

Stress Cardiac Magnetic Resonance (CMR)

Myocardial perfusion after COVID-19 infection: No persisting impaired myocardial blood flow in surviving patients

Thornton G.¹; Shetye A.¹; Knott K.²; Razvi Y.¹; Vimalasvaran K.³; Kurdi H.⁴; Artico J.⁴; Yousef S.⁴; Antonakaki D.⁴; Kellman P.⁵; Knight D.¹; Cole GD.³; Moon JC.¹; Fontana M.¹; Treibel TA.¹

¹University College London, London, United Kingdom of Great Britain & Northern Ireland

²King's College Hospital NHS Foundation Trust, London, United Kingdom of Great Britain & Northern Ireland

³Imperial College London, London, United Kingdom of Great Britain & Northern Ireland

⁴Barts Health NHS Trust, London, United Kingdom of Great Britain & Northern Ireland

⁵National Heart Lung and Blood Institute, Bethesda, United States of America

Funding Acknowledgements: Type of funding sources: None.

Background: Acute myocardial damage is common in hospitalized patients with severe COVID-19, with evidence of myocardial infarction and myocarditis demonstrated on cardiovascular magnetic resonance (CMR). Post-mortem studies have also implicated microvascular thrombosis, which may cause persistent microvascular disease.

Purpose: To determine the long-term coronary sequelae in recovered COVID-19 using multiparametric CMR including state-of-the-art inline quantitative stress myocardial blood flow (sMBF) mapping to assess global and regional sMBF.

Methods: Prospective, multicentre observational study of recovered COVID-19 patients scanned at three London CMR units. Results were compared to a propensity-matched, pre-COVID chest pain cohort (104 patients referred for perfusion CMR, with subsequently demonstrated unobstructed coronary arteries) and 27 healthy volunteers (HV). Perfusion image analysis was performed using a novel artificial intelligence approach deriving global and regional stress and rest MBF with a cut-off of >2.25 mL/g/min signifying normal sMBF and <1.82 mL/g/min abnormal sMBF (Kotecha JCVI 2019).

Results: 104 recovered, post-COVID patients (median age 62 years, 76% male; 89[87%] hospitalised, 41/89[46%] requiring ICU) underwent adenosine-stress perfusion CMR at a median 131(IQR 43-179) days from COVID-19 diagnosis. Median LVEF was 67% (IQR 60-71%; 12 (11.5%) with impaired LVEF), 51 patients (49%) had late gadolinium enhancement (LGE); 18% infarct-pattern and 33% non-ischaemic LGE. Global stress MBF in post-COVID patients was no different to age-, sex- and co-morbidities-matched controls (2.57 ± 0.77 vs. 2.40 ± 0.75 mL/g/min, $p = 0.11$, Figure 1), though lower than HV (3.00 ± 0.76 mL/g/min, $p = 0.001$). Post-COVID, multivariate predictors of low sMBF were male sex (OR 0.57, 95%CI 0.41-0.80, $p = 0.001$) and hypertension (OR 0.67, 95%CI 0.51-0.88, $p = 0.004$), but not COVID-19 disease severity (ICU admission) or presence of scar (ischemic/non-ischemic). 21/42 with reduced sMBF (<2.25 mL/g/min) had regional perfusion defects consistent with epicardial coronary disease.

Conclusions: COVID-19 survivors do not demonstrate evidence of reduced global MBF by CMR compared to risk factor matched controls. Stress perfusion CMR identifies etiology of acute myocardial damage (infarction/myocarditis) and presence of occult coronary ischemia.

Abstract Figure.

