Abstract 3 Figure 1

(a composite of cardiovascular death, stroke, heart failure admission, infective endocarditis) was significantly higher in the DM-AS group than the isolated-AS group (Hazard Ratio: 3.35; 95% confidence interval: 0.97–11.6; p=0.02).

Conclusion We compared clinical outcomes in severe AS patients with and without T2DM and investigated myocardial recovery in energetics, perfusion and contractile function after AVR. Diabetes was associated with increased morbidity and mortality after AVR. We showed here for the first time that the collective impact of T2DM and AS on the myocardium aggravates energetic impairment, coronary microvascular dysfunction and myocardial contractile dysfunction. While myocardial recovery following AVR was associated with similar improvements in perfusion and contractile function in severe AS patients with and without T2DM, post AVR improvements in energetics were only detected in isolated AS patients. However, despite the significant improvements in contractile function and perfusion after AVR in diabetes patients, these parameters remained lower in the group with diabetes comorbidity compared to isolated AS patients.

Conflict of Interest None

4 BRAIN PHENOTYPE OF TAKOTSUBO SYNDROME

Introduction Takotsubo syndrome presents like an acute myocardial infarction but typically occurs in the aftermath of psychological stress, affecting predominantly women. Here we present a complete grey and white matter structural

tractography and functional connectivity assessment in takotsubo syndrome patients and matched controls.

Methods Twenty-five acute (< 5 days from onset) takotsubo syndrome patients and 25 age, gender and comorbidity matched controls were recruited in an observational cross-sectional study. All patients and controls were scanned on the same magnetic resonance imaging (MRI) scanner at the same imaging centre and data were analysed with the same software versions. Surface-based morphometry was carried out on brain MRI scans to extract the cortical morphology based on volume, thickness, and surface area of all brain regions using Freesurfer. White matter hyperintensities were determined by using the lesion segmentation tool. Cortical morphology was compared between the two groups using a general linear model in SPSS corrected for age, gender, photoperiod and total brain volume. Resting state functional MRI was analysed using the functional connectivity (CONN) toolbox by comparing the two groups. Diffusion tensor tractography images were pre-processed using the Functional Magnetic Resonance Imaging of the Brain (FMRIB’s) Diffusion Toolbox and analysed using CONN toolbox. Significance was set at <0.05.

Results Total white matter and subcortical grey matter volume were smaller in takotsubo (p<0.001 both), driven by a reduction in brain surface area (p<0.001). All individual grey matter regions were smaller in takotsubo (hippocampus (p<0.001), para-hippocampus (p<0.001), amygdala (p=0.002), brainstem (p<0.001) and others) except for the thalamus and insula which were larger (p<0.001 both) compared to controls, in either hemisphere or combined. There was no difference in white matter hyperintensities between takotsubo syndrome patients and controls (p=0.3). Significant, numerous hyper- and hypo-functional connectivities were seen compared to matched controls (i.e. thalamus to left insula, temporal lobes, amygdala, cingulate gyrus, all p<0.05). All structural tractography connections were increased in takotsubo syndrome compared to controls (p<0.05).

Conclusion We show smaller grey and white matter volumes driven by reduced cortical surface area and increased cortical thickness, no difference in white matter hyperintensities, enhanced structural tractography connections with distinct changes in functional connectivity linked to emotion, mood, language, visual and auditory perception as well as autonomic control. Interventions aimed at attenuating these findings could be important in the rehabilitation of takotsubo patients.

Conflict of Interest None

5 IMPROVING THE DIAGNOSTIC ACCURACY OF APICAL HYPERTROPHIC CARDIOMYOPATHY

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Introduction The diagnosis of apical hypertrophic cardiomyopathy (ApHCM) is contingent on demonstrating apical maximum wall thickness (MWT) of ≥15 mm; the same threshold as other HCM subtypes. However, the myocardium naturally tapers towards the apex in healthy individuals, so ≥15 mm MWT is proportionately higher in the apex than in naturally
thicker basal segments. Using cardiac magnetic resonance (CMR), relative ApHCM has been described (typical ECG features, loss of apical tapering, cavity obliteration but hypertrophy <15 mm). Wall thickness measurement using machine learning now exceeds human performance. We aimed to redefine the optimal diagnostic threshold for ApHCM using segment-specific criteria based on a large cohort of healthy control subjects.

**Methods** Segmental wall thickness was measured using healthy subjects from the UK Biobank using a clinically validated machine learning algorithm. A normative reference range was established for all 16 segments, conditioned to body surface area (BSA), sex and age. Derived segment-specific wall thickness thresholds were used to define optimal disease thresholds for patients clinically managed with overt (MWT >15 mm) and relative ApHCM (MWT <15 mm, but typical ECG and imaging findings).

**Results** 4118 UK biobank subjects were used to define normal segmental thicknesses and reference ranges. These were applied to ApHCM (73 overt, 31 relative). There were no apical wall thickness age related differences. The upper limit of the 95% confidence interval corresponded to a combined maximum apical MWT for both males and females of 10.4 mm using non-indexed measurement, or 5.6 mm/m² when indexed to BSA. Non-indexed segmental threshold identified 100% of ApHCM patients (true positives), 81% (25 of 31) relative ApHCM and 3% (115 of 4118) of healthy UK biobank subjects (false positives). Indexed segmental thresholds improved the diagnostic potential in relative ApHCM without an increase in false positives (100% of ApHCM patients, 84% (26 of 31) of relative ApHCM patients, and 3% healthy UK biobank (127 of 4118).

**Conclusion** We propose new diagnostic criteria for ApHCM using segmental indexed apical wall thickness of >5.6 mm/m² to better identify inappropriate apical hypertrophy in those whose wall thickness does not meet current criteria for diagnosis.

**Conflict of Interest** nil

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**SILENT CEREBRAL MICROINFARCTION FOLLOWING MITRAL VALVE SURGERY**

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**Introduction** Mitral regurgitation (MR) is the second most common valvular pathology worldwide. When untreated, severe MR is associated with significant morbidity and mortality. Mitral valve surgery is recommended in symptomatic patients and those with evidence of adverse left atrial or left ventricular remodelling. Although uncommon, stroke is a recognised complication of mitral valve surgery and is associated with unfavourable outcomes. While silent cerebral microinfarction has been described following cardiac surgery, its incidence in mitral valve surgery and its impact on quality of life is presently unknown. The main aim of this study was to assess the incidence of perioperative cerebral microinfarction following mitral valve surgery and its impact on medium-term health-related quality of life (HRQoL). Methods Cerebral diffusion-weighted magnetic resonance imaging (DWI-MRI) was conducted pre-operatively and prior to discharge in 31 patients undergoing mitral valve surgery for mitral regurgitation. Blinded analysis was conducted by a neuro-radiologist. HRQoL assessment was undertaken at baseline and at a 6-month follow up with EuroQol-5 dimensions (EQ-5D-5L) and Hospital Anxiety and Depression Scale (HADS) questionnaires.

**Results** Thirty-one patients underwent paired cerebral DWI-MRI (mitral valve replacement (MVR) n=16 [52%] and mitral valve repair (MVR) n=15 [48%]). Prevalence of atrial fibrillation was similar in both groups (MVR n=9 [56%] vs. MVR n=7 [47%], p=0.59). Peri-operative cerebral microinfarction occurred in 9 patients (29%). Embolic events were numerically higher in the MVR group versus MVR group, but not statistically significant (n=7 [44%] vs. n=2 [13%], p=0.06). Presence of multiple lesions, large lesions >5 mm, small lesions <5 mm and the total number of lesions did not differ significantly between the two groups. Median volume of lesions was 

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**Abstract 6 Figure 1**

**Abstract 6 Figure 2**