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

^{1,2,3}Mayooran Shanmuganathan, ²Ambra Masi, ^{2,3}Matthew K Burrage, ^{1,3}Rafail A Kotronias, ¹Alessandra Borlotti, ^{1,3}Roberto Scarsini, ¹Abhirup Banerjee, ^{1,3}Dimitrios Terentes-Printzios, ²Qiang Zhang, ²Evan Hann, ²Elizabeth Tunnicliffe, ³Andrew Lucking, ³Jeremy Langrish, ³Rajesh Kharbanda, ^{1,3}Giovanni Luigi de Maria, ^{1,3}Adrian P Banning, ^{1,3}Robin P Choudhury, ^{1,3}Keith M Channon, ²Stefan K Piechnik, ^{1,2,3}Vanessa M Ferreira, on behalf of Oxford Acute Myocardial Infarction (OxAMI) Study investigators. ¹Acute Vascular Imaging Centre (AVIC), University of Oxford, John Radcliffe Hospital, Oxford, OX3 9DU, UK; ²Oxford Centre for Clinical Magnetic Resonance Research (OxCMR), John Radcliffe Hospital, NIHR Oxford Biomedical Research Centre, Oxford BHF Centre of Research Excellence, University of Oxford, Oxford, UK; ³Oxford University Hospitals NHS Trust, John Radcliffe Hospital, Oxford, OX3 9DU, UK

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Background After acute ST-segment elevation myocardial infarction (STEMI), CMR infarct characteristics including infarct size (IS), microvascular obstruction (MVO) and intra-myocardial 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

Adam Barrishi, John Graby, Ali Khavandi, Amardeep Dastidar, Jonathan Rodrigues. Department of Radiology, Royal United Hospital; Faculty of Health Sciences, University of Bristol; Department of Cardiology, Royal United Hospital; Department for Health, University of Bath; Department of Cardiology, North Bristol NHS Trust

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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.