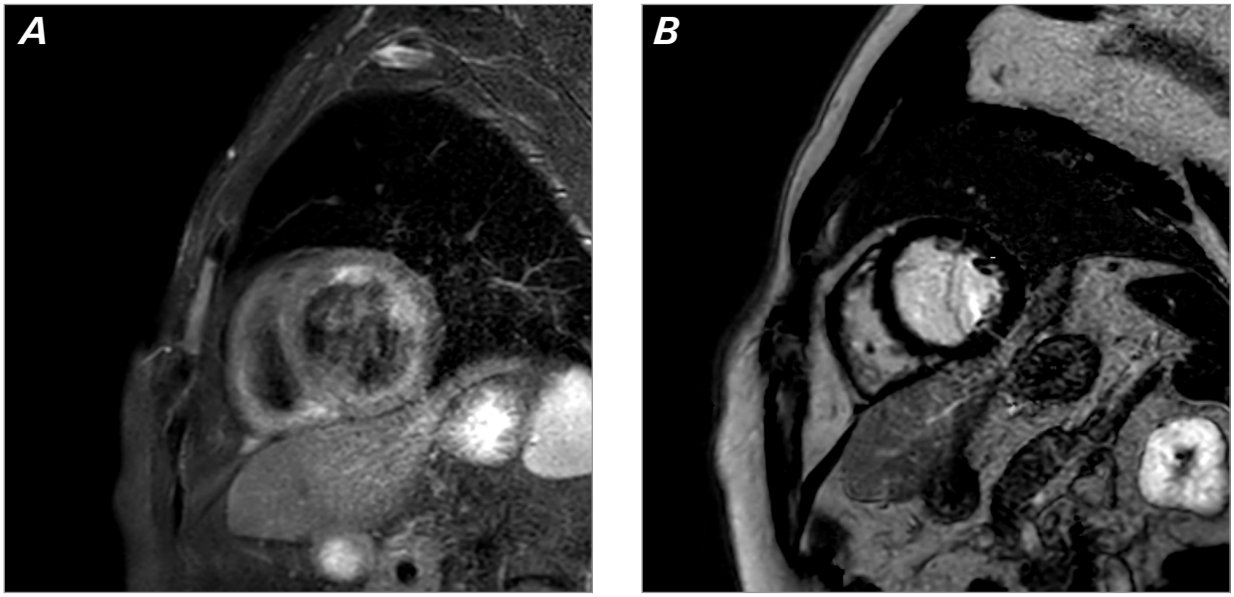


Fig. 5 *Cardiac magnetic resonance images show* ***A****) no definite myocardial edema (T2-weighted short-τ inversion recovery, black blood sequence, short-axis view) or* ***B****) late gadolinium enhancement of left ventricular myocardium (phase-sensitive inversion recovery sequence, short-axis view).*

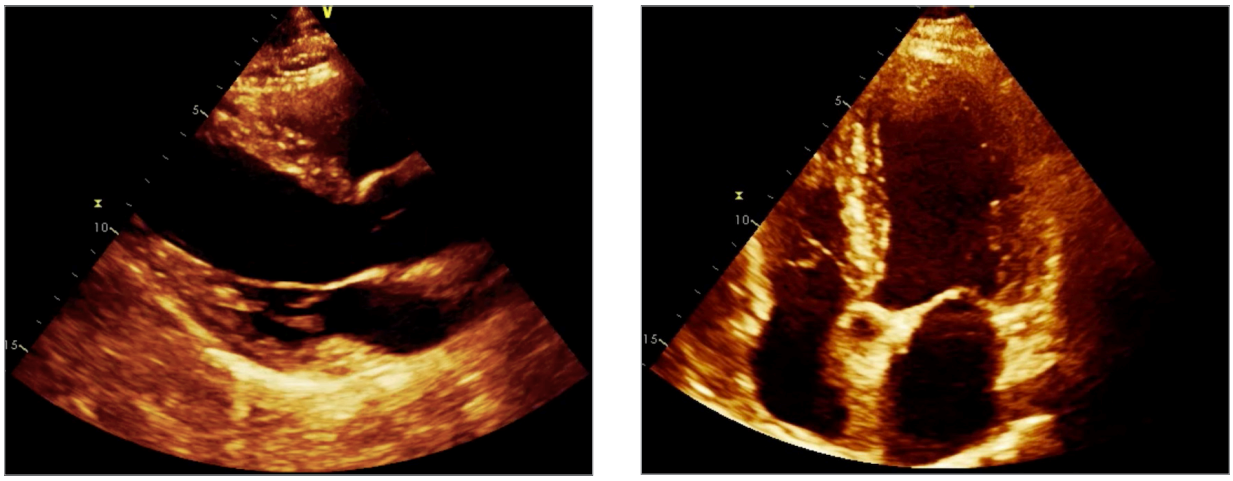


Fig. 6 *At 1-month follow-up, transthoracic echocardiograms in* ***A****) parasternal long-axis view and* ***B****) apical view show nondilated cardiac chambers with no thrombus.*

Supplemental motion image is available for Figure 6A.

by COVID-19 that resulted in severe symptoms or signs. Cardiac MRI did not reveal any classic features of LV myocarditis, such as T2-weighted MRI evidence of edema or gadolinium enhancement. There was no clinical, echocardiographic, or MRI features of Takotsubo cardiomyopathy. The time period between the first echocardiogram without the LV thrombus and the first episode of an embolic stroke (with echocardiogram showing multiple thrombi) was only 4 days. In patients with dilated cardiomyopathy, LV thrombus would generally develop in approximately 2 to 4 weeks. In this patient, the accelerated development of multiple left ventricle thrombi with an embolic phenomenon while on HF therapy supports the presence of a hypercoagulable prothrombotic state, which is well documented in patients with COVID-19.

This case highlights the fulminant cardiac manifestation of COVID-19, even in patients who have clinically recovered from mild disease, and indicates the possibility of the development of late complications in this scenario. We suggest routine cardiac surveillance with echocardiograms for all COVID-19 recovered patients after a few weeks, considering that cardiac MRI is not easily accessible.

Published: 30 November 2022

Conflict of Interest Disclosures: None

Funding/Support: None

References

1. Duong YT, Wright CG, Justman J. Antibody testing for coronavirus disease 2019: not ready for prime time. *BMJ*. 2020;370:m2655. doi:10.1136/bmj.m2655
2. Chen T, Wu D, Chen H, et al. Clinical characteristics of 113 deceased patients with coronavirus disease 2019: retrospective study. *BMJ*. 2020;368:m1091. doi:10.1136/bmj.m1091
3. Guo T, Fan Y, Chen M, et al. Cardiovascular implications of fatal outcomes of patients with coronavirus disease 2019 (COVID-19). *JAMA Cardiol.* 2020:5(7):811-818. doi:10.1001/jamacardio.2020.1017
4. Bangalore S, Sharma A, Slotwiner A, et al. ST-segment elevation in patients with Covid-19—a case series. *N Engl J Med.* 2020;382(25):2478-2480. doi:10.1056/NEJMc2009020
5. Dominguez-Erquicia P, Dobarro D, Raposeiras-Roubín S, Bastos-Fernandez G, Iñiguez- Romo A. Multivessel coronary thrombosis in a patient with COVID-19 pneumonia. *Eur Heart J.* 2020;41(22):2132. doi:10.1093/eurheartj/ehaa393
6. Courand PY, Harbaoui B, Bonnet M, Lantelme P. Spontaneous coronary artery dissection in a patient with COVID-19. *JACC Cardiovasc Interv.* 2020;13(12):e107-e108. doi:10.1016/j.jcin.2020.04.006
7. Kumar K, Vogt JC, Divanji PH, Cigarroa JE. Spontaneous coronary artery dissection of the left anterior descending artery in a patient with COVID-19 infection. *Catheter Cardiovasc Interv.* 2021;97(2):E249-E252. doi:10.1002/ccd.28960
8. Guagliumi G, Sonzogni A, Pescetelli I, Pellegrini D, Finn AV. Microthrombi and ST-segment-elevation myocardial infarction in COVID-19. *Circulation.* 2020;142(8):804-809. doi:10.1161/CIRCULATIONAHA.120.049294
9. Puntmann VO, Carerj ML, Wieters I, et al. Outcomes of cardiovascular magnetic resonance imaging in patients recently recovered from coronavirus disease 2019 (COVID-19). *JAMA Cardiol.* 2020;5(11):1265-1273. doi:10.1001/jamacardio.2020.3557
10. Lindner D, Fitzek A, Bräuninger H, et al. Association of cardiac infection with SARS-CoV-2 in confirmed COVID-19 autopsy cases. *JAMA Cardiol.* 2020;5(11):1281-1285. doi:10.1001/jamacardio.2020.3551