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EDITORIAL COMMENT

Cardiac magnetic resonance in myocarditis – do we need more tools?

Ressonância magnética cardíaca na miocardite – necessitamos de mais ferramentas?

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Based on its unique tissue characterization features, cardiac magnetic resonance (CMR) has become the non-invasive gold standard for the diagnosis of myocarditis and a class I recommendation to identify myocarditis in the latest European heart failure guidelines. The use of CMR in this context may avoid invasive procedures such as coronary angiography and endomyocardial biopsy.

In 2009, the Lake Louise criteria were published for the diagnosis of myocarditis using CMR.[1] These criteria have recently been updated,[2] driven by new data and technical advances, particularly the development of pixel-wise mapping of T1 and T2 relaxation times. The updated Lake Louise criteria adopt a '2 out of 2' approach, with one positive T2-based criterion (as a marker of myocardial edema) and one T1-based criterion (as a marker of myocardial injury), to increase specificity. This update is in line with the most commonly used approach worldwide, in which the combination of T2-imaging and late gadolinium enhancement (LGE) has been widely adopted. Developments in mapping techniques may further improve the accuracy of the technique and can be used to provide evidence for either of these two criteria. Evidence of pericardial involvement and systolic left ventricular (LV) dysfunction or regional wall motion abnormalities (WMA) are supportive criteria, but not essential to the diagnosis.

In this issue of the *Journal*, Ruivo et al. present a retrospective study of 78 patients with myocarditis (according to the original Lake Louise criteria) in which post-processing tissue tracking (TT) was applied to the acquired cine images.[3] As expected, deformation indices assessed by TT correlated with LV ejection fraction and WMA and, although more weakly, with the extent of myocardial necrosis/fibrosis. Despite the limitations inherent to its design, this is an interesting study that supports the potential of myocardial deformation analysis in myocarditis. In this setting, myocardial dysfunction is frequently focal and the predominantly subepicardial involvement may leave the contraction of other myocardial layers unaffected. The use of TT may therefore increase sensitivity for detection of subtle WMA. As the authors state, TT analysis could serve as a surrogate for detection of fibrotic alterations of the myocardium without the need for contrast media. This could be useful in cases in which the administration of contrast agents is not desirable, as is being tested with the combination of T2-based CMR with T1 mapping. However, the correlations described are weak and no clinically meaningful cutoff values could be found in this study, as there is significant overlap between affected patients and the

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published reference values, which hampers its clinical application.

Overall, the use of TT in these patients may increase sensitivity for WMA detection (as a supportive criterion for myocardial inflammation) but is very unlikely to add specificity to the diagnosis of myocarditis. Therefore, TT is not expected to have a significant clinical impact on the diagnosis of myocarditis using CMR, as this study demonstrates, but may prove to be a useful tool in specific scenarios and may add prognostic information to the visual assessment of WMA and quantification of ejection fraction. Further studies are definitely warranted on this topic.

Conflicts of interest

The author has no conflicts of interest to declare.

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