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Imaging Findings of COVID-19–Related Cardiovascular Complications

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INTRODUCTION

At the end of 2019, the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) was identified as a novel cause of respiratory infection in Wuhan, which spread rapidly resulting in the global pandemic of coronavirus disease (COVID-19).1 It is widely recognized that patients with cardiovascular (CV) risk factors, or established CV disease (CVD), are at increased risk of developing severe COVID-19, and those with myocardial injury have a worse prognosis.2–5 Studies have shown that COVID-19 can cause a broad spectrum of CV complications including myocarditis,6 myocardial infarction (MI),7 stress cardiomyopathy,8 heart failure (HF),9 arrhythmias,10 thromboembolic events,11 and cardiogenic shock.12 Advanced cardiac imaging is a reliable diagnostic tool for prompt diagnosis, risk stratification, monitoring, and management of patients with COVID-19–related CV manifestations. This review is focused on the role of multimodality imaging in identifying cardiac involvement in COVID-19 and the potential CV side effects of the available treatment and vaccines against SARS-CoV-2.

PATHOGENESIS OF COVID-19–ASSOCIATED CV COMPLICATIONS

Although the pathophysiological mechanisms of the myocardial injury caused by SARS-CoV-2 have not been entirely elucidated, multiple factors have been hypothesized to contribute directly or indirectly to the development of CV complications.

a. Direct virus-mediated cytotoxicity. Although this theory is supported by an autopsy series,
which confirmed the detection of viral genome detection within the myocardium,\textsuperscript{13,14} in clinical practice, the histologic evidence of myocardial injury in COVID-19 is limited. Low loads of viral genome were detected in histology specimens in 5 cases out of 104 patients undergoing endomyocardial biopsy (EMB) for myocarditis or unexplained HF during the COVID-19 pandemic.\textsuperscript{15} However, there was no evidence of myocardial injury detected in the autopsy of a patient with COVID-19 and acute respiratory distress syndrome, who died of sudden cardiac death, challenging the theory of a direct cardiotoxic effect of SARS-CoV-2.\textsuperscript{16}

e. Hypoxic injury. Hypoxia caused by SARS-CoV-2 infection induces intracellular acidosis and the release of ROS from mitochondria in cardiomyocytes, which destroys the cell membrane contributing to cardiomyocyte apoptosis.\textsuperscript{4,26}

f. Cardiovascular side effects of drugs and vaccines. It is widely known that antiretroviral therapy and other drugs used in the management of COVID-19 patients (azithromycin, tocilizumab, chloroquine, and hydroxychloroquine) can induce arrhythmias, or interact with some CV treatments.\textsuperscript{27} In addition, cases of thromboembolic events have been reported after ChAdOx1 nCoV-19/AIDS1222 (AstraZeneca COVID-19 vaccine) and Ad26.COV2.S (Janssen COVID-19 vaccine)\textsuperscript{28,29} vaccinations. More recently, there is a potential association of the mRNA vaccines, BNTb162b (Pfizer) and mRNA-1273 (Moderna) with myocarditis.\textsuperscript{30} It is currently believed that the thrombotic events have been associated with autoantibodies directed against the platelet factor 4 (PF4) antigen.\textsuperscript{31}

The underlying pathophysiological mechanisms causing COVID-19–related CV complications are presented in Fig. 1.
than the direct invasion and damage by SARS-CoV-2.

**Transthoracic Echocardiography**

In a study of 305 hospitalized patients with COVID-19, myocardial injury (defined by elevated troponin levels) was observed in 190 patients. Echocardiographic abnormalities, including global or regional left ventricular (LV) wall motion abnormalities, LV diastolic dysfunction, right ventricular (RV) dysfunction, and pericardial effusions were noted in almost two-thirds of patients with myocardial injury. In the same study, the detection of structural abnormalities by transthoracic echocardiography (TTE) was an independent predictive factor of mortality in the subgroup with myocardial injury. An international prospective study of 1216 patients from 69 countries with clinical indication for TTE revealed the spectrum of echocardiographic abnormalities. Abnormal TTE was found in 55%, including LV and RV abnormalities at 39% and 33% respectively, whereas the prevalence of echocardiographic abnormalities was slightly lower (46%) in the subgroup with no pre-existing CVD. The LV abnormalities were predominantly nonspecific in nature and the mechanism of dysfunction was often not identified unless the TTE findings were suggestive of acute MI (3%), myocarditis (3%), and stress cardiomyopathy (2%). In the same study, severe LV, RV, or biventricular systolic dysfunction was observed in 14% and cardiac tamponade occurred in 1%. In contrast, another study showed that RV dilatation and systolic dysfunction were more common (39%) compared with LV systolic dysfunction (10%) in 100 hospitalized patients with COVID-19. The most common echocardiographic findings among those with subsequent clinical deterioration were worsening RV and LV function (12 and 5 patients respectively).

Myocardial infarction, myocarditis, or acute pulmonary hypertension might be the cause of isolated RV dysfunction in COVID-19 patients. Acute cor pulmonale precipitated by acute pulmonary embolism (PE) or adult respiratory distress syndrome has also been reported. In addition, the implementation of positive end-expiratory pressure in the respiratory support of critically ill COVID-19 patients can contribute to RV dysfunction. Interestingly, RV dysfunction has been shown to correlate with poor prognosis in COVID-19.

Cases of pericardial effusion in the context of myocarditis detected on TTE, either in association with myocarditis or as an isolated presentation, along with rare cases of pericardial tamponade in patients with COVID-19 have also been reported. Although there are no studies currently investigating the prevalence of pericardial involvement in COVID-19, the diagnosis of pericarditis should be considered in COVID-19 patients presenting with chest pain, ST elevation in the electrocardiogram (ECG), and normal coronary arteries.

Speckle tracking echocardiography is a novel echocardiographic technique. It allows quantification of myocardial deformation, providing early detection of cardiac dysfunction and plays an important prognostic role in HF and patients with end-stage renal disease. In addition, impaired LV and RV longitudinal strain are suggestive of early ventricular dysfunction with prognostic value in COVID-19 patients. A specific pattern of prominent longitudinal strain reduction of the basal LV segments has been described in COVID-19 patients suggesting that these areas might be vulnerable to myocardial injury during SARS-CoV-2 infection.

**Cardiovascular Magnetic Resonance**

Cardiovascular magnetic resonance (CMR) imaging is increasingly recognized as an essential tool in the assessment of COVID-19–induced myocardial injury because of the unique capability of CMR for noninvasive tissue characterization. The specific CMR techniques used for the characterization of tissue damage in clinical practice. Guidelines have been published for safe CMR
scanning during the COVID-19 pandemic and the Society for Cardiovascular Magnetic Resonance recommends a specific protocol to perform in patients with suspected COVID-19 cardiac involvement. The primary advantage of CMR is the ability to differentiate between ischemic and nonischemic pathologies. Although EMB remains the gold standard for the diagnosis of acute myocarditis, its use in routine clinical practice is limited because of low diagnostic accuracy and periprocedural risks. The Lake Louise Criteria (LLC) were initially established in 2009 using specific CMR techniques, including T2-weighted sequences, early and late gadolinium enhancement sequences (EGE and LGE, respectively), for the diagnosis of acute myocarditis; expanding them to the use of parametric mapping in the updated version. Therefore, CMR represents a robust noninvasive technique for the diagnosis of COVID-19-related myocarditis. CMR typically shows diffuse myocardial edema, noninfarct patterns of LGE, and increased signal on T2-weighted imaging, T1- and T2-mapping sequences (Fig. 3). CMR can also confirm the involvement of pericardium revealing edematous and enhancing pericardial layers suggestive of pericardial inflammation. Moreover, it has incremental value in differentiating COVID-19-associated stress (Takotsubo) cardiomyopathy and MI in patients with SARS-CoV-2 infection presenting with chest pain, high troponin, and ECG abnormalities. The absence of LGE in dysfunctional LV segments with the evidence of edema allows the differentiation from MI when transmural or subendocardial LGE with a coronary distribution is detected (Fig. 4). There is also an increased incidence of stress cardiomyopathy in patients without COVID-19 during the pandemic compared with the pre-pandemic era indicating the intense emotional stress.

CMR abnormalities have been reported in patients who have recently recovered from COVID-19, though most of them have been nonspecific and it is unclear whether they were pre-existing findings and thus unrelated to COVID-19. A study of 100 patients investigated the outcomes of CMR in patients recently recovered from COVID-19, including 18 patients with asymptomatic SARS-CoV-2 infection, 49 patients with mild to moderate symptoms, and 33 patients with severe symptoms requiring hospitalization. Comparisons were made with healthy controls as well as with...
CMR was performed 2 to 3 months after the initial positive COVID-19 test. Significant high-sensitivity troponin I (hs-cTnI) elevation at the time of CMR was noted in 5% of the patients. Notably, high myocardial native T1 (73%), elevated myocardial native T2 (60%), myocardial LGE (32%), and pericardial LGE (22%) were detected in patients who recovered from COVID-19, whereas CMR abnormalities found in the risk-factor matched controls were less frequent including high native T1 and T2 (40% and 9%, respectively), myocardial LGE (17%), and pericardial LGE (15%). There was also mild LV and RV systolic dysfunction compared with healthy and risk-factor matched controls (LVEF 56% vs 60% and 61%, respectively; RVEF 56% vs 60% and 59% respectively). Similarly, a smaller study of 26 patients who remained symptomatic after recovery from COVID-19 found myocardial edema in 54%, LGE

Fig. 3. CMR findings in a 21-year-old COVID-19 patient with acute myopericarditis who presented with chest pain, high troponin levels (1143 ng/L, normal range <14 ng/L), and global mild left ventricular systolic dysfunction on the bedside transthoracic echocardiography (TTE). (A, B) Steady-state free precession (SSFP) MRI cine 4 chamber (4ch) and short axis (SA), the arrows show the small pericardial effusion. (C) T2-weighted imaging showing slightly increased signal in the basal anterior wall (arrow) extending to basal anterolateral wall suggestive of myocardial edema. (D, E) Extensive patchy subepicardial and midwall late gadolinium enhancement (LGE) in the basal inferior, midinferoseptal, anterolateral, and anterior walls (arrows).

Fig. 4. Coronary angiogram and CMR in a 69-year-old man who presented with anterior ST-elevation myocardial infarction (STEMI) in the context of COVID-19. CMR performed 25 days after the acute coronary syndrome. (A) Coronary angiogram showing proximal occlusion (arrow) of left anterior descending (LAD) artery with high burden of thrombus. (B) CMR axial Half-Fourier Acquisition Single-shot Turbo Spin Echo (HASTE) showing patchy bilateral lung changes of high signal in keeping with COVID-19. (C, D) Steady-state free precession (SSFP) MRI cine in end systole showing thinning and akinesia of mid to apical septal walls extending to apical lateral walls and apical cap. (E, F) Late gadolinium enhancement (LGE) imaging showing transmural LGE in the mid to apical septal walls extending to the apical lateral walls and apical cap (blue arrows). In addition, there is partial extension of enhancement into the basal septal segments with evidence of microvascular obstruction in the basal anterosep- tum (yellow arrow).
31%, and decreased RV functional parameters in the subgroup with positive conventional CMR findings.64 A larger study included 148 patients with severe COVID-19 [all requiring hospital admission, 48 of whom (32%) requiring ventilatory support] and troponin elevation who underwent CMR at a median of 68 days after discharge.65 LV function was normal in 89%, myocarditis-like scar noted in 26%, infarction and/or ischemia in 22%, and dual pathology in 6%. However, whether these abnormalities represent de novo COVID-19–associated changes or pre-existed in the context of clinically silent disease remains unclear. Importantly, the extent of these abnormalities was quite limited and with minimal functional consequences.

CMR findings have also been reported among athletes who have recovered from COVID-19.66–68 In a study of 145 university student-athletes who were asymptomatic or experienced mild to moderate symptoms, CMR was performed at a median of 15 days after the positive COVID-19 tests.67 CMR findings consistent with updated LLC for myocarditis were found in only 2 patients (1.4%), whereas 40 patients (27.6%) had small nonspecific foci of LGE. A smaller study of 26 competitive collegiate athletes who underwent CMR examination 11 to 53 days after diagnosis of COVID-19 showed that 4 athletes (15%) met the updated LLC for clinically suspected myocarditis and 8 athletes (30%) had nonspecific LGE.66 In the same study, none of the athletes had elevated serum troponin I levels or diagnostic ST/T wave changes on ECG and biventricular size and function were both normal on TTE and CMR.

CMR abnormalities detected in convalescent COVID-19 raised concerns for long-term CV complications even in asymptomatic patients. However, the clinical significance of these findings remains uncertain and further studies are needed to confirm the clinical role of imaging in a long-term setting. The potential role of CMR in screening athletes is debated in the literature.68 The COVID-HEART trial is an ongoing multicentre UK trial, which will clarify the association of COVID-19–related cardiac involvement with comorbidity, genetics, patient-reported quality of life measures, and functional capacity.70

**Table 1** summarizes the studies of TTE and CMR findings in patients with COVID-19.

**Computed Tomography**

For patients with active COVID-19 and non-ST-elevation MI, noninvasive diagnostic testing before catheterization was recommended during the pandemic.71 In hemodynamically stable cases with low or intermediate CV risk, computed tomography coronary angiography (CTCa) could be considered as an alternative to invasive coronary angiogram.72 CTCa also provides information on lung parenchyma and pulmonary vessels, while excluding significant coronary artery disease with almost 100% negative predictive value.73

A retrospective study of 100 COVID-19 patients who underwent computed tomography pulmonary angiography (CTPA) showed that 23% had acute pulmonary embolism (PE).74 Those with PE had more severe disease requiring more intensive care admissions and mechanical ventilation (PE vs non-PE: 74% vs 22%, and 65% vs 25%, respectively, P = .001 for both comparisons).74 These findings were confirmed by several studies22,40,75 highlighting the important diagnostic and prognostic role of CTPA in COVID-19 patients. Moreover, the detection of subepicardial hyperattenuation in contrast-enhanced computed tomography combined with cardiac gating can be useful in the evaluation of COVID-19 patients with suspected myocarditis.76

**CARDIOVASCULAR EFFECTS OF DRUGS AND VACCINES AGAINST SARS-CoV-2: IS THERE A ROLE FOR CARDIAC IMAGING?**

Chloroquine and hydroxychloroquine are well-established treatments in non-CV conditions and have now been used in the treatment of COVID-19.77,78 Although there are inconsistent data regarding their efficacy, and large, randomized controlled trials are needed to establish their use in clinical practice, they are widely known to cause arrhythmias.79,80 Azithromycin used in combination with hydroxychloroquine is known to prolong QT interval.81 A retrospective cohort study of 1438 patients hospitalized with COVID-19 showed that cardiac arrest was more likely in patients receiving both hydroxychloroquine and azithromycin than in patients receiving neither drug.82 In the clinical scenarios of arrhythmia, CMR plays an important role in risk stratification identifying possible pathologic substrates (myocardial edema, ischemia, fibrosis) in patients with new-onset arrhythmias.

Cases of thromboembolic events associated with thrombocytopenia have been reported after vaccination with both ChadOx1 nCoV-19/ AZD1222 (AstraZeneca COVID-19 vaccine) and Ad26.COV2.S (Janssen COVID-19 vaccine).28,29 Cases of myocarditis and pericarditis have also been reported after mRNA vaccines, BNTb162b (Pfizer) and mRNA-1273 (Moderna),30,83–85 but a similar pattern of these complications have not been reported after Ad26.COV2.S (Janssen
| Authors                  | Study Design                                      | Imaging Modality | Population                                                                 | Results                                                                 |
|-------------------------|--------------------------------------------------|------------------|-----------------------------------------------------------------------------|-------------------------------------------------------------------------|
| Giustino et al, 2020    | International, multicenter retrospective study    | TTE              | N = 305 hospitalized patients with COVID-19                                 | Myocardial injury was observed in 190 patients (62.3%)                  |
|                         | Cardiac Injury Research in COVID-19 Registry      |                  | Age, range (y): 63 (53–73)                                                 | TTE abnormal in 2/3rds of patients with myocardial injury:              |
|                         | (CRIC-19)                                         |                  | Male/Female, n: 205/305                                                     | • LV wall motion abnormalities (N = 45, 23.7%)                           |
|                         |                                                   |                  |                                                                             | • LV global dysfunction (N = 35, 18.4%)                                 |
|                         |                                                   |                  |                                                                             | • RV dysfunction (N = 50, 26.3%)                                        |
|                         |                                                   |                  |                                                                             | • Pericardial effusion (N = 22, 7.2%)                                   |
|                         |                                                   |                  |                                                                             | • Diastolic dysfunction grade II or III (N = 25, 13.2%)                  |
|                         |                                                   |                  |                                                                             | In-hospital mortality:                                                  |
|                         |                                                   |                  |                                                                             | • 5.2% in patients without myocardial injury                             |
|                         |                                                   |                  |                                                                             | • 18.6% in patients with myocardial injury and without TTE abnormalities |
|                         |                                                   |                  |                                                                             | • 31.7% in patients with myocardial injury and TTE abnormalities         |
| Dweck et al, 2020       | Prospective international survey                  | TTE              | N = 1216 hospitalized patients with COVID-19, 69 countries                  | 667 (55%) patients had abnormal TTE:                                    |
|                         | (www.escardio.org/eacvi/surveys)                  |                  | Age, range (y): 62 (52–71)                                                 | • 39% LV abnormalities                                                   |
|                         |                                                   |                  | Male/Female, n: 844/365                                                     | (predominantly not specific)                                            |
|                         |                                                   |                  |                                                                             | • 33% RV abnormalities                                                   |
|                         |                                                   |                  |                                                                             | (more common in patients with more severe COVID-19)                     |

(continued on next page)
| Authors | Study Design | Imaging Modality | Population | Results |
|---------|--------------|------------------|------------|---------|
| Szekely et al, 2020 | Prospective observational single-center study | TTE | N = 100 hospitalized patients with COVID-19 Age, mean ± SD (y): 66.1 ± 17.3 Male/Female, n: 63/37 | 68 (68%) patients had abnormal TTE: • 39% RV dilatation ± dysfunction • 16% LV diastolic dysfunction • 10% LV systolic dysfunction • 3% valvular heart disease 60% among deteriorating patients had RV dilatation and dysfunction (+/-DVT) |
| Kim et al, 2020 | Prospective Multicenter Registry | TTE | N = 510 hospitalized patients with COVID-19 Age, mean ± SD (y): 64 ± 14 Male/Female, n: 335/175 | • 35% RV dilatation • 15% RV dysfunction • RV dysfunction increased step-wise with RV dilatation • Adverse RV remodeling predicted mortality independent of clinical and biomarker risk stratification (HR 2.73; 95% CI, 1.72–4.35; P < .001) |
| Li et al, 2020 | Prospective observational single-center study | TTE | N = 120 hospitalized patients with COVID-19 | RVLS was a powerful predictor of higher mortality in patients |
| Study                  | Type                        | Methodology | Participants | Findings                                                                                                                                                                                                                                                                                                                                 |
|-----------------------|-----------------------------|-------------|--------------|------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Goerlich et al, 2020  | Retrospective observational single-center study | TTE         | N = 75 hospitalized patients with COVID-19 Cases (n = 39): basal LS <13.9% (absolute value) Controls (n = 36): basal LS >13.9% (absolute value) Age, mean ± SD (y): 61.9 ± 13.5 Male/Female, n: 44/31 | 52% had a reduced basal strain on STE (basal LS 10.0 ± 2.9% vs 16.9 ± 2.3%, P < .001) GLS was significantly lower in COVID-19 cases vs controls (13.9 ± 4.1% vs 18.8 ± 2.7%, P < .001) LVEF (%) was similar between groups (62.5 [55.0–64.4] vs 57.5 [47.5–62.5], P = .11) |
| Puntmann et al, 2020  | Prospective observational single-center study | CMR         | N = 100 patients recovered from COVID-19, CMR 71 (64–92) days from positive test Age, mean ± SD (y): 49 ± 14 Male/Female: 53/47 N = 50 age and sex matched healthy controls N = 57 risk factor matched controls | Patients recovered from COVID-19 had lower LVEF and RVEF, higher LVEDVi, and raised native T1 and T2 values compared with both control groups. Greater proportions of patients with ischemic (32% vs 17%) and nonischemic (20% vs 7%) LGE patterns than the risk factor matched control group. There was a greater proportion of cases with pericardial enhancement (22% vs 14%) and pericardial effusion (20% vs 7%) compared with the risk factor matched control group. |
| Huang et al, 2020     | Retrospective observational single-center study | CMR         | N = 26 patients recovered from moderate-severe COVID-19 Age, range (y): 38 (32–45) Male/Female: 10/16 | 58% had abnormal CMR: • Myocardial edema in 14 patients (54%) • LGE in 8 patients (31%) (continued on next page) |
| Authors                        | Study Design                                      | Imaging Modality | Population                                                                 | Results                                                                                                                                                                                                 |
|-------------------------------|---------------------------------------------------|------------------|----------------------------------------------------------------------------|-------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Kotecha et al, 2021           | Prospective observational multicentre study       | CMR              | N = 148 recovered COVID-19 patients (moderate-severe COVID-19)              | • LV function was normal in 89%  
• Myocarditis-like scar noted in 26%  
• Infarction and/or ischemia in 22%  
• Dual pathology in 6%  
No difference in LVEDVi, LVESVi, LVEF between recovered COVID-19 patients and risk factor matched controls. No difference in native T1, T2 values between recovered COVID-19 patients and risk factor matched controls. Higher proportion of subepicardial LGE noted in recovered COVID-19 patients compared with risk factor matched controls (22% vs 5%, \( P = .018 \)). |
|                               |                                                   |                  | Age mean ± SD (y): 64 ± 12                                                 | N = 40 risk factor matched controls  
N = 40 healthy volunteers |
|                               |                                                   |                  | N = 20 age and sex matched healthy controls                               | N = 20 age and sex matched healthy controls  
Global native T1, T2, and ECV values were significantly elevated in recovered COVID-19 patients with positive conventional CMR findings, compared with patients without positive findings and healthy controls  
Decreased RV function parameters (RVEF, RVCO, RVCI, and RVSV) were found in patients with positive conventional CMR findings, compared with healthy controls (\( P < .05 \)). |
| Study                        | Study Design                     | Imaging Methods | Study Group | Details |
|------------------------------|----------------------------------|----------------|-------------|---------|
| Rajpal et al, 2021           | Case Series (single centre)      | TTE, CMR       | N = 26 competitive college athletes recovered from COVID-19 (14 asymptomatic, 12 mild symptoms) | Normal biventricular size and function by TTE and CMR None had troponin elevation or diagnostic ST/T wave changes on ECG 4 athletes (15%) met the updated LLC for clinically suspected myocarditis 8 athletes (30%) had nonspecific LGE |
| Starekova et al, 2021        | Case Series (single centre)      | TTE, CMR       | N = 145 competitive college athletes recovered from COVID-19 (17% asymptomatic, 49% mild, 28% moderate symptoms) | TTE was unremarkable 2 athletes (1.4%) had myocarditis by LLC, troponin abnormal in the more severe case 40 patients (27.6%) had small nonspecific foci of LGE |
| Gorecka et al, 2021          | Prospective observational multicentre study (COVID-HEART study) | CMR            | Inclusion criteria: hospitalized patient population (age ≥ 18 y), or those recently discharged from hospital (within 28 d after discharge), with a diagnosis of COVID-19 Exclusion criteria: unable or unwilling to consent, contraindication to CMR, pregnancy or breast-feeding Risk factor matched controls: matched on age and CVD risk factors cohort | Ongoing trial |

AUC, area under the receiver operating characteristic curve; CI, confidence interval; CMR, cardiac magnetic resonance; COVID-19, coronavirus disease 2019; DVT, deep vein thrombosis; ECG, electrocardiogram; ECV, extracellular volume fraction; GLS, global longitudinal strain; HR, hazard ratio; LGE, late gadolinium enhancement; LLC, Lake Louise criteria; LS, longitudinal strain; LV, left ventricular; LVEDVi, left ventricular end-diastolic volume index; LVEF, left ventricular ejection fraction; LVESVi, left ventricular end-systolic volume index; NPs, natriuretic peptides; OR, odds ratio; RV, right ventricular; RVCI, right ventricular cardiac index; RVCO, right ventricular cardiac output; RVEDVi, right ventricular end-diastolic volume index; RVEF, right ventricular ejection fraction; RVESVi, right ventricular end-systolic volume index; RVLS, right ventricular longitudinal strain; RVSV, right ventricular stroke volume; SD, standard deviation; STE, speckle tracking echocardiography; TTE, transthoracic echocardiogram.
COVID-19 vaccine). However, presentations were mild, none with evidence of acute SARS-CoV-2 infection, nor met criteria for MIS. The CMR findings were consistent with myocarditis in all cases highlighting the role of CMR in detecting pathology even in less aggressive diseases. **Fig. 5** shows the CMR findings of a patient admitted with acute myocarditis 1 week after mRNA-1273 vaccination. **SUMMARY**

Advanced cardiac imaging plays an essential role in the diagnosis, prognosis, and management of COVID-19 patients. It provides noninvasive tools for the recognition of the broad spectrum of CV complications in COVID-19, along with the potential CV side effects of the available vaccinations against SARS-CoV-2. However, the clinical significance of the imaging findings after COVID-19 recovery remains unclear and needs further study in large, randomized clinical trials.

**CLINICS CARE POINTS**

- Cardiovascular (CV) disease increases the risk of severe COVID-19 presentation.
- CV manifestations may occur during the acute phase of COVID-19, and cardiac involvement appears to be correlated with COVID-19 severity.
- Evidence suggests sustained CV involvement is uncommon. Even if patients have scar, structural and functional abnormalities, there is no difference to carefully matched control groups in the entire COVID-19 severity spectrum.

**DISCLOSURES**

E. Nakou, E. De Garate, K. Liang, and M. Williams have nothing to disclose. C. Bucciarelli-Ducci is the chief executive officer (CEO; part-time) of the Society for Cardiovascular Magnetic Resonance (SCMR); she received speakers fees from Circle Cardiovascular Imaging. D.J. Pennell receives research support from Siemens, and speakers fees from Circle, Bayer, and Chiesi.

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