

Letter by Bulluck et al regarding article, “Dynamic Edematous Response of the Human Heart to Myocardial Infarction: Implications for Assessing Myocardial Area at Risk and Salvage”

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We read with great interest the recent article by Fernandez-Jimenez et al.¹, in which 16 ST-segment elevation myocardial infarction (STEMI) patients each underwent 5 cardiovascular magnetic resonance (CMR) scans following primary percutaneous coronary intervention (PPCI). They concluded that myocardial edema detected by T2-CMR follows a bimodal pattern, with the first phase appearing at 3 hours and dissipating by 24 hours, and the second phase appearing at day 4. The authors should be congratulated for completing such a challenging study.

However, whether the observed bimodal pattern in edema detected by T2-STIR in their study was actually due to the presence of intramyocardial hemorrhage (IMH) attenuating the T2-signal at 24 hours, cannot be excluded. O'Regan et al² has reported that the signal intensity-based thresholding technique underestimates the edema-based area-at-risk (AAR) quantified by T2-STIR in STEMI patients when IMH is present. Carrick et al³ found that the bimodal pattern of edema detected by T2-mapping was only observed in STEMI patients with IMH, whereas in the absence of IMH, only a unimodal pattern of edema was observed. Furthermore, Fernandez-Jimenez et al.¹ used T2-STIR imaging to detect IMH, which is less reliable than T2* CMR imaging for detecting IMH⁴. Therefore, the analysis undertaken by the authors to adjust for the presence of IMH should be interpreted with caution.

Interestingly, reviewing the patient-level data provided in supplemental figures 3 and 4 of the study revealed that not all STEMI patients displayed a bimodal edema pattern, being present in patients with large infarct sizes and IMH, but absent in those with smaller infarcts and lack of IMH. Therefore, it may have been more informative to present the data on the edema-based AAR by T2-STIR according to the presence or absence of IMH – this would determine whether the biphasic pattern of edema is present in STEMI patients without IMH.

Fernandez-Jimenez et al.¹ also suggested measuring edema-based AAR between days 4 and 7 following STEMI, as the AAR appeared to plateau between these 2 time-points. However, there was a large variability in edema-based AAR measured at days 4 and 7, and not all STEMI patients showed that the AAR had plateaued between these 2 time-points. Moreover, when we extracted patient-level data from supplemental figure 4, and undertook Bland-Altman analysis, we found wide limits of agreement ($\pm 14\%$ of the left ventricle) but no bias between these 2 time-points, confirming that there was substantial variability in the AAR measured at days 4 and 7, and questioning whether the AAR has plateaued between these 2 time-points.

We fully support the authors' view that there is a need to standardize the timing of CMR following PPCI so the assessment of infarct size, AAR and IMH can be standardized in future clinical studies. We have previously found that there was variation not only in the timing of CMR, but also in the technical approach to performing and analyzing CMR scans⁵, and therefore standardization of all aspects surrounding the use of CMR in the acute STEMI setting is warranted.

References

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