# COVID-19-associated myocarditis presenting as new-onset heart failure and atrial fibrillation

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Accepted 17 June 2021



**To cite:** Gaine S, Devitt P, Coughlan JJ, *et al*. *BMJ Case Rep* 2021;**14**:e244027. doi:10.1136/bcr-2021-244027

## SUMMARY

A 58-year-old man presented to the emergency department with recent-onset palpitations and progressive exertional dyspnoea. ECG demonstrated new-onset atrial fibrillation. Transthoracic echocardiogram showed global impairment in left ventricular systolic function with left ventricular ejection fraction of 20%. Cardiac MRI (CMRI) demonstrated generalised severe myocarditis. A SARS-CoV-2 PCR was positive for SARS-CoV-2 RNA. As such, we diagnosed our patient with COVID-19-associated myocarditis based on CMRI appearances and positive SARS-CoV-2 swab. This case highlights that COVID-19-associated myocarditis can present as new atrial fibrillation and heart failure without the classic COVID-19-associated symptoms.

## BACKGROUND

Myocardial injury associated with COVID-19 has been described and is associated with poor outcome.[1] It is increasingly recognised that patients may present with cardiac involvement without respiratory or infective symptoms that are classically associated with the disease.

The gold standard for diagnosis of myocarditis is endomyocardial biopsy (EMB). However, this is an invasive procedure and often not absolutely necessary to establish a presumptive diagnosis. Our patient remained afebrile throughout admission and was discharged for outpatient follow-up.

The case highlights that COVID-19-associated myocarditis can present as new atrial fibrillation (AF) and heart failure without the classic COVID-19-associated symptoms.

## CASE PRESENTATION

A 58-year-old man with no significant medical history presented to the emergency department with recent-onset palpitations and progressive dyspnoea over the past week. He denied any chest pain or syncope. There was no history of fever, cough or myalgia.

On admission he was tachycardic at 152 beats per minute, his blood pressure was 117/97 mm Hg and his respiratory rate was 14. He was afebrile and his oxygen saturation was 97% on room air. Physical examination revealed an irregularly irregular pulse, bibasal fine crepitations and an elevated JVP (jugular venous pressure). ECG showed AF and no ST segment changes.

His medical history was unremarkable. He took no regular medications. He was a smoker (50 pack-year) and drank 21 units of alcohol per week.

## INVESTIGATIONS

NT-pro-BNP (N-terminal pro-brain natriuretic peptide) on admission was elevated at 3428 pg/mL (normal range: 0–400 pg/mL), and high sensitivity troponin T was 25 ng/L (normal range: 0–14 ng/L) without significant increase on serial data. His white cell count was normal and his C reactive protein was 7 mg/L (normal range: 0–5 mg/L). His chest X-ray was consistent with acute heart failure with cardiomegaly, increased interstitial lung markings and small effusions. Transthoracic echocardiogram revealed a severely impaired left ventricular ejection fraction (LVEF) of 20% and mitral regurgitation (video 1). CT coronary angiography (CTCA) demonstrated minor coronary artery calcification with no obstructive coronary artery disease. Cardiac MRI (CMRI) demonstrated biventricular dysfunction with an LVEF of 30%. STIR sequences showed biventricular oedema and reduced myocardial T1 (figure 1 and video 2). These findings suggested generalised severe myocarditis. A SARS-CoV-2 PCR test was performed and returned positive. An autoimmune screen was performed and returned negative. Viral serology for other potential causes of myocarditis is not routinely performed at our centre as it is not recommended routinely by current guidelines, and as such this was not performed.

## DIFFERENTIAL DIAGNOSIS

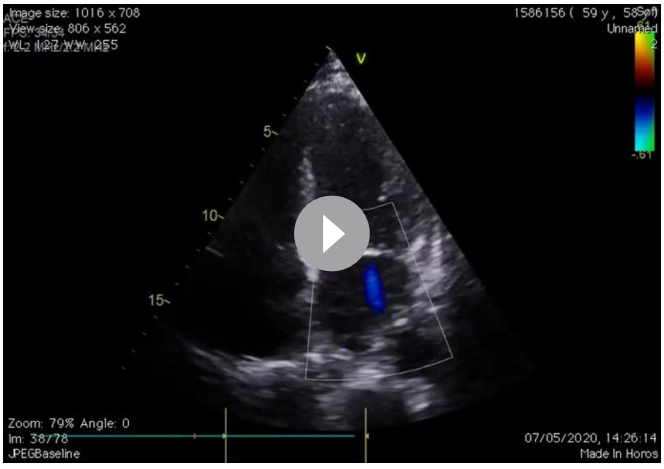
When the patient first presented, clinical examination, history and initial investigations were suggestive of a new diagnosis of congestive cardiac failure. This decompensation was felt to have been triggered or exacerbated by AF with rapid ventricular response.

Given the patient's alcohol history, an alcohol-related cardiomyopathy was considered. In addition, the presence of fast AF meant that a tachycardia-induced cardiomyopathy was also a potential diagnosis.

Given the patient's smoking history, age and sex, an ischaemic cardiomyopathy was also considered; however, CTCA demonstrated no obstructive coronary artery disease and only minor coronary artery calcification.

The finding of a severely reduced ejection fraction on transthoracic echocardiography supported a new diagnosis of heart failure with reduced ejection fraction.

We proceeded with CMRI which suggested generalised severe myocarditis. Given that the patient had a new onset of heart failure, reduced ejection fraction and MRI findings consistent with




**Video 1** Apical 4 chamber view on transthoracic echocardiography demonstrating globally reduced left ventricular function and mitral regurgitation.

myocarditis, we felt that a diagnosis of myocarditis was most likely in this case.

Given the COVID-19 pandemic and accumulating evidence linking SARS-CoV-2 to myocarditis, SARS-CoV-2 PCR was performed and was positive. We therefore diagnosed our patient with COVID-19-associated myocarditis.

EMB was not performed in this case due to cardiac catheterisation laboratory restrictions in place due to the COVID-19 pandemic. At our centre, we do not routinely perform EMB in all cases of myocarditis. Given we do not have biopsy evidence, it may be suggested that the SARS-CoV-2 positivity may have been incidental. While this is certainly possible, we felt this was less likely in this case.

## TREATMENT

The patient was commenced on intravenous diuretics and rate-control agents. He was anticoagulated and was commenced on an ACE inhibitor and mineralocorticoid antagonist.

## OUTCOME AND FOLLOW-UP

The patient remained afebrile throughout admission and was discharged for outpatient follow-up. We intend to perform repeat CMRI in 3 months, and he will be followed up at our outpatient clinic for optimisation of his heart failure medication regimen.

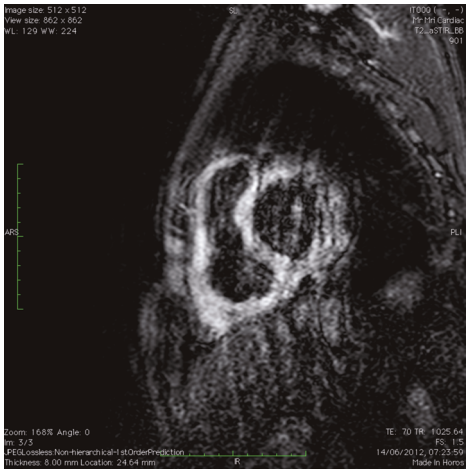
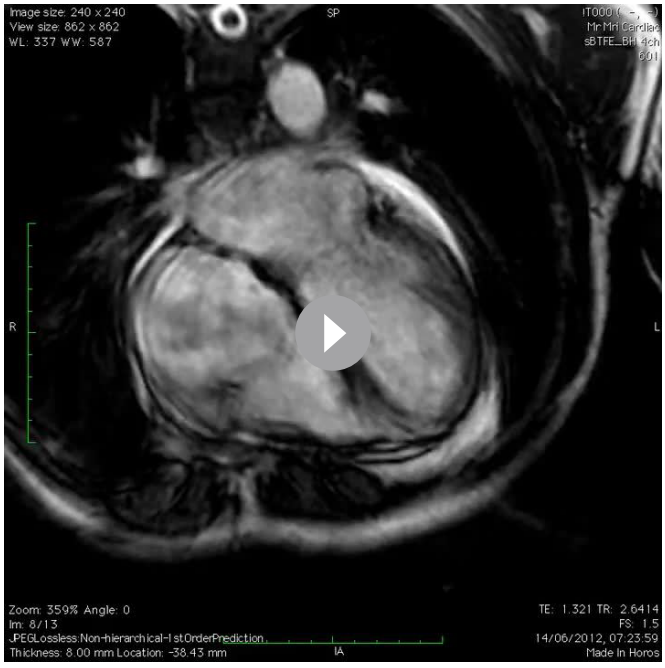



**Figure 1** Cardiac MRI T2 STIR image demonstrating diffuse oedema of both the right and left ventricles. The signal intensity ratio of the myocardium to skeletal muscle was 2.8, consistent with myocarditis.




**Video 2** Title of image: Cardiac MRI 4 Chamber Cine. Caption: 4 Chamber Cine demonstrating biventricular systolic dysfunction with global hypokinesia and a left ventricular ejection fraction of 30%

## DISCUSSION

COVID-19 presenting with acute decompensated heart failure and evidence of myocarditis on CMRI in the absence of prominent respiratory symptoms has been previously described in case reports.[2] Patients may present with cardiac involvement without manifesting the respiratory or infective symptoms that are classically associated with the disease. This case details a patient presenting with AF and acute decompensated heart failure who was subsequently found to have CMRI appearances consistent with myocarditis and a positive SARS-CoV-2 PCR. AF and atrial flutter as presenting features of COVID-19 have been previously described in case reports, but these cases were not associated with evidence of myocarditis or left ventricular dysfunction.[2 3] Several mechanisms have been proposed by which COVID-19 could result in arrhythmia. In the acute setting, direct viral injury to cardiac myocytes may result in disruption of the plasma membrane and aberrant conduction. Similarly, pericardial infection causing oedema or ischaemia from microvascular disease and endothelialitis may also result in arrhythmia. Activation of the sympathetic nervous system and hypoxaemia in patients with COVID-19 may also result in a proarrhythmic state. In a more chronic or healed myocarditis re-entrant arrhythmias due to myocardial scarring and proinflammatory cytokines such as interleukin 6 may also predispose to developing AF.[4]

A recent meta-summary of CMRI-proven or EMB-proven coronavirus-induced myocarditis cases also describes several similar presentations.[5] The Lake Louise CMRI criteria are used to establish the diagnosis of myocarditis in these cases.

EMB is the gold standard for diagnosis of myocarditis; however, it is an invasive procedure and often not necessary to reach a presumptive diagnosis. Nonetheless the SARS-CoV-2

genome has been detected within the EMB samples of patients with suspected myocarditis.[6]

## Learning points

- Myocarditis presenting as new-onset heart failure, reduced left ventricular ejection fraction and atrial fibrillation is a possible complication of SARS-CoV-2 infection.
- COVID-19-associated myocarditis should be considered in patients presenting with atrial fibrillation and heart failure even in the absence of respiratory symptoms.
- Cardiac MRI can be used to demonstrate features of myocarditis and may allow physicians to reach a presumptive diagnosis without the gold standard endomyocardial biopsy.

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**Contributors** All the listed authors have contributed in a substantial way to the creation of this manuscript. SG contributed to writing, editing and formatting of this project, and more specifically helped to edit the summary, background and case presentation sections to ensure that they fit the BMJ required format. JJC sourced the appropriate and interesting images for the manuscript and provided captions for the images and videos included in the article and was heavily involved in editing the manuscript. PD researched the topic and other similar cases in the literature and was instrumental to the discussion section, which he subsequently helped to edit in our drafts. IP also helped research the topic and contributed to designing and editing the submission. All authors contributed to the acquisition, analysis and interpretation of data. Furthermore all have been involved in drafting the work and revising it. All authors have agreed to be accountable for all aspects of the work related to accuracy or integrity of the manuscript.

**Funding** The authors have not declared a specific grant for this research from any funding agency in the public, commercial or not-for-profit sectors.

**Competing interests** None declared.

**Patient consent for publication** Obtained.

**Provenance and peer review** Not commissioned; externally peer reviewed.



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