refractory cases, acknowledging that we have no evidence-based data, preferring the less toxic and less expensive drugs (e.g. azathioprine and methotrexate), and tailoring the therapy on the single patient (e.g. cyclophosphamide should be used only in severe cases and avoided in young fertile women because it can cause infertility) and, importantly, with the patient informed consent.

References

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Imaging approach to the assessment of cardiomyopathies using delayed enhancement cardiovascular magnetic resonance

We read the article by Maharholdt et al. with great interest. This excellent review states the potential of delayed enhancement cardiovascular magnetic resonance (DE-CMR) to distinguish between ischaemic and non-ischaemic cardiomyopathies, as well as to differentiate non-ischaemic aetiologies.

The authors propose a non-invasive approach to routine diagnostic evaluation of patients with left ventricular dysfunction (LVD) using DE-CMR to evaluate the presence or absence of delayed enhancement (DE). In the setting of subendocardial or transmural DE, this pattern is consistent with the presence of coronary artery disease (CAD), and ischaemic cardiomyopathy (ICM) is the most likely diagnosis. However, if the pattern of DE is not of the ischaemic type, non-ischaemic cardiomyopathy (NICM) is likely to be present. A single pilot study presented last year at AHA Scientific Sessions by Patel et al. evaluated this approach showing its potential clinical utility.

We would like to point out that our group has recently addressed this issue in 71 patients with LVD without clinical suspicion of CAD as the underlying cause who underwent catheterization and CMR. Twenty-one of the 26 patients with angiographically proven CAD showed subendocardial or transmural DE, whereas only four of the 45 patients without obstructive CAD showed it ($P < 0.001$). Thus, we found an overall sensitivity of 81%, specificity of 91%, and diagnostic accuracy of 87% in determining the presence of obstructive CAD. Our findings are consistent with previous studies that evaluated patients with known CAD, suggesting that DE may be useful in distinguishing LVD related or not to CAD. As we have shown and in line with the results of Patel et al., this differentiation is also feasible in patients with LVD of unclear aetiology.

Late gadolinium enhancement improved information obtained from angiographic data, which may have important diagnostic, prognostic, and therapeutic implications. As suggested by McCrohon et al., patients with subendocardial or transmural scarring and unobstructed coronary arteries may have systolic dysfunction due to a silent previous MI and may be incorrectly diagnosed by coronary angiography as patients with NICM. In contrast, patients without scarring and with one-vessel disease with no proximal stenosis of a major coronary artery should be considered as having NICM from a diagnostic and prognostic point of view. As the absence of DE-CMR excludes the presence of significant CAD, it may be unnecessary to perform diagnostic coronary angiography routinely in this setting.

However, as Maharholdt et al. conclude, in addition to the diagnostic utility of DE-CMR, its independent prognostic value will need to be determined.

References


Imaging approach to the assessment of cardiomyopathies using delayed enhancement cardiovascular magnetic resonance: reply

We appreciate the interest of Soriano et al. in our review article1 and welcome their interesting data evaluating the potential clinical utility of delayed enhancement (DE) for the assessment of cardiomyopathies. They confirm that the majority of heart failure patients diagnosed with coronary artery disease (CAD) by angiography show typical subendocardial or transmural DE (81%), whereas this pattern of enhancement is rare in patients without CAD (9%). Thus, these findings support the conclusion of our review article that DE assessed by cardiovascular magnetic resonance is capable of differentiating between ischaemic and non-ischaemic forms of cardiomyopathy in patients with large, diffusely hypокontractile ventricles.

References