

Detection of intramyocardial haemorrhage by MRI —no single rule

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The Review by Betgem *et al.* (Intramyocardial haemorrhage after acute myocardial infarction. *Nat. Rev. Cardiol.* doi:10.1038/nrcardio.2014.188)¹ provides an excellent overview of the pathophysiology and clinical correlates of intramyocardial haemorrhage (IMH) after acute myocardial infarction. The authors identify cardiovascular MRI as the reference standard clinical assessment for detection of IMH and suggest that “T2* seems to be the most sensitive cardiac MRI sequence to detect intramyocardial haemorrhage.” However, no direct literature was provided to support that conclusion.

As mentioned in the Review, both T2 and T2* MRI sequences have been used to detect IMH. However, widespread clinical uptake of both approaches remains limited, in part owing to the long acquisition time in comparison to anatomical or cine imaging, and high sensitivity to motion artefact.² Patients who have had a myocardial infarction might not tolerate the long breath holds required for image acquisition, a factor which is not accounted for in animal studies.

In fact, the question of whether a single imaging sequence is the most sensitive for detection of IMH is difficult to answer based on the current literature. In a canine model of IMH, T2 and T2* imaging seem to have similar accuracy in comparison to histology, with 98% (94–100%) sensitivity and 90% (83–98%) specificity for detection of IMH by T2 imaging (from 80 images) and 95% (86–100%) sensitivity and 94% (88–100%) specificity for detection by

T2* imaging (from 55 images).³ However, direct comparisons of T2 and T2* for IMH in humans are sparse. The investigators of one study suggested that T2* imaging provides higher contrast than T2,⁴ however, we are aware of only one blinded head-to-head clinical comparison of T2 and T2*.² In that study T2 imaging detected IMH in 43% of patients ($n = 49$), as compared to 34% of these same patients with IMH detected by T2*.² The authors suggested that the increased sensitivity of T2 imaging was mostly due to improved image quality. Detection rates with T2-weighted imaging corresponded more closely than T2* to the independent MRI technique of susceptibility weighted imaging.² Additionally, detection of IMH by T2 imaging in the acute phase postinfarction is the only form of imaging that has direct evidence of prognostic utility in patients.⁵ Eitel *et al.* found that IMH detected by T2 imaging was associated with major adverse cardiovascular events at 6 months (16.4% in those with IMH versus 7.0% in those without, $n = 346$; $P = 0.006$), a factor that remained significant even after accounting for infarct size and other patient variables. To our knowledge, an independent prognostic association has not been found for IMH detected by T2* imaging.

The optimal method to detect IMH by cardiovascular MRI continues to be the subject of research. We suggest that at the present time, advice to dismiss T2 imaging might be premature and not supported by current evidence. A combination of T2 and

T2* imaging, therefore, remains the advised method to detect IMH following acute myocardial infarction.

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Competing interests

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