Rapid Fire Abstracts

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Adaptive myocardial mechanics in aortic stenosis patients

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Introduction: Left ventricular (LV) hypertrophy in aortic stenosis (AS) becomes maladaptive over time, leading first to a reduction in global longitudinal strain (GLS) and in a later stage a reduction in ejection fraction (EF). The myocardial state of impaired GLS but preserved EF is a key remodeling turning point in AS, yet little is known about the coping mechanics of the LV at or around this sensitive juncture.

Aim: 1) To study the relationship between LV mass index (LVMi) increase and measures of LV function, including strain in AS; 2) To investigate whether augmentation of global myocardial radial and circumferential strain (GRS, GCS) compensates for the GLS reduction in AS patients with preserved EF.

Methods: One-hundred and eleven patients with varying degrees of AS, and 20 age- and gender-matched healthy volunteers were prospectively enrolled. transthoracic echocardiography with offline strain analysis was performed using TomTec software. Intra- and inter-observer variability of linear LV internal dimensions/thickness, EF and strain indices was tested on 20 randomly selected patients.

Results: Clinical and demographic characteristics of cases and controls are shown in Figure 1. GLS was impaired in AS patients compared to controls. In AS with preserved EF (>50%), as LVMi increased, GLS progressively improved up to a point, beyond which any further increase in LVMi appeared counter-productive with impairment of GLS (Figure 1). EF preservation in these AS patients was mediated by a compensatory supernormal augmentation of GRS and a smaller augmentation of GCS (Figure 1). We observed a significant inverse correlation between GRS and GLS (r = 0.3, p = 0.002), and a similar trend between GCS and GLS (r = 0.275, p = 0.004). Intraclass correlation coefficient was high for all measurements (0.7-0.9).

Conclusion: In patients with AS and preserved EF, progressive myocardial hypertrophy improves GLS up to a point beyond which GLS drops and GRS increase to compensate. This plasticity of myocardial mechanics, in particular the supranormal augmentation of GRS is what enables the pathologically hypertrophied AS ventricle to delay the otherwise inexorable decline in its global systolic function.

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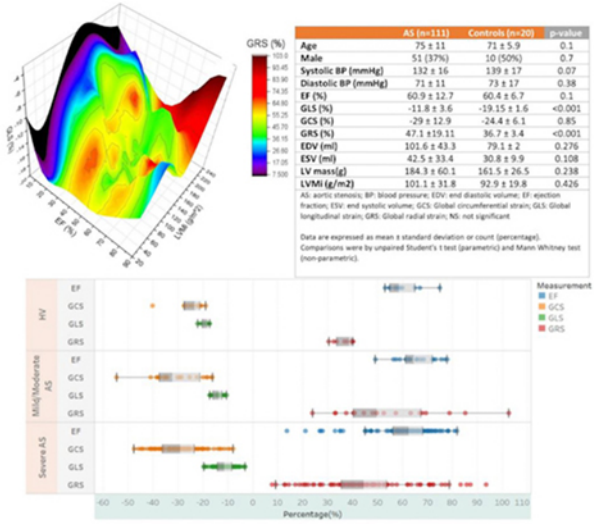

	AS (n=111)	Controls (n=20)	p-value
Age	75 ± 11	71 ± 5.9	0.1
Male	51 (37%)	10 (50%)	0.7
Systolic BP (mmHg)	132 ± 16	139 ± 17	0.07
Diastolic BP (mmHg)	71 ± 11	73 ± 17	0.38
EF (%)	60.9 ± 12.7	60.4 ± 6.7	0.1
GLS (%)	-11.8 ± 3.6	-19.15 ± 1.6	<0.001
GCS (%)	-29 ± 12.9	-24.4 ± 6.1	0.85
GRS (%)	47.1 ± 19.11	36.7 ± 3.4	<0.001
EDV (ml)	101.6 ± 43.3	79.1 ± 2	0.276
ESV (ml)	42.5 ± 33.4	30.8 ± 9.9	0.108
LV mass(g)	184.3 ± 60.1	161.5 ± 26.5	0.238
LVMi (g/m2)	101.1 ± 31.8	92.9 ± 19.8	0.426

AS: aortic stenosis; BP: blood pressure; EDV: end diastolic volume; EF: ejection fraction; ESV: end systolic volume; GCS: Global circumferential strain; GLS: Global longitudinal strain; GRS: Global radial strain; NS: not significant

Data are expressed as mean ± standard deviation or count (percentage).
Comparisons were by unpaired Student's t test (parametric) and Mann Whitney test (non-parametric).