

## 1 LONG-TERM PROGNOSIS AFTER ACUTE ST-SEGMENT ELEVATION MYOCARDIAL INFARCTION IS DETERMINED BY CHARACTERISTICS IN BOTH NON-INFARCTED AND INFARCTED MYOCARDIUM ON CARDIOVASCULAR MAGNETIC RESONANCE IMAGING

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**Background** After acute ST-segment elevation myocardial infarction (STEMI), CMR infarct characteristics including infarct size (IS), microvascular obstruction (MVO) and intramyocardial haemorrhage (IMH) as well as left ventricular ejection fraction (LVEF) carry prognostic value. However, the long-term prognostic value of changes in the non-infarcted myocardium is unknown.

**Aim** To evaluate acute changes in both the non-infarcted and infarcted myocardium post-STEMI and their long-term predictive value of major adverse cardiac events (MACE) using conventional CMR and T1-mapping indices.

**Methods** 219 patients undergoing primary percutaneous coronary intervention post-STEMI prospectively underwent CMR (3T) at 2 days and 6 months, with long-term follow-up for MACE – a composite of cardiac death, sustained ventricular arrhythmia and new-onset heart failure. CMR assessed LVEF, IMH, area-at-risk (AAR), IS and MVO using cine, T2-weighted, T2\*, T1-mapping and late gadolinium enhancement (LGE) imaging. Area without LGE was defined as non-infarcted myocardium (figure 1A). Infarct and non-infarct T1 were derived from T1-maps using anatomically matching LGE images. “High non-infarct T1” was defined as T1 > 1250 ms (>2SD above normal range 1184±30 ms). Within the

infarction, presence of MVO/IMH can lower infarct T1; this was dichotomised into “Higher infarct T1” (≥1300 ms) and “Lower infarct T1” (<1300 ms). Conventional CMR markers (LVEF, AAR, IS, MVO, IMH) and novel T1-mapping biomarkers were assessed for their ability to predict MACE using Kaplan-Meier and Cox-regression survival analysis.

**Results** 22/219 patients experienced a MACE at a median of 4 years (IQR 2.5–6 yrs). High non-infarct T1 was associated with lower LVEF (51 vs 55%, p=0.002) and higher NT-pro-BNP levels (290 vs 170 pg/ml, p=0.008) at 6 months, and a 2.5-fold increased risk of long-term MACE (figure 1B; p=0.035). Lower infarct T1, implying MVO/IMH, was associated with a 3-fold risk of MACE (p=0.020). AAR and IMH were not significant predictors of MACE. Both non-infarct T1 (p<0.001) and infarct T1 (p=0.003) remained independent predictors of MACE after adjusting for age, history of MI, ischaemic time, and peak troponin; both significantly improved risk-prediction beyond LVEF<40%, IS and MVO (figure C; C-statistic 0.69±0.06 vs 0.77±0.06, net reclassification index 42%; p=0.027).

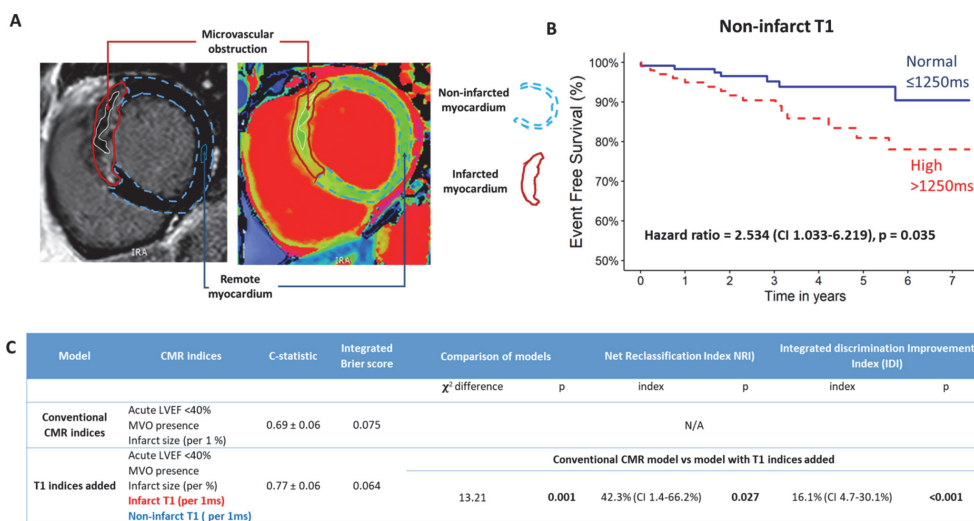
**Conclusions** Both acute non-infarct and infarct myocardial T1 on CMR post-STEMI are independent and incremental predictors of long-term MACE beyond conventional CMR biomarkers.

## 2 ASSESSING SPLENIC SWITCH-OFF IN ADENOSINE STRESS CMR FOR PATIENTS WITH ATRIAL FIBRILLATION: A PROPENSITY MATCHED STUDY

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**Background** Stress cardiac magnetic resonance imaging (CMR) is a valuable investigation in the functional assessment of coronary artery disease. The phenomenon of splenic switch-off is a validated indicator of adequate vasodilator stress unique to adenosine vasodilator stress CMR. Patients in atrial fibrillation (AF) may have a reduced adenosine response due to a lower



**Abstract 1 Figure 1** (A) T1-maps were segmented into infarcted and non-infarcted myocardium using anatomically matched LGE images. (B) Kaplan-Meier survival analysis of patients with and without high non-infarct T1 on the acute CMR scan after STEMI. (C) Multi-variable Cox regression analysis and comparisons of predictive power of models of conventional and novel CMR indices for predicting long-term MACE were performed.