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Meeting abstract

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2047 Further benefits of aortic valve replacement for severe aortic stenosis; impact on IV torsion as assessed by cardiac MRI

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Background

Compensatory LVH in severe aortic stenosis (AS) alters the manner in which LV fiber architecture interacts. Prior to end-stage AS, the LV has supranormal contractile performance, chiefly represented by compensatory augmentation in torsion. In part this is thought to be due to reliance on systolic rotatory properties of the LV to effect ejection of blood, not circumferential or radial strain, *per se*.

Hypothesis

We hypothesize that 1) as LVH regresses following AVR, torsion will appropriately decrease 2) CAD state may influence torsion.

Methods

Twenty-nine (29) patients (68 ± 12 yrs, 14 F) with late but not decompensated AS underwent cardiac MRI (CMR) for RF tissue tagging (1.5 T-GE-EXCITE, Milwaukee, WI) and 3D LV torsion analysis via 3D HARP (Diagnosoft, Palo Alto, CA) pre and post AVR, at early (6 ± 1 mo) and up to late (4 years ± 5 month) time points.

Results

Twenty seven patients who survived and/or returned had post-AVR torsion, rotation and LV functional data assessed by CMR. LV mass index decreased post AVR by 27% (90 ± 31 vs 69 ± 16 g/m2, $p < 0.05$). At the level of the LV cavity a trend to decreasing LVEF was seen (57 ± 17 vs 65 ± 13%, $p = 0.06$). Peak torsion (apex) decreased 25% post AVR (16 ± 7 to 12 ± 4%, $p = 0.1$). The 3D rotation displacement, torsion work (total LV torsion integrated over systolic ejection time) regressed (54 ± 30 to 35 ± 18 deg, $p < 0.01$) while average torsional displacement decreased 30% (5.9 ± 2.9 to 4.1 ± 2.0 deg, $p < 0.05$). Torsion at half-maximum time (end-systolic time) also decreased by 31% (2.9 ± 1.5 to 2.0 ± 1.0 deg, $p < 0.05$). Interestingly, in those in whom no concurrent CABG was performed (i.e.; no CAD) representing 8 patients, the torsional indices all appropriately decreased following AVR. Likewise, if CABG was performed (23 patients), there was no significant change in torsional indices early or late after AVR and this was matched by a blunting of EF improvement.

Conclusion

In aortic stenosis following AVR, increased LV torsion regresses in parallel with LV mass regression representing, not abnormal function, but a return towards a normal uncompensated state. As a population, torsion normalized. However, mitigating against torsion reduction, the presence of CAD markedly attenuated both the decline in LVH, as well as the normalization of LV torsion.

One of the important myocardial compensatory mechanisms for severe aortic stenosis is augmented LV torsion.

Following aortic valve replacement, torsion indices decline. Does this represent a return to a more normal non-compensated physiology or an unreversible insult to the LV?