

COVID-19-induced myocarditis: A multicenter cardiovascular magnetic resonance study

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Related article

by Haberka et al.

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Since the onset of the COVID-19 pandemic in early 2020, several studies on its cardiovascular complications and the importance of cardiovascular magnetic resonance (CMR) in diagnosis of myocarditis have been published. "A distinct septal pattern of late gadolinium enhancement specific for COVID-19-induced myocarditis: A multicenter cardiovascular magnetic resonance study" [1] compares patients with COVID-19-related myocarditis and non-COVID-19 myocarditis, for CMR findings.

The study was a multicenter, observational study conducted in 5 centers. The authors recruited 552 COVID-19 patients prospectively and 221 patients retrospectively, as the non-COVID-19 group, between 2018 and 2019. The median time interval between acute COVID-19 disease symptoms and CMR was 12 weeks. The COVID-19 group showed a lesser extent of late gadolinium enhancement (LGE), better left ventricular systolic function, lower left ventricular end-diastolic volume (LVEDV) but a higher rate of pericarditis and septal predilection of LGE as compared to the control group. About half of the patients showed a myocarditis-like injury with only 7.5% also having myocardial edema (only 3 had myocardial edema). In those patients, the LGE areas were larger, and pericarditis was more frequent (13.6% vs. 6%; $P = 0.03$). The control group had a higher rate of myocarditis-like injury and pericardial effusion. The authors did not find any relation between the LGE extent and obesity or age.

The use of Lake Louise Criteria (LLC) as markers for myocardial inflammation is useful

in patients with a clinical presentation consistent with acute myocardial inflammation [2]. But in patients without clinical evidence of myocarditis, areas with abnormal LGE may just reflect scars but not acute inflammation. Therefore, observed injuries in patients without associated edema as visualized by T2-dependent CMR may be scars or fibrosis due to other reasons, without active inflammation.

Septal fibrosis, as reported, is not specific for inflammation but, instead, is a frequent pattern in non-ischemic myocardial disease, such as dilated [3] and hypertrophic cardiomyopathy [4] and sarcoidosis [5] and can even be seen in healthy individuals [6]. Myocardium that is exposed to stress may show LV remodeling with myocyte hypertrophy and diffuse interstitial fibrosis, which may also include replacement fibrosis [7]. Therefore, such septal LGE patterns are unlikely due to inflammation and thus, in this context, should be interpreted with caution. Inflammatory injury due to viral disease is usually located in basal to mid-inferolateral regions [8], and studies in post-COVID related myocarditis have reported non-ischemic scar patterns, with some studies showing elevated T1 and T2 values [9] and others with T2 elevation only [10]. Wherever reported though, the pattern of injury was non-ischemic with the most common inferior, inferolateral, basal to mid-region scar [9, 11, 12].

One major limitation, as also pointed out by the authors, is the lack of access to T1 and T2 mapping, which was not available in all centers. In the context of COVID-related myocardial injury, mapping may be the most

accurate test for detecting myocardial inflammation and injury [9].

Pericarditis has not been a frequent finding in COVID-19 patients although in some studies [9, 13], the reported prevalence of pericardial inflammation was up to 40% [13], and most frequently it was adjacent to the lateral wall. It might be important to investigate the extent and prevalence of pericardial injury in those patients to explain the prolonged symptom duration.

COVID-19-related myocardial inflammation appears to follow similar injury patterns as in other viral diseases and, in affected patients, may reflect involvement of their vulnerable myocardial tissue. While the study by Haberka et al. [1] provides interesting results, it contradicts a large body of evidence and confirmatory studies would be needed to demonstrate that septal injury is indeed a specific marker for COVID-19 myocarditis, instead of non-specific fibrosis.

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