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The Janus of COVID-19: from registry data to prospective studies

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This commentary refers to 'Patterns of myocardial injury in recovered troponin-positive COVID-19 patients assessed by cardiovascular magnetic resonance', by T. Kotecha *et al.*, doi:1093/eurheartj/ehab075 and the discussion piece 'The evolution of cardiovascular COVID-19 research', by T. Kotecha *et al.*, doi:10.1093/eurheartj/ehab240.

To the Editor,

We congratulate the authors on their important study to provide further insights on the associations between imaging findings and troponin elevation, particularly in the context of recent SARS-CoV-2 (COVID-19) infection, which is of immediate relevance.[1]

Around 1 in 5 patients admitted to hospital with COVID-19 have troponin elevation; these patients have between a two- and four-fold increase in the risk of death, compared with COVID-19 patients with normal troponin levels. Troponin elevation in patients with COVID-19 not due to type 1 myocardial infarction is associated with greater mortality than non-COVID-19 causes of type 2 myocardial infarction or injury.[2] Given the prohibitive mortality in this higher-risk group, the data reflect a survival bias for patients surviving to discharge. Therefore, as Kotecha *et al.*[1] point out, their study is focused to a small subgroup of patients with convalescent disease. However, cardiac magnetic resonance scanning in patients during acute illness, particularly with more severe disease, is frequently impracticable.

It is important to appreciate that whilst the study is of high interest, troponin testing was selectively performed in patients according to clinical need. The study was retrospectively conducted based on recall of discharged patients, coronary angiography was not systematically performed, and historical rather than contemporary controls were selected. We look forward to the findings from the multicentre, UK-wide COVID-HEART study which will prospectively clarify myocardial involvement in this troponin-positive subgroup of patients with COVID-19 up to 28 days after discharge, as well as the convalescent period with a subsequent cardiac MRI at 180 days in addition to quality of life and exercise tolerance assessments (ISRCTN58667920).[3]

We have concerns that the findings are open to misinterpretation such as reflected by the headline in medical media 'Myocardial Injury Seen on MRI in 54% of Recovered COVID' and we applaud Professor Fontana and colleagues for emphasizing their results are focused to the subgroup with biochemical criteria for myocardial injury.[4]

As discussed by Professor Fontana at interview, the prevalence of myocardial involvement remains unknown in patients with COVID-19. Troponin testing is not routinely performed in patients attending the emergency department, and the wider body of community-positive patients with mild or asymptomatic disease remains largely unassessed. It should also be considered that high-sensitivity troponin is validated in patients with suspected acute coronary syndromes, and the importance of detectable troponin less than the existing upper reference range is equally of interest as the significance of this is unknown.

The Cardiovascular and Pulmonary Imaging in SARS Coronavirus disease-19 (CISCO-19) is one of the few, multicentre studies to prospectively assess multi-organ involvement in the convalescence phase of COVID-19 infection after hospitalization.[5] CISCO-19 is primarily designed to assess myocardial perfusion and associations with coagulation activation, with patients undergoing multidetector CT coronary angiography during the same study visit to take account of epicardial coronary artery disease. The eligibility criteria are focused to patients with an episode of hospital care but no other selection criteria. CISCO-19 will clarify the prevalence of cardiovascular involvement in symptomatic hospitalized patients with or without troponin elevation.

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