MYOCARDIAL PERFUSION, STRAIN AND PRE-CONTRAST T1 VALUES IN MODERATE ASYMPTOMATIC AORTIC STENOSIS

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Introduction In severe aortic stenosis (AS) myocardial perfusion and myocardial strain (in particular longitudinal strain-LS) are reduced. Reduced myocardial perfusion in severe AS is thought to occur in the subendocardium due to increased LV pressure, leading to reduced LS and increased fibrosis. Our group previously showed a good correlation between pre-contrast T1 values using SHMOLLI (Shortened Modified Look-Locker Inversion recovery) sequence and histological quantification of diffuse fibrosis in severe AS. We hypothesised that impaired myocardial perfusion in patients with moderate AS would relate to impaired LS and increased pre-contrast T1 values (reflecting more diffuse fibrosis).

Methods 31 patients (8 female) with asymptomatic moderate AS (by echo criteria) and normal ejection fraction were recruited. All subjects underwent CMR scanning at 1.5T for pre-contrast T1-mapping using the ShMOLLI sequence, stress and rest perfusion imaging, tagging and valve assessment. Average T1 values were analysed on a per-case basis. Perfusion scans were analysed to determine the myocardial perfusion reserve index (MPRI) and tagging to determine strain.

Results All patients (average age 67±12 years) underwent CMR scanning at 1.5T. The average MPRI was 1.3±0.4. Average T1 values were 956±32 ms. There was a significant correlation between MPRI and LS (<0.05, r=−0.4, figure 1) but not circumferential strain (CS). Average LS was −11.1±2%, average CS was −17%±5%. There was also a significant correlation of MPRI with aortic valve area (as measured by CMR planimetry mean (1.7 ±0.4 cm²), p<0.05 r=0.4). There was no significant correlation between MPRI and pre-contrast T1 values. Pre-contrast T1 values correlated strongly with LV mass (mean 147±40 g) (r=0.4 p<0.01) and peak aortic velocity (mean 3.1±0.4 m/s) (p<0.0001 r=0.5).

Conclusions In moderate AS, there is a significant correlation between reduced MPRI and impaired longitudinal but not circumferential strain. This supports the hypothesis that impairment of myocardial perfusion predominantly affects the subendocardial longitudinal myocardial fibres (rather than the mid-wall circumferential fibres) in moderate AS. The exact relationship of perfusion and diffuse fibrosis in moderate AS is yet to be fully established. In this study pre-contrast T1 values (as a surrogate marker for diffuse fibrosis) were not affected by changes in myocardial perfusion.