accelerated adenosine stress first-pass contrast enhanced CMR perfusion imaging at 1.5T MRI. Absolute stress MBF was derived. Visual analysis of the dynamic perfusion series was undertaken by two expert CMR readers. Diagnostic accuracy was determined on presence of significant CAD as defined by ICA and FFR ≤ 0.80 using Area under the Curve (AUC) from receiver operator characteristic curves.

**Results** At the patient level, there was significantly lower MBF in patients with CAD compared to those without CAD (2.03 [1.82 – 2.37] vs 2.68 [2.31 – 2.93] ml/g/min, p<0.001). There was a high diagnostic accuracy for the detection of CAD for quantitative stress MBF with AUC 0.84 (95% confidence interval [CI] 0.68–0.94, p<0.001), and for visual analysis with AUC 0.89 (95% CI: 0.75 – 0.97), p<0.0001). These were not significantly different (p=0.52). The optimal threshold for MBF detection of CAD was ≤2.50 ml/g/min, with sensitivity of 100% (95% CI: 77%-100%) and specificity of 71% (95% CI: 49%-87%).

**Conclusion** High-resolution quantitative near whole-heart myocardial perfusion imaging shows good diagnostic accuracy for detection of significant CAD, with comparable accuracy compared to expert visual analysis. This technique could be considered for automated widespread clinical use without the need for expert analysis of visual dynamic perfusion images.

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**5 CORRELATIONS BETWEEN CARDIAC MAGNETIC RESONANCE AND MYOCARDIAL HISTOLOGIC FINDINGS IN FABRY DISEASE**

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**Introduction** Fabry disease (FD) causes cardiac left ventricular hypertrophy (LVH) thought to be due myocyte lipid storage and compensatory sarcomeric increase. Myocardial T1 in FD by cardiac magnetic resonance (CMR) demonstrates T1 lowering, with T1 falling until overt LVH, then normalizing as further LVH occurs. However no histological validation has been provided to date. Therefore, aim of the study was to correlate CMR myocardial values and histologic findings.

**Materials and Methods** Fifteen FD patients (49 years [IQR39–63], 60% females) undergoing CMR (cines, native T1 mapping, LGE) and either endomyocardial biopsy (EMB, n=11) or septal myectomy (n=4). Tissue specimens were analyzed with light/electron microscopy. Histomorphometric analysis measured myocyte vacuolization either as a percent of myocyte number (%VM) or area (%VMA).

**Results** Histological changes preceded imaging changes: myocyte hypertrophy preceded LVH and fibrosis preceded LGE. % VM and %VMA correlated with LVH either as maximal wall thickness (MWT r=0.780, p<0.001; r=0.859, p<0.0001) or left ventricular mass index (LVMi r=0.823, p<0.001; r=0.847, p<0.0001). LVH patients had high%VM (>45%) and ≥80% by elevated MWT and LVMi respectively and high%VMA (>18% and ≥22% respectively). At least 45% of VM and 10% VMA were needed for T1 lowering. In patients without increased LVMi (67%) T1 fell as%VMA increased (r=0.883; p<0.001). In patients with increased LVMi, no clear relationship was reported (r=0.501; p=0.389).

**Discussion** This study compared CMR with myocardial histology in FD patients focusing on storage (vacuolization), LVH and native T1 values. Main findings were:

1. Histological changes preceded imaging changes: myocyte hypertrophy pre detectable LVH, storage pre detectable T1 lowering and fibrosis LGE.
2. Myocyte size increased with storage and clinical LVH.
3. Significant storage was necessary for both clinical LVH and T1 lowering.
4. The relationships between storage and clinical LVH was non-linear.
5. Native T1 values fell with storage until the presence of overt LVH (i.e. increased LVMi), when the relationship trend was lost.

**Conclusion** In FD, histological changes precede imaging changes. LVH correlated with myocyte storage with 45% vacuolization (10% VMA) needed for detectable LTI lowering strongly correlates with myocyte vacuolization in early disease but not in advanced disease.

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