independent predictor of impact. Moreover, the diagnostic value as well as the clinical impact of CMR was highest when performed early.

Abstract 4 Table 1 Predictors of clinical impact – univariate and multivariate logistic regression analysis

Variables	Univariate analysis				Multivariate analysis			
	Sig.	OR	95% CI		Sig.	OR	95% CI	
			Lower	Upper			Lower	Upper
Age	0.008	1.024	1.006	1.041	0.002	1.035	1.013	1.058
Sex	0.77	1.091	0.609	1.954	0.604	0.831	0.413	1.673
Troponin	0.209	1	1	1.001	0.474	1	1	1.001
STEMI	0.224	1.63	0.742	3.577	0.966	0.981	0.412	2.338
iEDV	0.291	1.006	0.995	1.017	0.316	1.006	0.994	1.019
LVEF	0.597	0.995	0.975	1.015	0.847	1.002	0.98	1.025
RWMA	0.121	1.616	0.881	2.966	0.959	1.02	0.475	2.192
Oedema	0.078	1.765	0.938	3.323	0.527	1.298	0.579	2.912
LGE	0.004	2.393	1.318	4.345	0.017	2.411	1.17	4.968

5 STRUCTURAL PREDICTORS OF ATRIAL FIBRILLATION IN HYPERTROPHIC CARDIOMYOPATHY USING CARDIAC MAGNETIC RESONANCE IMAGING

^{1,2,*}A Ghosh Dastidar, ¹E De Garate, ²P Singhal, ^{1,3}J Rodrigues, ¹A Baritussio, ¹A Scatteia, ^{1,2}A Nightingale, ^{1,2}AG Stuart, ^{1,2}C Bucciarelli-Ducci. ¹NIHR Bristol Cardiovascular Biomedical Research Unit, Bristol Heart Institute, UK; ²School of Clinical Sciences, Faculty of Health Sciences, University of Bristol, UK; ³School of Physiology, Pharmacology and Neuroscience, Faculty of Biomedical Sciences, University of Bristol University of Bristol, UK

10.1136/heartjnl-2016-309668.5

Background Atrial fibrillation (AF) is the most common sustained arrhythmia in hypertrophic cardiomyopathy (HCM) and is associated with major adverse cardiovascular events. Cardiac magnetic resonance (CMR) with its superior tissue characterisation property is currently the imaging modality of choice for HCM.

Aims To identify the structural predictors of AF in HCM using CMR.

Methods 114 consecutive HCM patients were identified after reviewing approximately 3,100 CMR scans from our registry (Jan 2014 to Mar 2015). Comprehensive CMR protocol was used including cines, early and late gadolinium enhancement imaging. The diagnosis of HCM was based on left ventricular (LV) maximum wall thickness ≥ 15 mm (or 13–14 mm in the presence of familial history and/or ECG changes), in the absence of other cardiac/systemic disorders producing a similar degree of hypertrophy. Clinical notes were evaluated to identify a documented episode of AF. Univariate and multivariate logistic regression analyses were performed to determine the CMR imaging predictors of AF in HCM.

Results The final study sample consisted of 104 patients with HCM with median age 60years (IQR = 54–70) and 70% male, (10 patients excluded due to uncertain/overlapping diagnosis). 70% had non-apical HCM; the remainder 30% apical HCM. 16% (n = 17) had a documented episode of atrial fibrillation. The univariate predictors of AF included left atrial volume and the ratio of left atrial volume to LV end systolic volume whereas in the multivariate model the ratio of left atrial volume to LV end systolic volume remained the only significant predictor (p = 0.034, OR = 2.236, CI = 1.06–4.70) (Table 1).

Conclusion Our study suggests that the ratio of left atrial volume to LV end systolic volume is the best predictor of AF in HCM. The simple CMR derived ratio may have potential role for AF risk stratification in HCM.

Abstract 5 Table 1 Predictors of AF in HCM

	Sig.	OR	95% C. I.		Sig.	OR	95% C. I.	
			Lower	Upper			Lower	Upper
Age	0.061	1.048	0.998	1.1	0.4	1.025	0.968	1.085
LA volume	0.001	1.043	1.022	1.064	0.056	0.905	0.817	1.003
LVEF	0.318	0.976	0.93	1.024	0.068	1.025	0.998	1.052
LA/ESV	0.001	1.81	1.286	2.547	0.034	2.236	1.063	4.702
LGE	0.349	2.743	0.332	22.648	0.855	1.298	0.079	21.262
Apical HCM	0.955	1.033	0.33	3.237	0.571	0.589	0.094	3.677
Max. Thickness	0.881	1.009	0.893	1.141	0.902	1.015	0.797	1.293
LV mass	0.229	0.988	0.968	1.008	0.417	0.986	0.952	1.02

Variable(s) entered on step 1: Age, Left atrium volume, LVEF- left ventricular ejection fraction, LA/ESV- left atrial volume/left ventricular end systolic volume, LGE- late gadolinium enhancement, Apical HCM, Max. Thickness.

6 PREVALENCE AND CMR CHARACTERISTICS OF APICAL HCM

*E De Garate, A Ghosh Dastidar, P Singhal, G Venuti, A Amadu, A Baritussio, A Scatteia, C Lawton, J Rodrigues, C Bucciarelli-Ducci. *NIHR Cardiovascular Biomedical Research Unit, Bristol Heart Institute, Bristol, UK*

10.1136/heartjnl-2016-309668.6

Hypertrophic cardiomyopathy (HCM) is the most common cause of sudden cardiac death in young adults. The 3 main phenotypes are asymmetric (most common), concentric and apical. Literature suggests apical HCM is rare and more benign, but data is scarce. We sought to describe prevalence and characteristics of apical HCM in a large CMR service.

Methods We reviewed 3,100 scans (Jan 2014–Mar 2015). Protocol included cines, early and late gadolinium enhancement imaging. 114 consecutive HCM patients were identified. Asymmetric HCM defined as septal/ free wall thickness ratio > 1.3 ; apical HCM as apical wall thickness > 15 mm or apical/basal wall thicknesses ≥ 1.3 –1.5. Concentric HCM defined as symmetrical hypertrophy of ventricular wall without regional preferences. Non-apical HCM (asymmetric and concentric phenotypes) were compared with apical HCM. Fisher's exact t-test and unpaired t-test were performed for statistical significance (P-value < 0.05 , statistically significant). Univariate and multivariate logistic regression analyses were performed to determine CMR predictors of apical HCM.

Results 10 patients were excluded, leaving 104 patients, median age 60years; 70% male. 70% had non-apical HCM (5 patients concentric HCM, the rest asymmetric HCM) and 30% apical HCM. Mean maximum LV wall thickness, indexed LV mass, stroke volume, prevalence of LVOTO and SAM were greater in non-apical group. Presence of LGE was high in both groups ($> 85\%$) and wasn't statistically different. Univariate predictors of apical HCM included maximum LV wall thickness, indexed stroke volume, LVOT obstruction (Table 1). In the multivariate model, maximum wall thickness remained the only significant predictor.