IMAGE FOCUS

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Myocarditis detected after COVID-19 recovery

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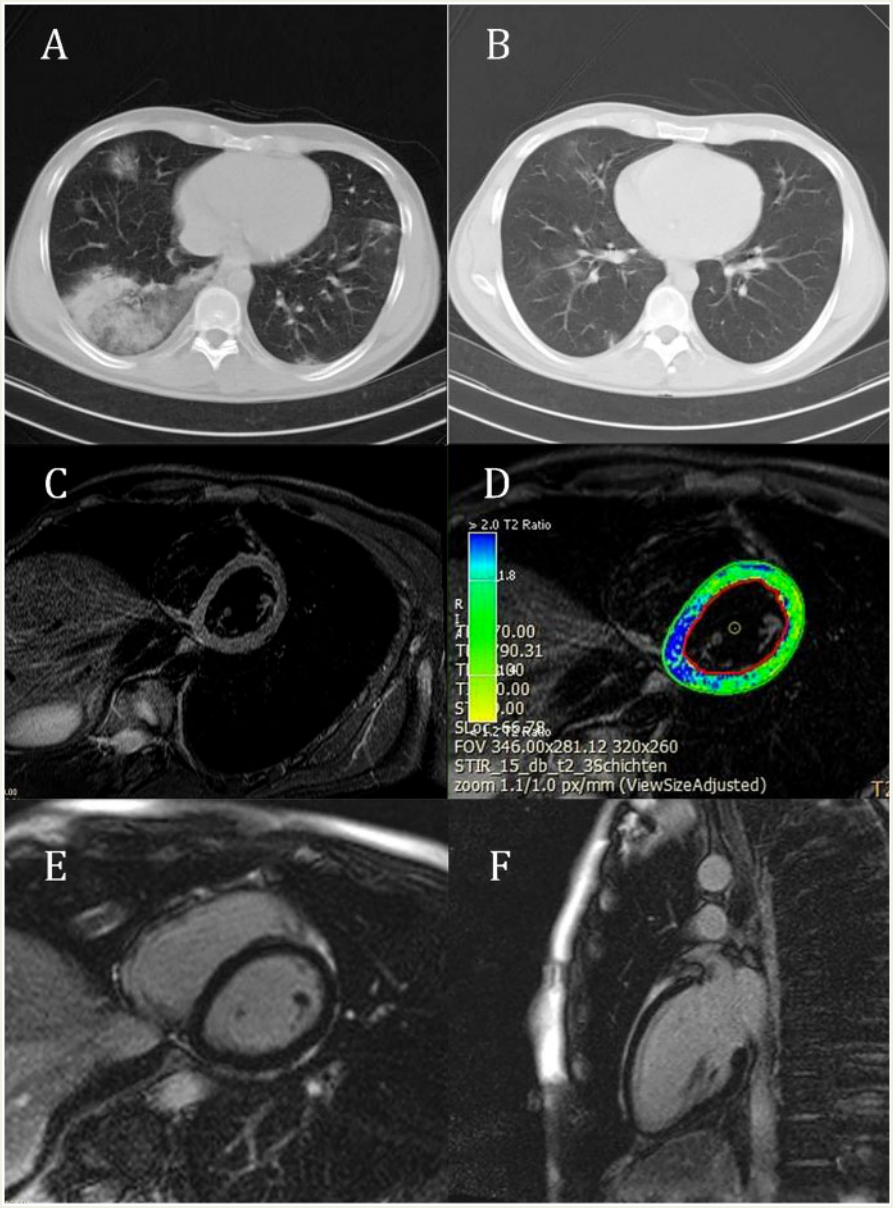
A 31-year-old internal medicine registrar presented with dyspnoea on exertion and low-grade fever. He had a history of COVID-19 pneumonia and was discharged 3 weeks previously. He did not have any other specific past medical history. At his previous admission, RT–PCR was positive for SARS-CoV-2, lung CT (*Panel A*) showed bilateral ground-glass and consolidative opacities mostly in the right lower zone, and the laboratory data showed high C-reactive protein (CRP) levels of 105 mg/L and erythrocyte sedimentation rate (ESR) of 70 mm/h but normal high sensitivity troponin T of <0.03 ng/mL. In the second week, he experienced pleuritic chest pain with no ECG changes. Troponin was not checked at that time. He was discharged after 10 days with two subsequent negative RT–PCRs and in good condition. CT at discharge (*Panel B*) showed significant resolution of consolidation with bilateral multifocal ground-glass appearance.

Three weeks after discharge, he presented with dyspnoea on exertion and low-grade fever. On examination, he had a blood pressure of 110/80 mmHg, respiratory rate 18 breaths/min, heart rate 70 b.p.m., temperature 37.8°C (axillary), and O_2 Sat 96%. Blood cell count (CBC) was normal, CRP was 3.3 mg/L, and high sensitivity troponin T was <0.03 ng/mL. RT–PCR was negative for SARS-CoV-2. A 12-lead electrocardiogram was normal. The transthoracic echocardiography had a poor acoustic window but



revealed mild left ventricular (LV) dysfunction. Cardiac magnetic resonance revealed normal LV size with mildly reduced ejection fraction (LVEF) of 50% (Supplementary material online, *Video S1*). T2-weighted oedema sequence with its post-analysis T2 ratio (*Panel C* and *D*) showed oedema/inflammation in the mid inferoseptal and inferior wall. Late gadolinium enhancement (*Panel E* and *F*) showed subepicardial fibrosis in the mid inferior wall. The clinical and imaging features were suggestive for active myocarditis. The patient was clinically stable with no arrhythmia or signs of heart failure, so we did not perform an endomyocardial biopsy. Other serological tests for Coxsackie, Parvovirus B19, Epstein–Barr, and human herpesvirus were negative. The myocarditis could be residual myocardial inflammation as a result of COVID-19, and the cardiac involvement might be overlooked during pneumonia. Medical treatment was started with bisoprolol and lisinopril.

Supplementary material is available at *European Heart Journal Cardiovascular Imaging* online.

Conflict of interest: none declared.