Prognosis and clinical outcomes after TAVI, regarding the extravalvular cardiac damage defined by echocardiography prior the procedural

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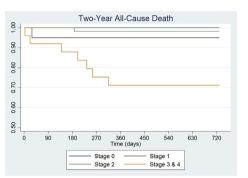
Background: Recent studies have shown that the extent of extravalvular (extra-aortic valve) cardiac damage in patients with severe aortic stenosis (AS) have important prognostic implications for clinical outcomes after aortic valve replacement (AVR).

Aims: The aim of the present study is to evaluate the prognostic impact of a defined staging classification ("Généreux Staging Classification") (GSC) characterizing the extent of extravalvular cardiac damage in patients with severe AS undergoing percutaneous transcatheter aortic valve implantation (TAVI).

Methods: A total of 102 consecutive patients, admitted in our institution between 2011–2017, with severe AS (echo-defined by peak aortic velocity, mean transvalvular gradient or aortic valve area) and symptoms related to AS (dyspnea, heart failure, angina or syncope) undergoing TAVI, were included. These patients were pooled and classified according to the presence or absence of cardiac damage as detected by echocardiography prior to TAVI, regarding the GSC: no extravalvular cardiac damage (Stage 0), left ventricular damage (Stage 1), left atrial or mitral valve damage (Stage 2), pulmonary vasculature or tricuspid valve damage (Stage 3), or right ventricular damage (Stage 4). Two-year outcomes were compared using Kaplan–Meier techniques and multivariable Cox proportional hazards models were used to identify 2-year predictors of mortality.

Results: Out of 102 patients, 57 were male (55.9%). Mean age was 83.46 ± 4.23 years. 2 patients (2.1%) were classified as Stage 0; 20 patients (20.3%) as Stage 1; 55 patients (54.2%) as Stage 2; 22 (21.6%) as Stage 3; and 3 patients (2.9%) as Stage 4. Two-year mortality was 0.0% in Stage 0, 5.0% in Stage 1, 5.5% in Stage 2, and 44.0% in Stages 3–4. After multivariable and univariate analysis, stage of cardiac damage was independently associated as predictor for all-cause mortality at 2-years, after TAVI (HR 2.8 [1.3±6.2], p<0.01). There were not another identificable predictors of 2-years death (age, sex, hypertension [78.5%] of total patients], dislipemia [64.7%], diabetes [30.3%], smoking [78.5%], O2-chronic obstructive pulmonary disease [27.5% of total patients], renal insufficiency [78.5%], previous coronary artery disease [37.3%], peak aortic velocity, mean transvalvular gradient, and aortic valve area).

Conclusions: Given the strong association demonstrated in this study between advanced staging of cardiac damage and worse clinical outcomes after TAVI in short-middle term survival, consideration of the GSC in patients with severe AS in future recommendations for risk stratification might be useful.



Two-year all-cause death in TAVI by GSC.