

Prognostic role of global work index in asymptomatic patients with aortic stenosis

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Background: In asymptomatic patients with aortic stenosis (AS), the optimal timing for intervention is still challenging. Previous studies demonstrated that advanced stages of cardiac damage are associated with excess mortality. The role of myocardial work to identify cardiac dysfunction in AS and to predict prognosis has not been investigated.

Purpose: We aimed to evaluate the modification of myocardial work indices related to AS stages and their prognostic value.

Methods: This study analysed clinical, echocardiographic and outcome data of 170 patients with asymptomatic AS (aortic valve area ≤ 1.5 cm²) and preserved ejection fraction (LVEF $\geq 50\%$). Exclusion criteria were: significant associated cardiac valve lesion, left bundle branch block and sub-optimal quality of speckle-tracking image analysis. The control group included 50 patients matched for age and sex. Global work index (GWI), global constructive work (GCW), global wasted work (GWW) and global work efficiency (GWE) were estimated by LV pressure-strain loops. In AS group, LV pressure was evaluated by adding trans-aortic mean gradient to systolic blood pressure. The following staging classification was used: no cardiac damage associated with the valve stenosis (Stage 0), left ventricular damage (Stage 1), left atrial or mitral valve damage (Stage 2), pulmonary hypertension or tricuspid valve damage (Stage 3), or right ventricular damage or subclinical heart failure (Stage 4).

Results: While global longitudinal strain was significantly lower in AS

than in control group (18.7 \pm 2.8 vs 20.7 \pm 2.1%, $p < 0.001$), increased values of GCW and GWI (respectively 2948 \pm 598 vs 2360 \pm 353 mmHg%, and 2528 \pm 521 vs 2005 \pm 302 mmHg%, $p < 0.001$) were observed in patients with AS. Besides, GWW was significantly increased in AS vs controls (139 \pm 90 vs 90 \pm 49 mmHg%, $p = 0.001$), with no changes in terms of GWE (95 \pm 4 vs 96 \pm 2%, $p = 0.110$). When patients were stratified according the stages of cardiac damage, MW indices didn't differ significantly, except for the GWI, which was significantly lower in Stage 3 to 4 compared to Stage 0 and Stage 2 (2268 \pm 469 vs 2623 \pm 503 vs 2610 \pm 503 mmHg% respectively, $p = 0.025$). During a mean follow up of 27 months (IQ range 12–48 mo), 18 patients had a CV death. The best GWI value associated with outcome was 1866 mmHg% (sensitivity 45%, specificity 96%, AUC = 0.701, $p = 0.01$). The presence of a GWI at baseline lower than 1866 mmHg% was associated with a higher rate of CV events at 4-year follow-up (57% vs 7%, log-rank $p < 0.001$). On multivariable Cox-regression analysis, BNP values ($P = 0.014$) and GWI < 1866 mmHg% ($P = 0.033$) emerged as independently associated with CV death.

Conclusion: In asymptomatic patients with AS, advanced stages of cardiac damage are characterized by reduced values of GWI, that are associated with increased mortality. Thus, the evaluation of MW indices may allow a better phenotyping of asymptomatic patients at higher risk of developing cardiovascular events during follow-up.