Abstract 112 Figure 2 ROC analysis of MPR assessed using CMR for detecting microvascular ischaemia as defined by high IMR (>40) in the absence of significant epicardial stenosis ($\text{FFR} > 0.8$). True positives: high IMR > 40 ; true negatives: normal IMR < 20 . Area under the curve 0.87 ± 0.06 .

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NOVEL DIAGNOSTIC CRITERION FOR MICROVASCULAR ISCHAEMIA USING CMR PERFUSION IMAGING: VALIDATION AGAINST INVASIVE INDEX OF MICROVASCULAR RESISTANCE

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Background In over 50% of patients with angina, the underlying ischaemia is related to microvascular dysfunction (MVD), rather than epicardial coronary artery disease (CAD). Clinically, MVD remains a major diagnostic challenge, which

hinders targeted therapy and confers impaired clinical outcomes. Myocardial perfusion reserve (MPR), as assessed by cardiovascular magnetic resonance (CMR), is impaired in those with microvascular angina. We sought to objectively diagnose microvascular ischaemia using CMR by defining an MPR cut-off, validated against invasive coronary microvascular physiology (Index of Microvascular Resistance, IMR).

Methods 75 subjects (50 patients with angina and suspected CAD; 25 healthy controls) underwent CMR to assess LV function, MPR (adenosine stress/rest first-pass perfusion imaging) and viability (late gadolinium enhancement). All patients underwent invasive coronary angiography with pressure-wire assessment of IMR and fractional flow reserve (FFR). A total of 120 coronary arteries were assessed. CMR images were analysed by observers blinded to clinical and angiographic data. MPR was defined as the ratio of stress/rest myocardial signal intensity upslope gradients during gadolinium first-pass perfusion imaging, normalised to LV blood pool enhancement. Infarcted myocardium was identified using LGE and excluded from analysis.

Results For reference, myocardium downstream of significant epicardial stenosis ($\text{FFR} < 0.8$) had lower MPR than healthy controls (1.3 ± 0.4 vs 1.9 ± 0.4 , $p < 0.001$, figure 1). Downstream of unobstructed epicardial coronary arteries ($\text{FFR} > 0.8$), non-infarcted myocardium had intermediate MPR: (unobstructed-CAD 1.6 ± 0.4 , obstructed-CAD 1.3 ± 0.4 ; controls: 1.9 ± 0.4 , $p < 0.001$ by ANOVA). When further stratified by IMR, myocardium with $\text{IMR} < 20$ had comparable MPR to normal controls (1.9 ± 0.5 vs 1.9 ± 0.4 , $p = 0.98$); as IMR increased, there was progressive reduction in MPR ($\text{IMR} < 20$: 1.9 ± 0.5 , $\text{IMR} 20-40$: 1.5 ± 0.4 , $\text{IMR} > 40$: 1.3 ± 0.4 ; all $p < 0.01$ by ANOVA). Myocardium with high $\text{IMR} > 40$ but unobstructed epicardial coronary arteries had equivalent MPR to ischaemic myocardium supplied by significant epicardial stenosis (1.3 ± 0.4 vs 1.3 ± 0.4 , $p = 0.48$, figure 1). Downstream of unobstructed epicardial coronary arteries of CAD patients, MPR 1.5 detected microvascular ischaemia (defined by $\text{IMR} > 40$) with a sensitivity of 82%, specificity of 83%, and accuracy of 83% on ROC analysis ($\text{AUC } 0.87 \pm 0.06$, figure 2).

Conclusions Microvascular ischaemia can be objectively diagnosed using CMR perfusion imaging. Reduced MPR is related to increased microvascular resistance, as validated by invasive

Abstract 113 Figure 1 In myocardium downstream unobstructed epicardial coronary arteries, myocardial perfusion reserve (MPR) decreases as index of microvascular resistance (IMR) increases. Myocardium with high $\text{IMR} > 40$ had equivalent MPR to ischaemic myocardium supplied by significant epicardial stenosis. Bars represent mean $\pm$ SD. FFR = fractional flow reserve.

IMR. The novel MPR criterion of 1.5, to detect high IMR>40, can confirm the clinical diagnosis of microvascular ischaemia, enabling targeted therapy and disease monitoring.

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DETECTING ISCHAEMIA IN FLOW LIMITING MULTI-VESSEL DISEASE – IS 3D PERFUSION CMR WHERE THE MONEY LIES?

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Introduction Myocardial perfusion cardiovascular magnetic resonance (CMR) is a highly accurate non-invasive imaging modality in the diagnosis of coronary artery disease (CAD). High-resolution (hi-res) two-dimensional (2D) perfusion CMR can better detect sub-endocardial ischaemia although with the disadvantage of lesser myocardial coverage. Three-dimensional (3D) perfusion provides whole heart coverage but has a comparatively inferior resolution. The superiority of fractional flow reserve (FFR) over visual angiographic assessment in determining functional significance of a coronary stenosis is now well established. We studied the diagnostic agreement

between hi-res 2D and 3D perfusion CMR in patients with significant multi-vessel flow limiting CAD as confirmed by FFR on a per patient and per vessel basis.

Methods Patients with suspected stable CAD referred for invasive coronary angiography as part of their routine clinical care were prospectively recruited. Prior to revascularisation (if performed) all patients underwent both hi-res 2D adenosine vasodilator stress and 3D adenosine stress perfusion scans during the same sitting. Visually, coronary stenoses less than 50% were deemed not to be flow limiting, whereas those 80% or more were considered as flow limiting. For stenoses between 50%–80% FFR study was performed, with FFR of 0.8 or less considered functionally significant. Blinded, independent, qualitative visual analysis by two experienced readers was performed to confirm existence of true perfusion defects on both 2D and 3D perfusion CMR datasets. Vascular territories in relation to CMR perfusion defects were assigned as per AHA 16 segment classification.

Results Prevalence of CAD was 62%. Of the 29 patients studied, 6 (21%) had single-vessel disease, 8 (27%) had two-vessel disease (2VD), 4 (14%) had three-vessel disease (3VD), and 11 (38%) had no significant CAD. In view of the small sample size qualitative analysis was undertaken to determine concordance between hi-res 2D and 3D perfusion CMR. Prevalence of perfusion defects relating to the three vascular territories have been detailed in Table 1.

Conclusions Our study shows an excellent agreement for both modalities in detecting FFR positive CAD on a per patient basis. On a per vessel basis, between the two, agreement in

Abstract 114 Figure 1 3D perfusion images showing perfusion defects in LAD (see blue arrows) and circumflex territory (see red arrows)