fibrosis. Nine age- and sex- matched post-mortem myocardial samples served as histological controls.

Results When compared to controls, the myocardium of patients with severe AS had reduced vessel density, total quantity of SMA +ve and CD31 +ve, in addition to the expected increase in fibrosis. There was absence of CD31 +ve endothelium in SMA +ve arterioles, indicating endothelial loss. (Figure 1) Importantly, patients with an aortic valve area (AVA) $\leq 0.8\text{cm}^2$ had greater endothelial loss compared to those with an AVA >0.8 and $\leq 1.0\text{cm}^2$ ($1.34\% \pm 0.44\%$ vs $2.84 \pm 1.03\%$, $p=0.006$), and endothelial loss also correlated with myocardial perfusion reserve index (MPRI), $r=0.66$, $p=0.019$. MPRI improved significantly post AVR (from 0.95 ± 0.17 to 1.50 ± 0.43 , $p=0.018$).

Conclusion In severe AS, there is microvascular rarefaction and loss of endothelium, which is more pronounced in patients with the most severe aortic valve narrowing. This appears to be an underlying mechanism for reduced myocardial perfusion reserve, which may be reversible post AVR.

21 INTRACARDIAC THROMBI IN CARDIAC AMYLOIDOSIS, A COMMON FINDING

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Background Cardiac amyloidosis (CA) has been associated with a high prevalence of intracardiac thrombi, but this was reported in small cohorts of high risk patients (with a clinical indication for transoesophageal echocardiography). It is therefore not known whether such observations are applicable to the general CA population.

Purpose To assess the prevalence of intracardiac thrombi in patients with CA.

Methods 324 consecutive patients with CA were studied prospectively using a standard CMR protocol at 1.5T, including early and late gadolinium imaging and T1 mapping. Early

Abstract 21 Figure 1 Acquisition of stack through the LAA. Early gadolinium images of the LAA (bottom row) acquired using a 5 mm contiguous stack through the LAA (top row) and an inversion time of 440 ms to confirm the presence or absence of thrombus vs normal pectinate muscle. The thrombus in the left atrial appendage can only be visualised in the last two images (red arrows in panel 4 and 5) and could have been missed with the acquisition of only one image

gadolinium images (segmented imaging, trigger 2) of the left atrial appendage (LAA) were acquired using a 5 mm contiguous stack and a TI of 440 ms.

Results The study participants comprised 155 with light chain CA (AL), 166 with transthyretin amyloidosis (ATTR), 2 with Apo A-I, and 1 with Apo A-IV CA. The prevalence of intracardiac thrombi was 5.2% in AL, 7.2% in ATTR; 6.2% overall. 90% of thrombi were in the LAA. This was higher when there was atrial fibrillation (9.1% AL, 14.3% ATTR) but intracardiac thrombi were also present in sinus rhythm (SR) 3.1% (4.5% AL, 1.1% ATTR). In all patients with AF the thrombi were present despite long term anticoagulation. The presence of intracardiac thrombi was associated with a greater degree of systolic dysfunction and myocardial amyloid infiltration (higher native T1 and ECV).

Conclusions The prevalence of intracardiac thrombi in CA and AF is high despite long term anticoagulation, with significant thrombus prevalence even in SR, meriting vigilance for intracardiac thrombi in all. CMR with early gadolinium imaging of the LAA is a valuable screening tool for thrombi in the LAA and should be routine part of the clinical protocol when amyloidosis is suspected. Current guidelines for electrical cardioversion after prolonged anticoagulation without screening for thrombus in the LAA should not be applied to patients with CA.

22 IMPAIRED STRESS-INDUCED OXYGENATION IN HYPERTROPHIC CARDIOMYOPATHY IS ASSOCIATED WITH AN INCREASED RISK OF VENTRICULAR ARRHYTHMIA

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Background Myocardial ischaemia is believed to promote fatal life-threatening ventricular arrhythmias in hypertrophic cardiomyopathy (HCM). Oxygen sensitive cardiac magnetic resonance (CMR) or blood oxygen level dependent (BOLD) imaging can detect blunted myocardial oxygenation during vasodilator stress in HCM. Whether or not impairment in stress oxygenation is associated with ventricular arrhythmia risk is unknown.

Objectives To investigate the relationship between blunted stress oxygenation and ventricular arrhythmia in HCM and examine the determinants of stress oxygenation in HCM.

Methods 103 genotyped HCM patients and 32 (age, gender and body mass index matched) healthy controls underwent adenosine stress BOLD, stress first pass perfusion imaging and late gadolinium imaging (LGE) to assess stress oxygenation, myocardial perfusion reserve index (MPRI), and fibrosis burden respectively. Stress oxygenation response (BOLD ΔSI) was estimated as a relative increase in oxygen sensitive BOLD signal intensity from rest to peak vasodilator stress. All HCM patients had 24-holter monitoring to assess for ventricular tachycardia (≥ 3 beats, ≥ 120 beats per minute).

Results As expected, both MPRI (1.5 ± 0.4 v 2.0 ± 0.3 , $p < 0.0001$) and stress oxygenation ($9.1\% \pm 4.1\%$ v 17.0%)

Abstract 22 Table 1 Univariate and multivariable predictors of ventricular arrhythmia in hypertrophic cardiomyopathy

UVA	OR	95% CI	p-value	MVA	OR	95% CI	p-value
Age	1.04	1.01–1.08	0.02	Age	1.04	0.99–1.08	0.08
Sex	0.95	0.35–2.60	0.92	BOLD $\Delta SI \leq 6.5\%$ (lowest quartile)	3.04	1.02–9.05	0.04
BMI	1.06	0.96–1.16	0.26	LGE mass	1.06	1.01–1.12	0.04
Sarcomere+	0.54	0.22–1.37	0.20	Max LVWT ≥ 30	0.48	0.32–7.151	0.49
Max LVWT ≥ 30	1.50	0.13–17.26	0.75	Syncope	2.08	0.27–15.87	0.86
LV mass	1.01	0.99–1.01	0.26	Family History of SCD	0.27	0.06–1.22	0.11
LVEF	0.96	0.90–1.02	0.19	Abnormal BP response	1.34	0.23–8.05	0.75
LGE mass	1.07	1.02–1.13	0.005				
MPRI	0.43	0.12–1.55	0.43				
LVOT obstruction	0.78	0.20–3.06	0.72				
Family history of SCD	0.40	0.11–1.48	0.17				
Syncope	3.21	0.60–7.04	0.17				
Abnormal BP response	1.88	0.42–8.47	0.41				
BOLD $\Delta SI \leq 6.5\%$ (lowest quartile)	3.86	1.47–10.11	0.006				

Abnormal BP response Abnormal Blood Pressure response during exercise; BMI Body Mass Index; BOLD Blood Oxygen Level Dependent imaging; CI Confidence Interval; EF Ejection Fraction; LGE late gadolinium enhancement; LV left ventricular; LVOT Left Ventricular Outflow Obstruction ≥ 30 mm Hg; LVWT left ventricular wall thickness; MPRI myocardial perfusion reserve index; MVA Multi-variable; OR Odds Ratio; SCD sudden cardiac death; *Sarcomere+: presence of sarcomeric mutation; UVA univariate;

$\pm 1.6\%$, $p < 0.0001$, figure 1C) were significantly impaired in HCM patients compared to controls. Among HCM patients, twenty six (25%) had ventricular tachycardia and demonstrated more severe impairment in stress oxygenation ($6.9\% \pm 0.8\%$) than other patients ($9.9\% \pm 0.5\%$, $p = 0.001$). The prevalence of ventricular arrhythmia increased with decreasing quartiles of oxygenation (figure 1F). HCM patients in the lowest quartile of stress oxygenation (BOLD $\Delta SI \leq 6.5\%$) were at a three-fold risk of ventricular arrhythmia (OR 3.04, $p = 0.04$) compared to other patients, despite adjusting

for differences in LGE mass and other sudden cardiac death risk factors (table 1). Sarcomeric mutation status ($\beta = -2.32$, $p = 0.007$) was the only independent predictor of the extent of stress oxygenation after adjusting for univariate confounders (wall thickness, MPRI and LGE mass). Sarcomeric HCM displayed more severe impairment in stress-induced oxygenation than genotype negative HCM and healthy controls (figure 1E).

Conclusion In HCM, impaired stress-induced oxygenation is associated with an increased risk of ventricular arrhythmia and

Abstract 22 Figure 1 (A) Grey scale rest and stress BOLD image from a healthy control (B) BOLD colour maps demonstrate a relative increase in BOLD SI, from rest (green) to stress (red), by 19.8% in healthy control compared to blunted rise in signal intensity in phenotype negative sarcomeric mutation carrier (Sarc+P-) (7.6%) and genotype negative HCM (5.6%) (C) Stress myocardial oxygenation is significantly blunted in HCM patients compared to healthy controls (D) Dissociation between MPRI and BOLD ΔSI can be seen in a Sarc+P patient - MPRI 2.2 (normal) versus BOLD ΔSI 7.6% (low) (E) Sarcomeric HCM had the most blunted stress oxygenation amongst all HCM patients (F) Prevalence of ventricular tachycardia rose with decreasing quartiles of myocardial oxygenation. (Error bars represent mean $\pm$ standard deviation; *** $p < 0.0001$ comparison with control; ## $p = 0.001$ comparison with G-HCM; BOLD SI blood oxygen level dependent imaging signal intensity; MYBPC3 myosin binding protein C 3; MPRI myocardial perfusion reserve index; G-HCM genotype negative HCM; Sarc+Sarcomeric; Sarc+P pre-hypertrophic sarcomeric mutation carriers; VT ventricular tachycardia)

may represent a novel biomarker of arrhythmic risk. Sarcomeric mutation status independently predicts a blunted stress oxygenation response in HCM.

23 PREVALENCE AND CORRELATES OF MID-WALL LATE GADOLINIUM ENHANCEMENT IN PATIENTS WITH ISCHEMIC CARDIOMYOPATHY

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Background Late gadolinium enhancement (LGE) imaging is an established technique for the assessment of myocardial replacement fibrosis/scar. The presence of mid-wall LGE has been described in ~30% of patients with dilated cardiomyopathy (DCM) of non-ischemic aetiology and is known to be associated with poor clinical response to pharmacological therapy and worse outcome. Conversely, little is known regarding the clinical significance of additional mid-wall LGE in patients with ischemic cardiomyopathy (ICM). The aim of the present study was therefore to investigate the prevalence and clinical/imaging correlates of mid-wall LGE in a consecutive cohort of patients with ICM.

Methods The UHSM-redCAP database was searched for patients with ICM (defined as the presence of LV ejection fraction <50% in the context of multivessel disease and/or previous myocardial infarction) who had clinically-indicated CMR with LGE imaging for the assessment of LV volumes, LV regional/global systolic function and presence/extent of myocardial replacement fibrosis/scar.

Results A total of 606 consecutive patients with ICM were identified and included in the study. Mean age of the study population was 64 ± 11 years and 85% of the patients were males. Mid-wall LGE was observed in 34 (6%) patients. No significant difference in age (64 ± 11 vs. 65 ± 11 years), male gender (85% vs. 94%), and prevalence of NYHA functional class 3–4 (29% vs. 32%) was observed between ICM patients without vs. with additional mid-wall LGE.

However, ICM patients with mid-wall LGE had larger LV end-diastolic volume (261 ± 79 ml vs. 220 ± 60 ml; $p < 0.001$), larger LV end-systolic volume (176 ± 74 ml vs. 140 ± 54 ml; $p < 0.001$) and lower LV ejection fraction ($34\% \pm 8\%$ vs. $38 \pm 9\%$; $p = 0.019$).

Conclusion Additional mid-wall LGE is present in a small proportion (6%) of patients with ICM and is a marker of worse LV dilatation and systolic function. The clinical meaning of this finding (i.e. dual pathology vs. epiphenomenon of severe LV dilatation and wall stress) and its implication with respect to outcome remains to be determined.

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24 ADENOSINE STRESS NATIVE T1 MAPPING DEMONSTRATES IMPAIRED MYOCARDIAL PERFUSION RESERVE IN NON-ISCHEMIC DILATED CARDIOMYOPATHY

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Background Impaired myocardial perfusion reserve has been demonstrated in non-ischemic dilated cardiomyopathy (NIDCM) by positron emission tomography (PET) and adenosine-stress first-pass perfusion cardiac magnetic resonance (CMR) imaging. Adenosine stress native T1 mapping is a novel CMR technique able to assess myocardial perfusion without the use of contrast agents. Aim of the present study was to determine the clinical utility of this novel CMR technique in NIDCM.

Methods A total of 20 consecutive patients (mean age 61 ± 12 years, 80% males) with diagnosis of NIDCM who consented to be enrolled in the UHSM CMR registry were included in the present study. CMR at 3T including 1. cine imaging for the assessment of LV volumes, mass and global longitudinal strain (GLS) by tissue-tracking imaging; 2. rest and stress (adenosine 140 mcg/kg/min) MOLLI T1 mapping of mid

Abstract 23 Figure 1 Bland Altman Plot comparing phase contrast MRI measured PWV and cfPWV measured by oscillometry. Parametres used: Mean of difference = -0.1623, SD ± 4.70760 ; LoA: lower limit= -9.389196, upper limit = 9.064596