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Persistent chest pain after recovery of COVID-19: microvascular disease-related angina?

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A 42-year-old woman, presented with fever, anosmia, and a positive polymerase chain reaction for severe acute respiratory syndrome coronavirus 2.

One month later, she complained of oppressive chest pain increasing on exertion suggestive of angina. She did not have previous symptoms or history of coronary artery disease. A 12-lead electrocardiogram demonstrated sinus rhythm with asymmetric T-wave inversion in precordial anterior leads (Supplementary material online, *Figure S1*).

A coronary computed tomography showed angiographically normal coronary arteries (Supplementary material online, *Figure S2* and *S3*).

An adenosine-stress cardiac magnetic resonance (CMR) scan was performed (*Figure 1*) at a state of the art 1.5 T scanner (Philips Intera-dStream). Left and right indexed ventricular volumes and function were within normal ranges [indexed left ventricular end-diastolic volume: 79 mL/m^2 (60–93 mL/m^2); indexed right ventricular end-diastolic volume: 80 mL/m^2 (60–87 mL/m^2); left ventricular ejection

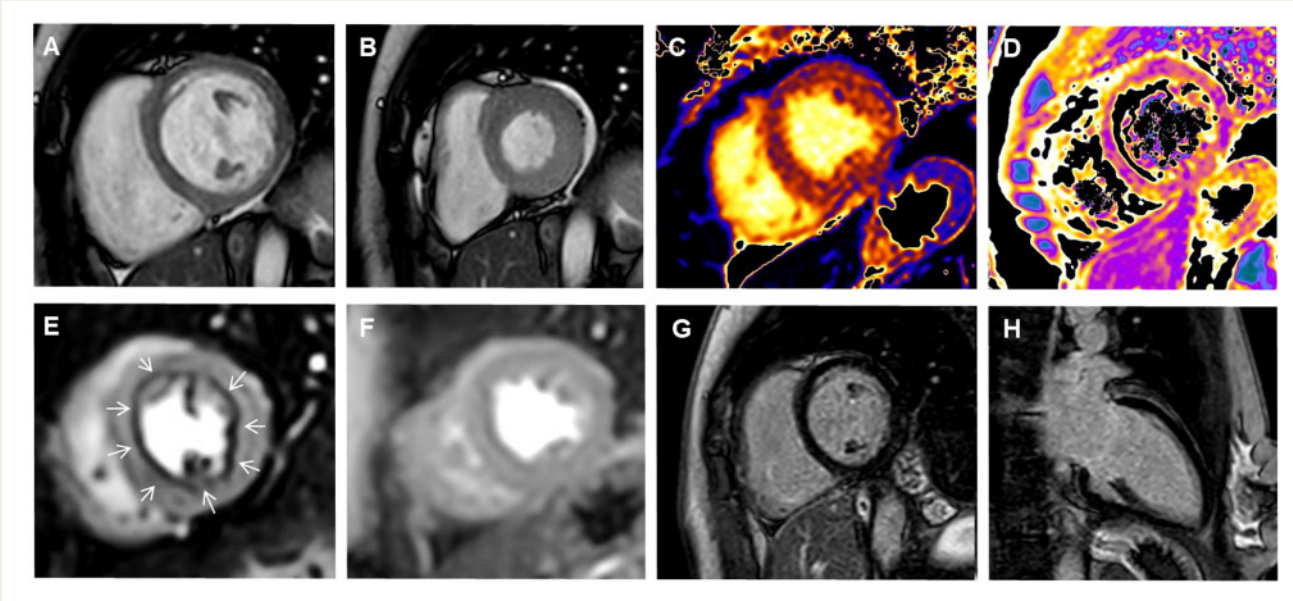

Figure 1 Mid short-axis cine slices in (*A*) diastole and (*B*) systole. Myocardial native T1 mapping (*C*) and T2 mapping (*D*) showed normal values. First-pass stress perfusion magnetic resonance imaging (*E*) with a significant circumferential subendocardial perfusion defect (arrows). Rest perfusion magnetic resonance imaging (*F*). No late gadolinium enhancement was detected (*G* and *H*).

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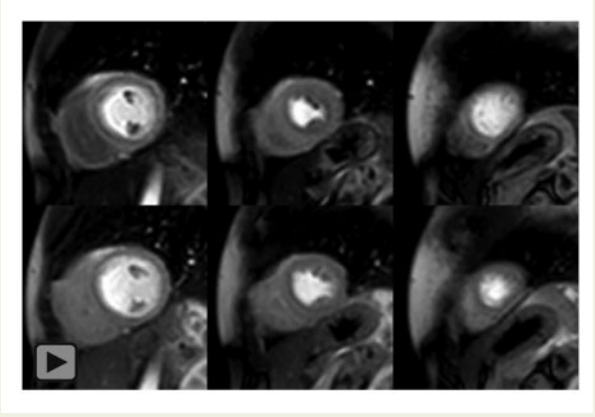

Video 1 Stress and rest perfusion cardiac magnetic resonance imaging.

fraction 69% (57–75%); right ventricular ejection fraction: 68% (53–76%)].[1] Native MOLLI T1 mapping was performed (at mid-ventricular level[2]). Septal native T1 mapping was normal (1040 ms) (normal: 930–1056 ms). Ventricular GraSE T2 mapping was 54 ms (normal: 46–58 ms) ruling out myocardial edema. A first-pass stress perfusion CMR showed a significant circumferential subendocardial perfusion defect which highly suggested microvascular dysfunction (*Figure 1E*, arrows), with normal perfusion at rest (*Figure 1F*) (*Video 1*). No late gadolinium enhancement was detected (*Figure 1G* and *H*). During the test, the patient presented the same angina she previously reported.

Treatment with aspirin, statins, ranolazine, and nitrates was initially prescribed. Nitrates were stopped because of bad clinical tolerance. Low doses of beta-blockers and calcium antagonist could be added. She reported partial improvement of symptoms, without major cardiovascular events during follow-up.

Some patients report persistent symptoms after recovering from acute coronavirus disease 2019 (COVID-19). Chest pain is present in ~20% of patients.[3] Mechanisms of microvascular disease in COVID-19 include endothelial injury with endothelial dysfunction and microvascular inflammation, and thrombosis.[4,5]

This case demonstrates that coronary microvascular ischaemia could be the mechanism of persistent chest pain in patients that have recovered from COVID-19. When obstructive coronary disease is excluded, functional non-invasive tests are recommended to rule out myocardial ischaemia. At present, the pattern of recovery, or the preferred management of post-COVD-19 microvascular dysfunction remains unresolved.

Supplementary material

Supplementary material is available at *European Heart Journal - Case Reports* online.

Consent: The authors confirm that written consent for submission and publication of this case report including images and associated text has been obtained from the patient in line with COPE guidelines.

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