Abstract: P6484

Invasive and non-invasive characterisation of low gradient aortic stenosis

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Topic(s):
Valvular Heart Disease – Pathophysiology and Mechanisms

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Background: Low gradient severe aortic stenosis (LGAS) is associated with unfavourable outcomes when compared to high gradient aortic stenosis (HGAS), yet the contributing pathophysiology is poorly understood.

Methods: Symptomatic LGAS and HGAS patients undergoing trans-catheter aortic valve implantation (TAVI) underwent 3T stress perfusion cardiac magnetic resonance imaging (CMR) pre-(within 24 hours) and post-(4-6 months) TAVI. Left ventricular (LV) contractility and coronary flow/pressure were measured during hyperaemia and rapid pacing, immediately before and after TAVI, using a conductance LV catheter and dual-pressure and Doppler sensor--tipped guidewire in the mid-left anterior descending coronary artery.

Results: 24 patients were recruited resulting in 19 suitable datasets (LGAS N=9, HGAS N=10, equally matched for comorbidities and B-natriuretic peptide level). LGAS patients had a smaller LV end diastolic volume index (p=0.035) and lower LV mass index (LVMI) (p=0.037). Pre-TAVI stress global endocardium-epicardium gradient was 0.88±0.09 and global myocardial perfusion reserve (MPR) 2.0±0.48 in 14 patients (6 LGAS and 8 HGAS patients, no difference between groups). Pre-TAVI, baseline coronary data demonstrated lower augmentation pressure (AP, p=0.035) and augmentation index (Alx, p=0.02) in the LGAS group. LGAS patients also exhibited a shorter ejection time (p=0.015), larger forward compression waves during rest, hyperaemia and rapid pacing, and smaller backward expansion waves (BEW) (p=0.001). Lower baseline end systolic pressure (p=0.004), inotropy (dp/dt+, p=0.045), lusitropy (dp/dt-, p=0.069), and stroke work (p=0.019) were observed in the LGAS group. Whilst LV size was smaller the LGAS group, rapid pacing induced a more significant drop in end systolic volume (p=0.045) and ejection fraction (p=0.015) in patients with HGAS. Post-TAVI, the hyperaemic BEW fell sharply (p<0.001), along with coronary VT (p=0.02), and average pulse velocity (p=0.028), and AP and Alx remained lower (p=0.034 and p=0.031, respectively). The forward expansion wave was reduced in LGAS during rapid pacing. The HGAS group displayed a more profound drop in dp/dt+ (p=0.011) and dp/dt- p=0.014) at rest following intervention. Repeat CMR demonstrated statistically significant reduction in LV size and LVMI (p=0.012 and p=0.001, respectively) with significant increase in 3D global peak radial, circumferential and longitudinal strain (p=0.004, p=0.001 and p=0.018, respectively). Post-TAVI stress global endocardium-epicardium gradient was 0.88±0.13 and MPR 2.46±0.59 (improved from pre-TAVI, p=0.05). There was no difference in remodelling patterns or perfusion between the two groups.

Conclusion: This is the first study detailing the combined invasive and CMR pathophysiological changes in LGAS. Despite invasive parameters indicating a disease of less severe AS, the level of perfusion abnormality is disproportionate which may in part, relate to their adverse prognosis.
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