

Letter by Barison et al Regarding Article, “Cardiac Magnetic Resonance Postcontrast T1 Time Is Associated With Outcome in Patients With Heart Failure and Preserved Ejection Fraction”

To the Editor:

T1 mapping cardiovascular magnetic resonance (CMR) is achieving growing importance because it allows quantification of diffuse fibrosis not detectable with conventional late gadolinium enhancement technique. Mascherbauer et al¹ wrote an elegant article on the diagnostic and prognostic role of postcontrast T1 mapping in patients with heart failure with preserved ejection fraction, a condition of increasing clinical relevance. In particular, they demonstrated that postcontrast T1 mapping values correlated with established clinical, echocardiographic, hemodynamic, and biohumoral markers of disease and independently predicted major events during a 17±13-month follow-up. They used a relatively simple and robust imaging protocol²: a midventricular short axis was acquired 15 minutes after contrast injection (0.1 mmol/kg gadolinium) using a conventional inversion recovery spoiled gradient-echo sequence with multiple inversion times. Although they did not correct for precontrast T1 values or for hematocrit, to derive the extracellular volume fraction, postcontrast T1 values showed excellent correlation with cardiac fibrosis at histology.

Overall, this article represents a milestone on the diagnostic and prognostic importance of CMR to study interstitial remodeling in patients with heart failure with preserved ejection fraction, which accounts for approximately 50% of heart failure cases and holds poor prognosis. In these patients, CMR has indeed become the gold standard method to assess not only biventricular, biatrial, and pericardial morphology, biventricular volumes, and systolic function, but also ultrastructural characterization.^{3,4} Nevertheless, the authors did not provide a comprehensive analysis of myocardial tissue characterization: after excluding patients with ischemic heart disease, amyloidosis, and sarcoidosis, as well as myocardial areas with late gadolinium enhancement, they did not mention the presence and extent of late gadolinium enhancement in the study population as a conventional marker of macroscopic myocardial fibrosis compared with the microscopic fibrosis assessed with postcontrast T1 mapping. Moreover, no data about precontrast signal intensity (on T1 mapping, T2 mapping, or T2-short tau inversion recovery imaging) were presented as conventional markers of myocardial edema and ongoing cell damage. A comparison between these conventional methods of myocardial tissue characterization with the novel postcontrast T1 mapping findings presented in the article would create a more comprehensive picture on the diagnostic and prognostic role of T1 mapping CMR in patients with heart failure.

Finally, little is known about gadolinium kinetics in patients with heart failure, which is potentially characterized by marked interstitial expansion and abnormal gadolinium wash-in and wash-out kinetics from both the myocardium and the blood pool. In this article, T1 acquisitions were performed only 15 minutes after bolus, with no serial T1 acquisitions to demonstrate a blood–myocardium steady-state equilibrium or to exclude a pathological gadolinium kinetics.

Further studies are required before T1 mapping becomes clinically useful for characterization of the cardiac interstitium and clinical management of patients with heart failure, compared with conventional CMR imaging as well as with other imaging techniques. In particular, future studies are needed to address all potentially relevant parameters (contrast delivery rate, dose, agent, acquisition timing, sequences) known to affect results to improve the sensitivity and reproducibility of the technique across different vendors and different centers.⁵

Disclosures

None.

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