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Meeting abstract

2045 Dichotomization of lvh regression and diastolic function in severe pressure overload hypertrophy

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Background

Elegant invasive human and animal studies from the University of Zurich and the Medical University of South Carolina have shown that in subjects with pressure overload due to severe aortic stenosis (AS), following aortic valve replacement (AVR), while myocyte regression is relatively rapid, the interstitial collagen content regression lags.

Hypothesis

This manifests as a blunted improvement in diastolic function as assessed by cardiac MRI when related to LVH regression over time after AVR for AS.

Methods

Ten patients with severe, but compensated AS underwent serial baseline 3D cardiac MRI (CMR) with 1.5 T EXCITE (GE, Milwaukee, WI). Standard 3D volumetrics were measured. LV diastolic function was assessed by a single phase velocity mapping slice placed at the tips of the mitral valve leaflets, acquired in a through-plane manner with temporal resolution of 20–25 ms. Interrogation of resultant time-velocity curves was performed to resolve: 1) E:A ratio as mean and peak absolute and relative velocities 2) decceleration time (DT). Patients were followed out to a maximum of 4 years after AVR. A paired test was used for normally distributed metrics with a Wilcoxan signed rank score used for non-normally distributed data (E:A ratios).

Results

All patients survived AVR and were available for serial follow-up over time periods (mean 3 ± 1) out to 4 years. E:A ratios and/or morphology almost always uniformly improved (9/10 patients; 90%) from 0.9:1 to 1.7:1 ($p < 0.005$) accounting for 4 patients improving one complete diastolic function stage. This was moderately well correlated with the LV mass index regression ($r = 0.49$, $p = 0.15$). Deceleration time also improved (192 vs 233 ms, $p < 0.005$). While EF improved (55 ± 22 to 65 ± 11%, $p < 0.05$) as did LV geometry (1.07 ± 0.2 to 0.94 ± 0.24 g/m^2, $p < 0.05$), neither were tightly correlated with improvements in diastolic function (p = NS). Furthermore, using predictive modeling from historic data, the improvement seen in diastolic function as related to improvements in LV mass regression was incomplete and tardy.

Conclusion

Following AVR for severe AS, as expected marked improvements in LV mass regression occur. However, despite modest and clinically important improvements in diastolic function it is incomplete and tardy as related to the measured regression of LVH. Thus, it is likely that the interstium, failing to be as heavily modulated by relief of pressure overload as are the myocytes, contributes to a paradoxical *increase* in diastolic stiffness. This interplay between collagen content and myocyte results in a *less* then expected improvement in myocardial diastolic properties following afterload relief, contributing to residual dyspnea.

Despite marked improvement in LVH regression in patients with severe aortic stenosis and improvements in diastolic function, all measured by CMR, LVH changes outstrip diastolic functional changes. The reason for this paradox is considered explained at the collagen level.