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 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

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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.

hyperaemic coronary flow reserve. Thus, we hypothesised that patients in AF would achieve splenic switch-off less frequently than those in sinus rhythm (SR).

Methods 1100 adenosine stress CMR studies were retrospectively identified from a prospectively maintained clinical CMR database over a 5-year period (2016–2021). 71 patients in AF on ECG at the time of CMR were identified. The SR cohort was drawn by propensity score matching to the AF group for age, sex, and body mass index. The adenosine dose administered, symptoms, and heart-rate change observed during infusion and scan result were recorded. Splenic switch-off was evaluated subjectively through visual assessment and semi-quantitatively via changes in splenic and myocardial signal intensity from rest to stress. Multivariate logistic regression analysis assessed for predictors of splenic switch-off.

Results 70 AF patients were matched to 70 in SR (mean age: 72 ± 8 years vs 72 ± 9 years, $p=0.96$; %male: 86% vs 84%, $p=0.81$). Subjective splenic switch-off occurred significantly less frequently in AF than in SR (34/70 [49%] vs 53/70 [76%], $p=0.003$). Objective assessment supported this, with a smaller splenic signal intensity difference between stress and rest in AF vs SR (median splenic stress:rest peak signal intensity ratio 0.92 [IQR, 0.61–1.11] vs 0.56 [IQR, 0.45–0.75], $p<0.001$). A heart-rate increase over 10bpm in response to adenosine was a significant predictor of visual splenic switch-off in SR patients but not in AF patients. Fewer AF patients had inducible ischaemia than SR patients (9/70 [13%] vs 17/69 [25%], $p=0.058$). However, this difference was not driven by inducible ischaemia rates between AF and SR patients who did not achieve splenic switch-off (6/36 [17%] vs 4/17 [24%], $p=0.403$).

Conclusions In this retrospective propensity matched study, splenic switch-off occurred significantly less frequently in patients with AF. This may risk the under-diagnosis of inducible ischaemia, but requires further assessment.

3 RATIONALE AND DESIGN OF THE MEDICAL RESEARCH COUNCIL PRECISION MEDICINE WITH ZIBOTENTAN IN MICROVASCULAR ANGINA (PRIZE) TRIAL MRI SUB-STUDY

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Introduction Microvascular angina is caused by cardiac small vessel disease and dysregulation of the endothelin system is implicated. The chronic elevation of circulating ET-1 in microvascular angina may be influenced by genetic factors. The minor G allele of the non-coding single nucleotide polymorphism (SNP) rs9349379 enhances expression of the endothelin 1 gene in human vascular cells, increasing circulating concentrations of ET-1. The prevalence of this allele is higher in patients with microvascular angina. Zibotentan is a potent, selective inhibitor of the ETA receptor. We have identified zibotentan as a potential disease-modifying therapy for patients with microvascular angina.

The Precision medicine with Zibotentan in microvascular angina (PRIZE) trial is a prospective, randomized, double-blind, placebo-controlled, sequential cross-over trial. We will assess the efficacy and safety of adjunctive treatment with oral zibotentan (10 mg daily) in patients with microvascular angina and assess whether rs9349379 (minor G allele; population prevalence ~36%) acts as a theragnostic biomarker of the response to treatment with zibotentan. The participants will receive a single-blind placebo run-in followed by treatment with either 10 mg of zibotentan daily for 12 weeks then placebo for 12 weeks, or vice versa, in random order.

After randomisation in PRIZE, subjects will be invited to participate in the cardiac MRI sub-study. Myocardial perfusion is generally impaired in patients with microvascular angina. The rationale for undertaking this MRI sub-study is to determine whether, compared with placebo, treatment with zibotentan improves myocardial blood flow.

Methods Patients will undergo multiparametric CMR at three points (baseline prior to therapy and after each 12 week treatment phase). At each scan they will have assessments of myocardial blood flow (both rest and stress using the Kellman quantitative perfusion method), left ventricular function and mass, aortic stiffness assessment and tissue characterisation (T1 mapping (MOLLI and ShMOLLI), T2 mapping and late gadolinium enhancement imaging).

Conclusion PRIZE invokes precision medicine in microvascular angina. Should our hypotheses be confirmed, this developmental trial will inform the rationale and design for undertaking a larger multicentre trial. The MRI sub-study contributes to this study by providing vital mechanistic and safety information.

4 INTER-MODALITY AGREEMENT AND TEST-RETEST REPRODUCIBILITY OF CMR AND ECHOCARDIOGRAPHY FOR ASSESSING MYOCARDIAL DEFORMATION IN TYPE 2 DIABETES MELLITUS

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Background Inter-modality agreement allows interchangeable use of imaging modalities that is crucial for clinical decision-making, while the reproducibility of a technique is fundamental for monitoring disease progression or response to treatment. We aimed to investigate the test-retest reproducibility and inter-modality agreement of transthoracic echocardiography (TTE) and cardiac magnetic resonance (CMR) imaging in assessing left ventricular (LV) and left atrial (LA) myocardial deformation in people with type 2 diabetes mellitus (T2D).