

Authors' response

We thank Yalcinkaya et al. for their interest in our article [1]. We agree with their conclusion that late gadolinium enhancement during cardiovascular magnetic resonance (CMR) provides incremental information for differentiating a range of structural changes in conditions similar to the one discussed in our case report. The ability of CMR to characterize myocardial tissue was the main focus of our discussion.

The authors correctly mention that amyloid patients show differences in longitudinal relaxation time (T1) between subepicardium, myocardium, subendocardium, and blood. Whilst our patients demonstrated early subendocardial passage through the null point (during T1 mapping) similar to amyloid patients and generally poor nulling with diffuse linear midmyocardial wall hyperenhancement, the hyperenhancement was most apparent in the mid and basal inferolateral and anterolateral left ventricular segments, a pattern not commonly seen in amyloidosis. This persuaded us to proceed to cardiac biopsy to conclusively establish the diagnosis. We also would like to point out that newer T1 mapping based techniques, such as modified look-locker inversion recovery sequences, have been shown to robustly estimate the extent and severity of diffuse interstitial fibrosis that may be missed by conventional late gadolinium enhancement imaging. We anticipate that these newer sequences will be increasingly used during CMR examination of diffusely infiltrating pathological conditions, such as amyloidosis [2].

Yalcinkaya et al. indicate that tagging of the myocardium during CMR may be able to demonstrate any active displacement and deformation of tags in regions conflicting with septal hyper-

trophy. This is correct, although we are unable to comment on myocardial tagging findings as it was not performed in our case. The complexity and time taken for acquisition and analysis of tagging has made the transition of this technique from a research tool into everyday use very challenging. We however note that with the recent availability of feature-tracking CMR, some of the limitations of myocardial tagging are easily overcome and the former may become an integral part of CMR examination to detect abnormal strain and strain rate patterns in amyloid patients [3].

In conclusion, we entirely agree with the authors that tissue characterization ability of CMR makes this modality first amongst the others, compared to other competing techniques in the assessment of diffusely infiltrating conditions, such as amyloidosis. With the availability of newer sequences based on T1 mapping we speculate that CMR will only widen this lead over other imaging modalities.

Conflict of interest: none declared

References

1. Dwivedi G, MacFadyen RJ, Varma C, Sharma V, Neil D, Steeds RP. Progressive breathlessness in an Afro Caribbean hypertensive subject. *Cardiol J*, 2012; 19: 646-649.
2. Brooks J, Kramer CM, Salerno M. Markedly increased volume of distribution of gadolinium in cardiac amyloidosis demonstrated by T1 mapping. *J Magn Reson Imag*, 2013 Feb 28. doi: 10.1002/jmri.24078. [Epub ahead of print].
3. Hor KN, Gottliebson WM, Carson C et al. Comparison of magnetic resonance feature tracking for strain calculation with harmonic phase imaging analysis. *J Am Coll Cardiol Cardiovasc Imag*, 2010; 3: 144-145.

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