Circulation: Cardiovascular Imaging

CARDIOVASCULAR IMAGES

Diffuse Myocardial Inflammation in COVID-19 Associated Myocarditis Detected by Multiparametric Cardiac Magnetic Resonance Imaging

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A 79-year-old male was hospitalized due to fatigue, shortness of breath, and recurrent syncopes. He denied symptoms of fever or pain. He had a previous history of asthma. No history of cardiovascular disease was reported, and previous cardiac check-ups were unremarkable (last described left ventricular ejection fraction was 65%).

Initial physical examination in the emergency department revealed heart rate of 75 bpm, blood pressure of 101/64 mm Hg, body temperature of 35.6°C, oxygen saturation of 94%, and moderate wheezing on auscultation. On initial blood test, CRP (C-reactive protein) was measured at 13.80 mg/L and high sensitive troponin T at 18.8 ng/L, but leucocyte blood count and NT-proBNP (N-terminal prohormone of brain natriuretic peptide) were normal. Electrocardiogram, chest x-ray, and echocardiography were normal. Patient was referred for contrast-enhanced computed tomography to rule out pneumonia or pulmonary embolism that revealed pulmonary ground-glass peripheral infiltrates in the left upper lobe and discrete pleural and pericardial effusions (Figure 1). Because of the outbreak of coronavirus disease 2019 (COVID-19), a nasopharyngeal swab was performed at admission, real-time reverse transcriptase-polymerase chain reaction assay returned positive for severe acute respiratory syndrome coronavirus 2. Due to respiratory and hemodynamic worsening, the patient was moved to the intensive care unit. Blood tests showed an increase in CRP (64.23 mg/L), leucocyte blood count (14.60 g/L), troponin T (63.5 ng/L), and NT-proBNP (1178.0 pg/mL). Cardiac magnetic resonance (CMR) was performed at 1.5 T at day 10 after admission (Figure 2). CMR analysis showed normal left ventricular size (left ventricular end-diastolic volume index: 68 mL/m^2; left ventricular mass index 42 g/m^2) and mild systolic dysfunction (left ventricular ejection fraction: 49%) with discrete global hypokinesis, and normal right ventricular volume and function (Movies I and II in the Data Supplement). Pericardial effusion was confirmed, localized mainly around the left ventricular lateral wall (≈10 mm). T2-weighted short TI inversion recovery sequences displayed diffuse interstitial myocardial edema with an increased T2 signal intensity ratio. Presence of diffuse myocardial inflammation was confirmed by T2 mapping (global T2 relaxation times: 62 ms; center-specific cutoff value for acute myocarditis: ≥55.9 ms; global myocardial T2 relaxation time represents a mean value of all 16 heart segments).[1] Late-gadolinium enhancement imaging (inversion time by using the Look-Locker technique: 240 ms) was negative for focal myocardial lesions, but prolonged T1 relaxation times could be measured (global T1 relaxation times: 1035 ms; center-specific cutoff value for acute myocarditis: ≥1000 ms; global myocardial T1 relaxation time represents a mean value of all 16 heart segments).[1] CMR parameters fulfilled the revised 2018 Lake Louise criteria for the diagnosis of myocarditis.[2] Medical treatment

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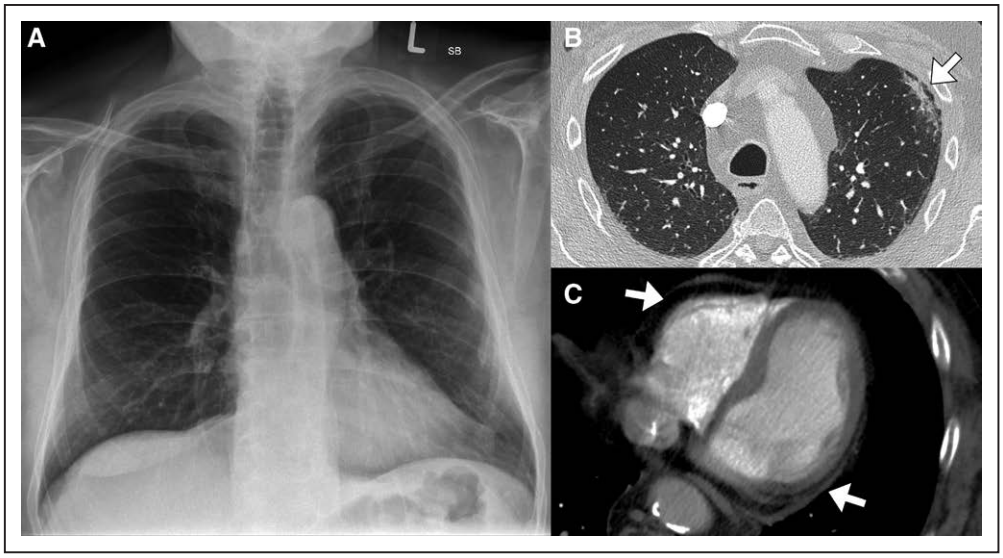

Figure 1. Conventional and computed tomography imaging findings.
A, No pulmonary abnormalities were seen on chest x-ray. Contrast-enhanced computed tomography revealed pulmonary infiltrates in the peripheral left upper lobe with an atypical pattern (white arrow; **B**) and mild pericardial effusion (white arrows; **C**).

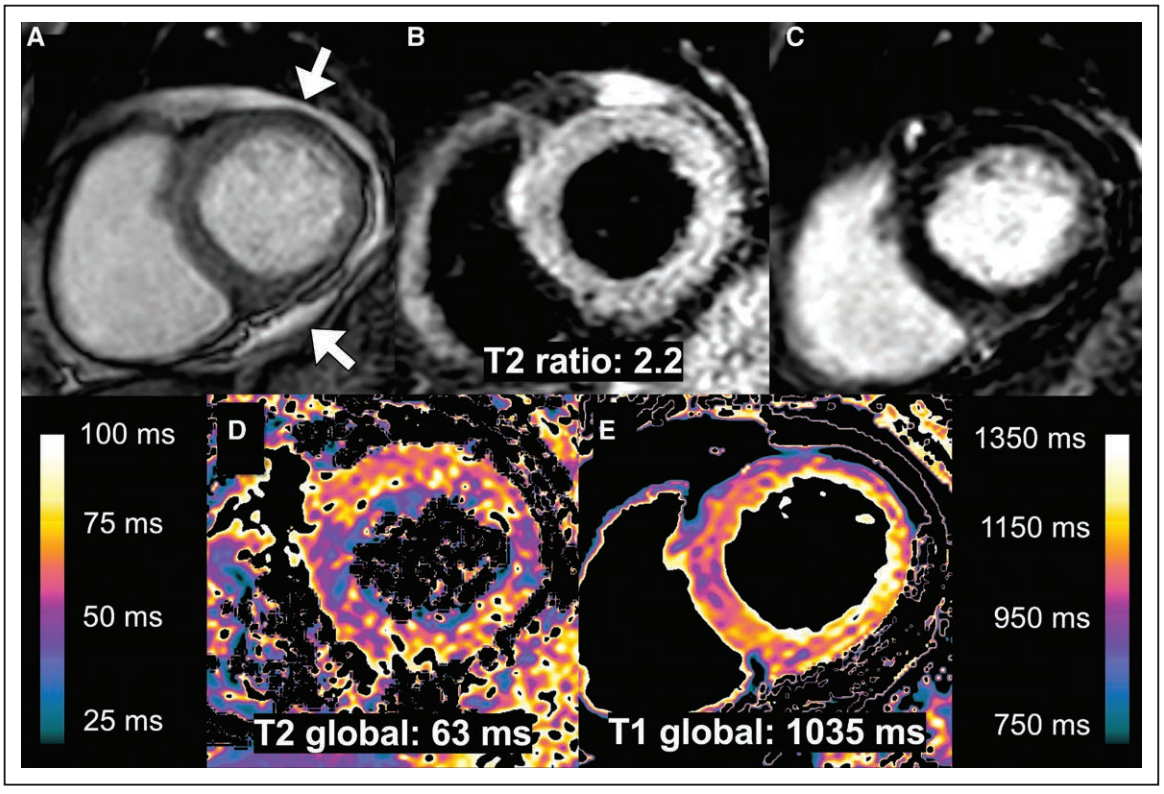

Figure 2. Cardiac magnetic resonance images of basal segments of the left ventricle in short axis view and end-diastole.
A, Cine images (balanced steady-state free precession) showed normal biventricular size and pericardial effusion (white arrows) around the left ventricle. **B**, Fat-suppressed images (T2-weighted short TI inversion recovery) revealed diffuse myocardial edema with an increased T2 ratio. **C**, No focal enhancement was identified on late-gadolinium enhancement imaging. **D** and **E**, Quantitative measurement of global T2 (**D**) and native T1 (**E**) relaxation times displayed significantly prolonged T1 and T2 values in the left ventricle.

of heart failure was initiated, and the patient recovered on follow-up.

First reported in December 2019 in Wuhan, China, infection with severe acute respiratory syndrome coronavirus 2 is spreading around the world, and COVID-19 has become a serious challenge for the international public health system. Although primarily affecting the respiratory system, cardiac involvement is often clinically observed. Patients with COVID-19 infection and associated myocardial injury (ie, elevated levels of troponin) may have a markedly increased mortality compared with patients with normal troponin levels.[3] As myocardial injury is significantly associated with fatal outcome of COVID-19, the underlying potential mechanism of this phenomenon might be myocardial inflammation.[3]

In our case, the patient showed elevated troponin levels and syncopes but presented with only relatively mild symptoms of pneumonia. Imaging studies could not reveal a spreading infection from the lung to the myocardium and also myocardial involvement was not an accompanying symptom from underlying septic shock. The clinical presentation of the patient and the signs of diffuse myocardial inflammation could be related to direct damage of cardiomyocytes by the virus or myocardial injury triggered by a cytokine storm and imbalanced response by type 1 and type 2 T-helper cells.[4]

Interestingly, no subepicardial late-gadolinium enhancement, which is typically seen in acute viral myocarditis, was visible in our case, and diagnosis of COVID-19 associated diffuse acute myocardial inflammation was evidenced by the use of T1 and T2 mapping sequences. Therefore, our case highlights the importance of CMR with the use of new quantitative mapping sequences as a powerful diagnostic tool for detecting diffuse myocardial injury, especially in the context of inflammatory, potentially cytokine-triggered, disease. Awareness of atypical presentations and imaging findings, as described in our case (no fever, progressive cardiac involvement, diffuse myocardial inflammation), can be essential for prompt isolation of infectious patients and correct CMR-based diagnosis of COVID-19 induced myocarditis. Clinicians should be aware of mild courses of the disease with primarily cardiac symptoms. As cardiac involvement might be associated with an unfavorable outcome, attention to potential cardiovascular complications before and during treatment of COVID-19 is vitally important, and aggressive treatment might be necessary in patients with previously underlying cardiovascular diseases.[4]

ARTICLE INFORMATION

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Disclosures

None.

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