for the significance of the difference between areas under the ROC curves). During follow-up, 63 deaths occurred: 34 ATTRwt, 29 ATTRm. Whilst native T1 was not predictive of death, ECV was (HR, 1.130; 95% confidence interval, 1.06–1.2; p<0.001) and remained independent after adjustment for age, N-terminal pro b-type natriuretic peptide, left ventricular (LV) ejection fraction, E/E’, LV mass index, global longitudinal strain and tricuspid annular plane systolic excursion.

Conclusions CMR-determined native myocardial T1 and ECV provide excellent diagnostic accuracy for identification of ATTR cardiac amyloidosis and both variables track DPD-determined amyloid burden well. In this study, whilst T1 was not a predictor of mortality, ECV was independently associated with mortality.

USPIO-ENHANCED CMR COMPREHENSIVE METHODOLOGICAL INVESTIGATION AND APPLICATION IN ACUTE MI

Introduction Quantification of active myocardial inflammation may improve diagnosis, guide management and provide trial end-points for novel therapies. Ultrasmall particles of iron oxide (USPIO) are phagocytosed by activated leukocytes and USPIO-enhanced CMR is increasingly used to assess tissue inflammation. We aimed to: 1. Compare T2* imaging with T1 mapping, which is proposed as an alternative for ‘native’ cardiac iron measurement; 2. Determine whether imaging at a single time point post-USPIO is sufficient to detect active accumulation in tissue; 3. Determine whether USPIO signal from infarct and remote zones in acute myocardial infarction (MI) reflects active myocardial accumulation or passive ‘wash-through’ in oedematous myocardium.

Methods Four healthy volunteers and six patients with acute MI underwent 1.5T CMR, including T1 and T2* mapping, before and at multiple time points following 4 mg/kg ferumoxytol.

Results Normalised T2* of spleen, an organ with high active leukocyte activity, dropped post-USPIO and remained low over the study period (Figure 1), with no correlation seen between spleen T2* and blood T1 (rho=−0.43, p=0.875). In comparison, T1 recovery in spleen correlated strongly with T1 recovery in blood (rho=0.924, p<0.001). In healthy myocardium, an organ with low leukocyte activity, T1 and T2* recovery both correlated strongly with blood T1 (rho=0.953, p<0.001; rho=0.935, p<0.001 respectively).

In MI, absolute T2* values dropped and remained significantly lower in infarcted (15 vs 27ms, p<0.001, 22 vs 38 ms, p=0.001) and remote myocardium (21 vs 27ms, p=0.05, 28 vs 38 ms, p=0.024) compared to healthy controls. T2* and T1 recovery curves post-USPIO were significantly different in both infarcted (p=0.028) and remote myocardium (p=0.004; Figures 2).

Conclusions 0T2* is sensitive to active tissue accumulation of USPIO, likely because T2* reflects field gradients, such as those generated by compartmentalised (phagocytosed) USPIO. T1, which is due to short range dipolar interactions that reduce as USPIOs wash-out, simply tracks passive wash-through. T1 is therefore less suitable for detecting active leukocytes. Measuring T2* at a single time point post-USPIO is insufficient to determine tissue accumulation from passive wash-through. USPIO signal from infarct and remote zones in acute MI appears to genuinely reflect active myocardial accumulation, presumably due to phagocytosis by activated leukocytes.