Conclusion Adenosine-induced increase in MBF measured using perfusion mapping is accurate for the confirmation of hyperaemia during stress CMR studies and is superior to traditional, clinically used markers of adequate stress.

19 MYOCARDIAL PERFUSION MAPPING IN CARDIAC AMYLOIDOSIS- UNEARTHING THE SPECTRUM FROM INFILTRATION TO ISCHAEMIA

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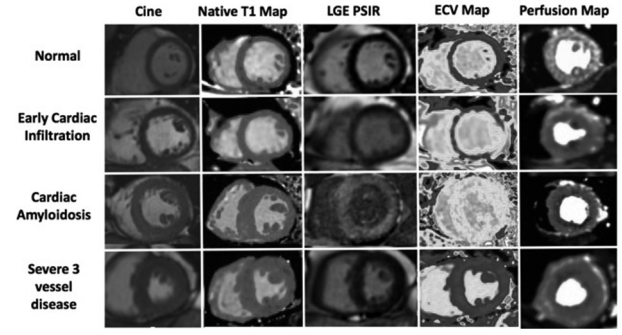
Background Cardiac involvement is the main driver of outcome in systemic amyloidosis, but the relationship between amyloid deposits and outcomes is not well understood. The simple explanation of physical, mechanical replacement of the interstitium by amyloid seems insufficient. Preliminary studies support the hypothesis that myocardial ischaemia could contribute to cell damage.

Purpose (1) To assess myocardial ischaemia in cardiac amyloidosis. (2) To compare patients with cardiac amyloidosis to patients assessed on invasive coronary angiography (ICA) to have normal coronary physiology (NCP), microvascular dysfunction (MVD) and triple vessel coronary disease (3VD). (3) To assess correlation of perfusion mapping to markers of disease severity and prognosis.

Methods 86 patients and 20 healthy volunteers (HV) underwent CMR at 1.5T (Siemens) with standard cine, PSIR-LGE, T1,T2,Extracellular Volume (ECV) mapping and adenosine stress with myocardial blood flow (MBF) mapping. Thirty-eight patients also underwent ICA with 3 vessel assessment of Index of Microcirculatory Resistance and Fractional Flow Reserve: 7 had cardiac amyloidosis, 8 had NCP, 15 had MVD and 8 had 3VD.

Results Cardiac amyloidosis patients had severe reduction in stress MBF and myocardial perfusion reserve (MPR) (1.22 ml/g/min±0.70 and 1.62±0.63) compared to HV (3.21 ml/g/min ±0.64,p<0.001 and 4.17±0.78, p<0.001), NCP (2.66±0.56, p<0.001 and 2.51±0.43, p=0.036) and MVD (2.10±0.31, p<0.001 and 2.29±0.87, p=0.014) with the degree of reduction being similar only to patients with 3VD (1.44±0.54, p=1.000 and 1.64±0.68,p=1.000) (figure 1). Rest MBF was also lower in amyloidosis than HV. Cardiac amyloidosis stress MBF and MPR inversely correlated with amyloid burden (ECV, r=−0.715, p<0.001, transmurality of LGE, p<0.01), systolic dysfunction (EF, r=0.405, p<0.01), and blood biomarkers (NT-proBNP (r=−0.678, p<0.001) and Troponin T (r=−0.628, p<0.001)). There was a correlation between stress MBF and native T1 (r=−0.588, p<0.001) but not T2 (p=0.591). Stress MBF and MPR were early disease markers, being elevated in patients with early cardiac amyloid infiltration (raised ECV, no LGE, p<0.01 vsHV).

Conclusion Myocardial ischaemia is common in cardiac amyloidosis – with stress MBF and MPR similar to that of patients with 3VD. The reduction correlates with the degree of amyloid infiltration and markers of adverse prognosis, highlighting the potential role of myocardial ischaemia as a key mechanism in the pathophysiology of cardiac amyloidosis.



Abstract 19 Figure 1 Short axis cine SSFP images in end-diastole, corresponding native T1 mapping, late gadolinium enhancement (LGE) images, ECV Mapping and stress myocardial blood flow mapping in a normal subject, a patient with early cardiac infiltration (raised ECV, no LGE), a patient with cardiac amyloidosis, a patient with severe three vessel coronary disease

20 ENDOTHELIAL LOSS AS A CAUSE OF IMPAIRED MYOCARDIAL PERFUSION RESERVE ON CMR IN SEVERE AORTIC STENOSIS

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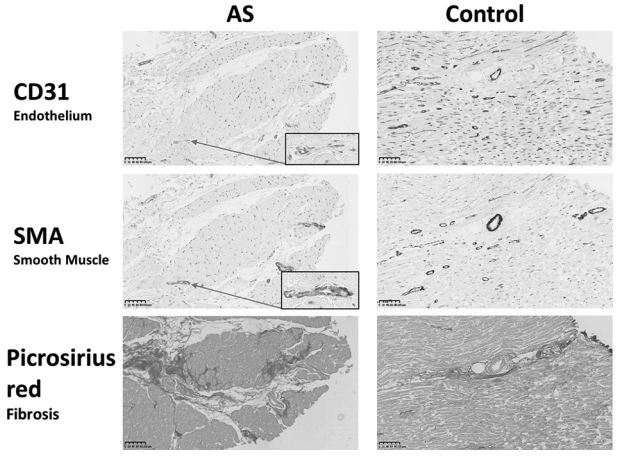
Background Impaired myocardial perfusion reserve occurs in pressure overload hypertrophy such as in severe aortic stenosis (AS) despite unobstructed epicardial coronaries. However the pathological mechanisms underlying this are poorly understood. We sought to assess myocardial perfusion reserve in severe AS by stress perfusion cardiovascular magnetic resonance (CMR), and examine the findings in relation to the histological evidence of vascular changes in the myocardium.

Methods Fourteen patients with severe AS and unobstructed epicardial coronaries underwent adenosine stress perfusion CMR before and 6 months after surgical aortic valve replacement (AVR). Myocardial biopsies were obtained during AVR and stained using CD31 +for endothelium, smooth muscle actin (SMA) for smooth muscle, and picrosirius red for



Abstract 20 Figure 1

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) $\leq 0.8cm^2$ had greater endothelial loss compared to those with an AVA >0.8 and$\leq 1.0cm^2$ (1.34% ±0.44% vs 2.84±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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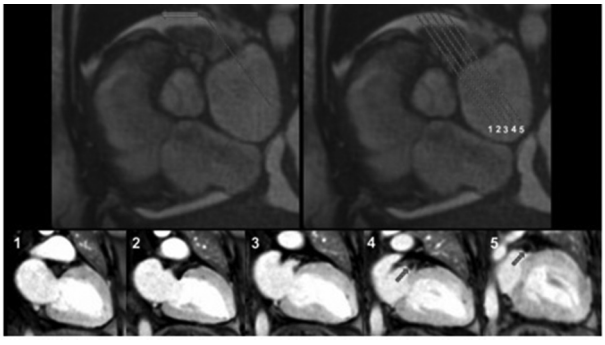
Background Cardiac amyloidosis (CA) has been associated with a high prevalence of intracardiac thrombi, but this was reported in small cohorts of high risk patients (with a clinical indication for transoesophageal echocardiography). It is therefore not known whether such observations are applicable to 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 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.



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

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 arrythmia 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 ($\geq$3 beats,$\geq$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%±4.1% *v* 17.0%