

WALKING POSTER PRESENTATION

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Pathophysiology of myocardial remodeling in survivors of ST-elevation myocardial infarction revealed by native T1 mapping: inflammation, remote myocardium and prognostic significance

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Background

The pathophysiology and prognostic significance of remote myocardium in the natural history of STEMI is uncertain. Cardiac magnetic resonance (CMR) provides a non-invasive assessment of myocardial pathology that is spatially and temporally coordinated. Native T1 quantified by CMR (T1 relaxation time, milliseconds) is a fundamental tissue property determined by water content and cellularity. We aimed to investigate the clinical significance of remote myocardium in survivors of acute ST-elevation myocardial infarction (STEMI) using native T1 mapping.

Methods

We performed a prospective single center cohort study in reperfused STEMI patients who underwent CMR 2 days and 6 months post-MI and long term follow-up (18 months minimum). Native T1 CMR (MOLLI investigational prototype sequence: 3 (3) 3 (3) 5) was measured in regions-of-interest in remote and injured myocardium. Infarction was depicted on late gadolinium contrast enhancement imaging. Adverse remodeling was defined as an increase in left ventricular end-diastolic volume $\geq 20\%$ at 6 months. Major adverse cardiac events (MACE) were defined as cardiac death or hospitalization for non-fatal MI or heart failure. Results are mean \pm SD unless specified.

Results

300 STEMI patients (mean age 59 years, 74% male) gave informed consent (14 July 2011 - 21 November 2012). Of these, 288 STEMI patients had evaluable native T1 CMR and follow-up data (median duration 845 days). Infarct size was $18\pm 14\%$ of left ventricular mass. Two days post-STEMI, native T1 in remote myocardium was lower than native T1 in the infarct zone (961 ± 25 ms vs. 1097 ± 52 ms; $p < 0.01$). In multivariable linear regression, remote zone native T1 was independently associated with incomplete ST-segment resolution (9.42 (2.37 to 16.47); $p = 0.009$), the log of the initial CRP concentration (regression coefficient 3.01 (95% CI 0.016 to 5.55); $p = 0.038$) and the peak monocyte count within 2 days of admission (10.20 (0.74, 19.67); $p = 0.035$).

At 6 months, left ventricular end-diastolic volume increased by 5 (25) ml ($n = 262$ patients with evaluable data) overall, and adverse remodeling occurred in 30 (12%) patients. Remote zone native T1 was a multi-variable predictor of the change in left ventricular end-diastolic volume from baseline (0.13 (0.01, 0.24); $p = 0.035$).

39 (13.5%) patients experienced a MACE including 20 (6.9%) patients with a post-discharge MACE. Remote zone native T1 was an independent predictor of post-discharge MACE (hazard ratio 1.016, 95% CI 1.000, 1.032; $p = 0.048$) including after adjustment for changes in LVEF ($p = 0.032$), LV end-diastolic volume ($p = 0.053$), and monocyte count ($p = 0.036$).

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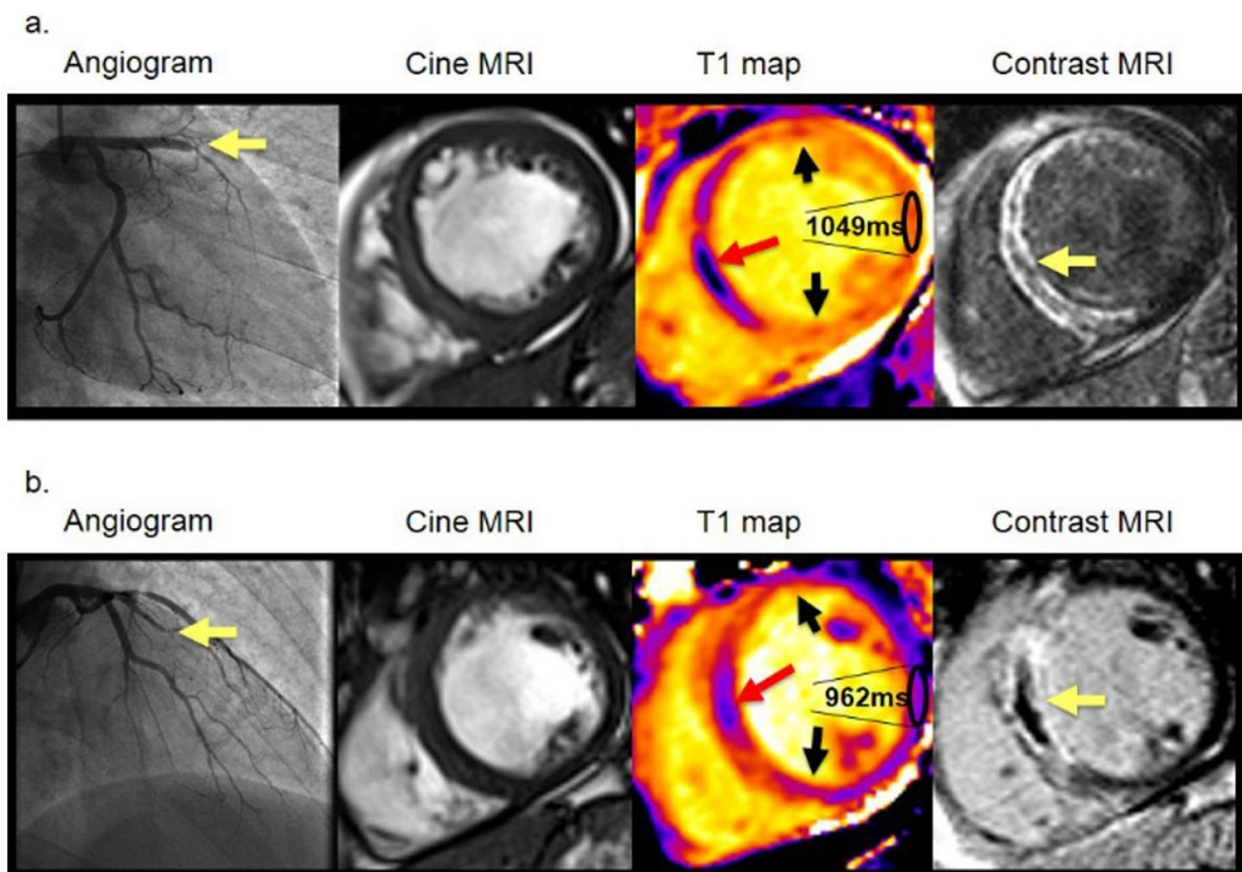


Figure 1 Two patients with acute anterior STEMI treated by primary PCI and with the same standard anti-thrombotic therapies. Each patient had TIMI grade 3 flow at the end of the procedure. (a) Patient with high remote zone native T1. Six month follow-up MRI revealed final infarct size was 39.2% of left ventricular mass and significant adverse remodeling occurred with left ventricular end-diastolic volume of 145.7 ml/m². This patient was subsequently hospitalised for new onset heart failure and had an defibrillator device implanted. (b) Patient with average remote zone native T1 value. The infarct size at 6 months revealed by contrast-enhanced MRI was 31.0% of left ventricular mass and left ventricular end-diastolic volume of 84.3 ml/m². This patient had an uncomplicated clinical course.

Conclusions

Remote zone tissue characteristics early post-MI are temporally linked with reperfusion injury and inflammation and independently predict left ventricular remodeling and MACE in STEMI survivors.

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