Effects of transcatheter aortic valve implantation on left ventricular mass and global longitudinal strain: tissue Doppler and strain evaluation

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ABSTRACT

Introduction: Transcatheter aortic valve implantation is the option of choice for high surgical risk patients suffering from symptomatic aortic stenosis. We aimed to evaluate the influence of baseline global longitudinal strain on left ventricular mass regression after the procedure.
Methods: We enrolled 23 patients with pure symptomatic severe aortic stenosis who underwent CoreValve prosthesis (Medtronic, Minneapolis, MN) implantation. Everyone had echocardiography registration before the procedure and after six months in order to analyze two-, three-, and four-chamber peak longitudinal strain and global longitudinal strain.
Results: After the procedure New York Heart Association class, peak and mean aortic valve gradients ($p < 0.001$ for all) improved. Interventricular septum and posterior wall thicknesses decreased ($p < 0.001$ for both). Moreover, left ventricular mass index for body surface area changed from 190 ± 44 to 143 ± 30 g/m^2, ($p < 0.001$). Finally, global longitudinal strain significantly increased (from 9.4 ± 0.9 to 11.5 ± 0.8 %; $p < 0.001$), as well as its components. Baseline global longitudinal strain correlated with left ventricular mass regression ($r = 0.560$; $p = 0.005$; 2-sided) and predicted it at linear regression analysis ($B = 23.707$; $p = 0.005$; adjusted $R^2 = 0.281$).
Conclusions: Global longitudinal strain and its components improved six months after the procedure. Moreover, baseline global longitudinal strain seemed to predict left ventricular mass regression in patients with pure aortic stenosis undergoing transcatheter aortic valve implantation. This finding could be related to the extent of myocardial fibrosis which is also responsible for lack of left ventricular mass regression and poorer prognosis.

Keywords: *aortic valve, echocardiography, transcatheter valve implantation.*

INTRODUCTION

Aortic valve stenosis is a quite frequent valvular disease which could require surgical treatment (1, 2). Aortic valve replacement by open chest surgery is still considered the gold standard for symptomatic patients,

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but recently transcatheter aortic valve implantation (TAVI) has become an option for patients at high surgical risk (3-8). For one third of these patients, not accepted for surgical treatment due to advanced age, comorbidities, and high surgical risk (1), medical evidence suggests that TAVI is feasible and provides a hemodynamic and clinical improvement up to two years, even if long-term durability is still debated (3). Aortic stenosis causes increased left ventricular (LV) afterload, which leads to LV

hypertrophy and remodeling towards heart failure, increased morbidity and mortality (9-12). Surgical treatment, on the one hand, has already been demonstrated to reduce LV mass and improve systolic and diastolic LV parameters, thus prolonging survival (13-15). TAVI, on the other, seems to improve diastolic function and to favor left ventricular mass regression (LVMR) after six months (16). The impact of the procedure on LV systolic parameters like peak longitudinal strain have not yet been extensively evaluated. The aim of the present study was to assess LVMR and peak longitudinal strain improvement six months after TAVI compared to pre-procedural assessments. Moreover, we wanted to investigate whether longitudinal strain parameters before the procedure could predict LVMR.

METHODS

Subjects. From January 2011 to August 2011 we consecutively enrolled 23 patients with pure symptomatic severe aortic stenosis and left ventricular ejection fraction > 45 % who underwent successful TAVI at the Cardiologic Unit of University Civil Hospital of Brescia, Italy.

Exclusion criteria were: history of ischemic heart disease, high blood pressure, hypertensive cardiomyopathy, dilated cardiomyopathy, and congenital heart defects. Patients were treated with TAVI if the aortic valve area was < 1 cm², if the European System for Cardiac Operative Risk Evaluation Score (EuroSCORE; Supplementary data online, Appendix S1) (17) was > 20 % or if one or more of the following criteria was met: contraindication for surgery, severely reduced pulmonary function, liver cirrhosis, or metastatic cancer.

All patients underwent the TAVI procedure with a third-generation self-expanding Co-

reValve prosthesis (Medtronic, Minneapolis, MN). The procedure was performed at the catheterization laboratory under local anesthesia and mild sedation with fluoroscopy guidance. The prosthesis was implanted via the transfemoral approach (3). Procedural success was defined as implantation of a functioning aortic prosthesis valve without intraprocedural mortality and with a paravalvular leak < 2.

We state that our study complies with the Declaration of Helsinki and that all patients gave their informed written consent to access their data for scientific purposes. The ethical committee of our hospital approved the study.

The pre-procedural echocardiography acquisition was performed the same day of the procedure.

Blood pressure measurement. Blood pressure was assessed using a standard, calibrated sphygmomanometer. The mean of three sitting and standing blood pressure calculations was recorded. The arm in which the highest sitting diastolic pressures were found was the arm used for all subsequent readings throughout the study. Every effort was made to have the same staff member obtain blood pressure measurements in each individual patient, at the same time of day, using the same equipment. Systolic pressure was recorded when the initial sound was heard (Phase I of the Korotkoff sound), while diastolic pressure was recorded at the disappearance of the sound (Phase V of the Korotkoff sound). The cuff was deflated at a rate not greater than 2 mmHg/s.

Echocardiography. Echocardiograms were done using Vivid 7 (General Electric Medical Systems, Milwaukee, WI, USA) equipment using a 3.5 MHz transducer, with the patients in the left lateral decubitus position, in accordance with the standardization of the American Society of Echocardiography (18). Digital loops were stored

on the hard disk of the echocardiograph for on-line and off-line analyses and transferred to a workstation (EchoPac; GE Health-care, Waukesha, WI, USA) for off-line analysis. All studies were read by two echocardiographists blinded to all patient information. The echocardiographic measurements of the left ventricular end-diastolic diameter (LVEDD), interventricular septal thicknes (IVST), and posterior wall thickness (PWT) were performed off-line according to recommendations of the American Society of Echocardiography (18). Left ventricular volume and ejection fraction were obtained by the modified bi-plane Simpson method. LV mass was calculated according to the formula: $0.8 \times [1.04 \times (LVEDD + PWT + IVST)3 – LVEDD3] + 0.6$ (g), and indexed for body surface area. LVMR was defined as the difference between LV mass index for body surface area after six months and before the procedure. End-diastolic relative wall thickness was the ratio of $2 \times PWT/LVEDD$. Aortic valve area was calculated by the continuity equation, and the maximum pressure gradient across the restrictive orifice was estimated by the modified Bernoulli equation (19). Mean transaortic pressure gradient was calculated by averaging the instantaneous gradients over the ejection period on the continuous-wave Doppler recordings. The assessment of longitudinal peak systolic strain was performed offline to the apical two-chamber, four-chamber, and long-axis views of the left ventricle. In brief, the endocardial contour was manually traced at an end-systolic frame. The software then automatically traced a concentric region of interest including the entire myocardial wall. Strain analysis was performed by dividing each LV image into six segments per view. With the beginning of the QRS complex and the aortic valve closure time as reference points, the peak longitudinal strain was the maximal nega-

tive strain value during the ejection phase. Systolic global longitudinal strain was calculated by averaging the peak systolic values of the 18 segments (20, 21).

Statistical analysis. All analyses were carried out using IBM SPSS Statistics 20 for Windows (SPSS, Inc., Chicago, IL). Continuous variables were tested for normality with the Kolmogorov-Smirnov test and are represented by mean ± standard deviation, while categorical variables are represented as frequency (n) and percentage of the sample. Paired-samples Student's t-test and Wilcoxon signed rank test were performed to analyze the difference between means for continuous variables between baseline and 6-month follow-up, and χ2 test was performed to assess the difference between proportions for categorical variables (Fisher's exact test if dichotomic). Bivariate Spearman's correlation and linear regression were run between LV strain parameters at baseline and LVMR to investigate their predictive value. For all statistical tests probability values < 0.05 were considered significant.

RESULTS

Whole population characteristics at baseline are summarized in *Table 1*. Mean age was 83 ± 8 years, ten patients were male (43.5%) and the Logistic EuroSCORE

Table 1 - *Baseline characteristics of study population.*

Variable	Value
Age (years)	83 ± 8
Sex (n and % of males)	10/23 (43.5%)
Body mass index (kg/m²)	26.88 ± 4.94
Body surface area (m²)	1.77 ± 0.17
Logistic EuroSCORE (%)	24.9 ± 15.1
Aortic valve area index (cm²/m²)	0.33 ± 0.15

EuroSCORE = European System for Cardiac Operative Risk Evaluation Score.

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was $24.9 \pm 15.1\%$. Mean body mass index was 26.88 ± 4.94 kg/m^2. One patient was in New York Heart Association (NYHA) class I (4.3%), seven in NYHA class II (30.5%) and 15 in NYHA class III (65.2%). The peak and mean baseline transvalvular gradients were 88 ± 21 mmHg and 56 ± 16 mmHg, respectively. Calculated aortic valve area index at baseline was 0.33 ± 0.15 cm^2/m^2. At six months of follow-up NYHA class improved with ten patients in NYHA class I (43.5%), 12 in NYHA class II (52.2%), and three in NYHA class III (4.3%) ($p < 0.001$) (*Table 2*), while systolic blood pressure increased from 115 ± 9 to 122 ± 8 ($p = 0.024$) and diastolic pressure from 78 ± 6 to 83 ± 6 ($p = 0.031$).
A complete study data set was available for all patients: the echocardiographic parameters evaluated at baseline and during

follow-up of six months are summarized in *Table 2* and *Figure 1*.
There were significant periprocedural reductions in peak (to 17 ± 7 mmHg; $p < 0.001$) and mean (to 10 ± 5 mmHg; $p = 0.005$) transvalvular gradients. LV ejection fraction improved from $56 \pm 6\%$ to $58 \pm 11\%$ without reaching statistical significance ($p = 0.626$), as well as LVEDD index ($p = 0.079$). Vice versa, IVST and PWT significantly decreased in six months ($p < 0.001$ for both). LV mass index for body surface area showed a highly significant reduction during follow-up ($p < 0.001$); in addition relative wall thickness ($p = 0.011$), longitudinal strain and global longitudinal strain strongly improved ($p < 0.001$ respectively).
The mean LVMR was $23 \pm 14\%$. The distribution of the LVMR grade is shown in *Figure 2*.

Table 2 - *NYHA functional class, blood pressure and left ventricular echocardiographic parameters.*

Variable	0 months	6 months	p
NYHA functional class	I: 1(4.3%) II: 7 (30.5%) III: 15 (65.2%) IV: 0 (0.0%)	I: 10 (43.5%) II: 12 (52.2%) III: 1 (4.3%) IV: 0 (0.0%)	< 0.001
Systolic blood pressure (mmHg)	115 ± 9	122 ± 8	0.024
Diastolic blood pressure (mmHg)	78 ± 6	83 ± 6	0.031
Peak aortic valve gradient (mmHg)	88 ± 21	17 ± 7	< 0.001
Mean aortic valve gradient (mmHg)	56 ± 16	10 ± 5	0.005
LV ejection fraction (%)	56 ± 6	58 ± 11	0.626
IVST (mm)	15 ± 2	13 ± 1	< 0.001
PWT (mm)	14 ± 2	12 ± 1	< 0.001
LVEDD index (mm/m2)	29 ± 4	28 ± 3	0.079
Relative wall thickness	0.56 ± 0.17	0.48 ± 0.09	0.011
LV mass index (g/m2)	190 ± 44	143 ± 30	< 0.001
Peak longitudinal strain 2 chambers (%)	-8.9 ± 0.9	-10.9 ± 0.9	< 0.001
Peak longitudinal strain 3 chambers (%)	-9.8 ± 0.9	-11.8 ± 1.0	< 0.001
Peak longitudinal strain 4 chambers (%)	-9.5 ± 1.0	-11.9 ± 1.1	< 0.001
Global longitudinal strain (%)	-9.4 ± 0.9	-11.5 ± 0.8	< 0.001

NYA = New York Heart Association; LV = left ventricular; IVST = interventricular septal thicknes; PWT = posterior wall thickness; LVEDD = left ventrHicular end-diastolic diameter.
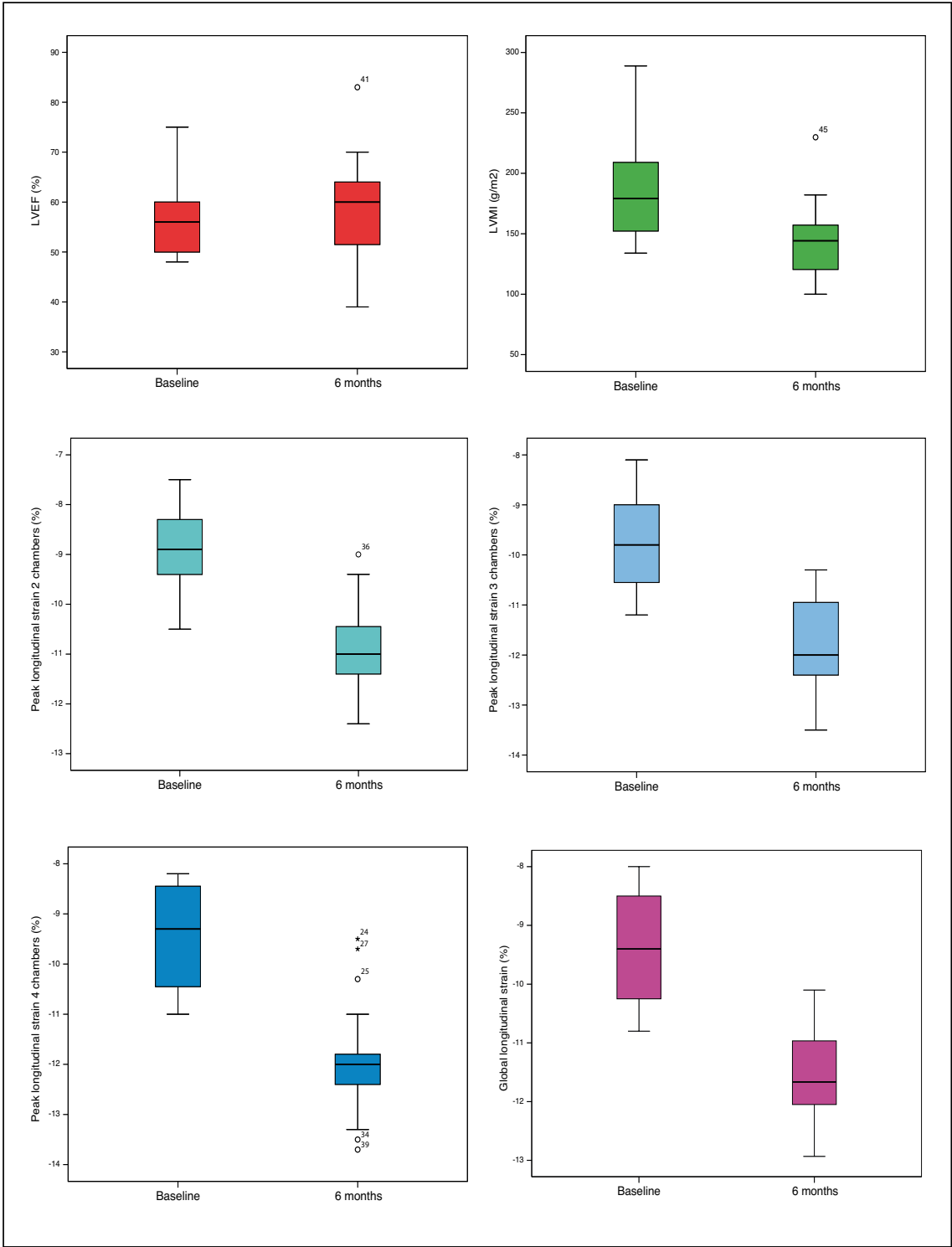
Figure 1 - *Box plots of left ventricular ejection fraction, mass index and strain.*

Figure 2 - *Left ventricular mass regression six months after TAVI.*
TAVI = transcatheter aortic valve implantation.

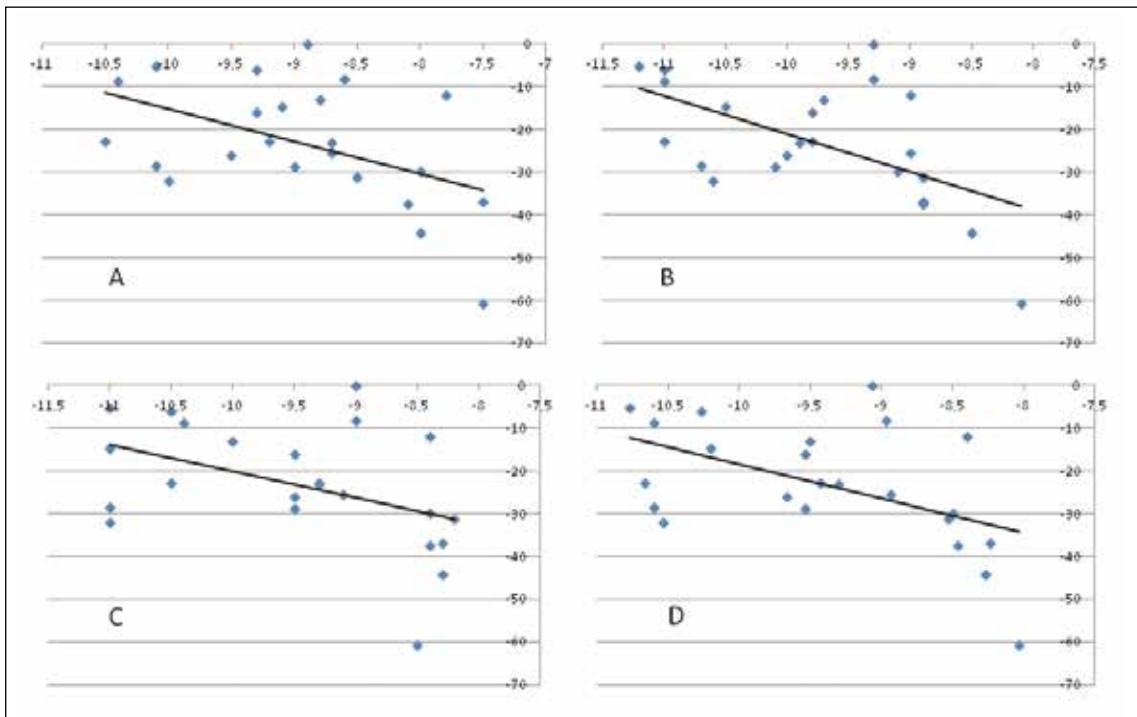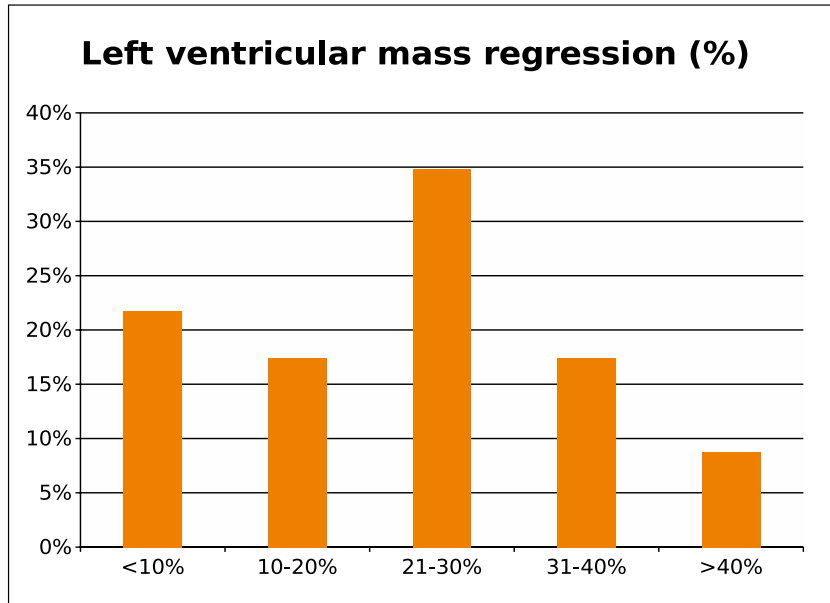Left ventricular mass regression (%)

Figure 3 - *Scatter plots of LVMR and LV longitudinal strain parameters at baseline (A = peak longitudinal strain 2 chambers; B = peak longitudinal strain 3 chambers; C = peak longitudinal strain 4 chambers; D = global longitudinal strain).*
LVMR = left ventricular mass regression; LV = left ventricular.

Table 3 - *Bivariate Spearman's correlations between LVMR and LV longitudinal strain parameters at baseline.*

Parameter	Spearman's ρ coefficient	p
Peak longitudinal strain 2 chambers (%)	-0.454	0.030
Peak longitudinal strain 3 chambers (%)	-0.549	0.007
Peak longitudinal strain 4 chambers (%)	-0.439	0.036
Global longitudinal strain (%)	-0.505	0.014

LVMR = left ventricular mass regression; LV = left ventricular.

Table 4 - *Linear regression between LVMR and LV longitudinal strain parameters at baseline.*

Parameter	p	β	95% CI	Adjusted R²
Peak longitudinal strain 2 chambers (%)	0.023	-0.472	-13.922 – -1.151	0.186
Peak longitudinal strain 3 chambers (%)	0.005	-0.560	-14.858 – -2.914	0.280
Peak longitudinal strain 4 chambers (%)	0.038	-0.436	-11.995 – -0.394	0.152
Global longitudinal strain (%)	0.012	-0.514	-14.240 – -1.970	0.229

LVMR = left ventricular mass regression; LV = left ventricular.

There were no gender-related differences in LVMR, being $-27 \pm 16\%$ in males and $-20 \pm 12\%$ in females (p = 0.376). Bivariate correlations between LVMR and LV longitudinal strain parameters at baseline are shown in F*igure 3* and *Table 3,* while linear regression in *Table 4.* They all were statistically significant.

DISCUSSION

LV hypertrophy in aortic stenosis is an adaptive mechanism to ensure a normal relation between systolic wall stress and ejection fraction (22). Nevertheless, it has widely been associated with impaired long-term survival, myocardial infarction, sudden death, heart failure, and cerebrovascular accidents (23-26). Therefore, LVMR is an important target in this kind of patient, due to the influence of residual LV hypertrophy on long-term survival (27, 28). LVMR ensues after the relief of LV outflow obstruction following aortic valve replacement, which leads to subsequent improved hemodynamics, clinical status, and prognosis (29-31).

Strain imaging is the most appropriate method to assess LV myocardial contractility as it is able to catch subclinical changes in LV performance in aortic stenosis patients (32, 33). In fact, peak longitudinal strain is reduced with increasing severity of aortic stenosis (34) and, in addition, increased LV mass and higher relative wall thickness are associated with reduced LV regional and global myocardial deformation assessed by 2D speckle tracking (35). As a consequence, LV global longitudinal strain improves after surgical aortic valve replacement (13, 36). In particular, Gelsomino et al. recently demonstrated that global longitudinal strain accurately predicts LVMR in patients with pure aortic stenosis undergoing surgical treatment (13).

This study confirms data reported by our group in 2012 regarding LV diastolic function improvement and mass reduction after TAVI (16). Moreover, to the best of our knowledge, only a very recent study analyzed LV longitudinal strain 12 months after the cited procedure (37). We showed that global longitudinal strain significantly improves at 6-month follow-up. Moreover,

our findings seem to extend the predictive value of baseline global longitudinal strain regarding LVMR after aortic valve replacement even in the TAVI context. Myocardial fibrosis in an aortic stenosis heart is responsible for lack of LVMR and poor clinical outcome. In this study we confirmed the hypothesis according to which impaired global longitudinal strain before aortic valve replacement (TAVI in this case) may be due to a certain amount of fibrosis, thus conditioning future lack of LVMR and prognosis (38).

Importantly, our study deals with patients older than cited studies (about 15 years older than Spethmann's) (37). This is the reason for a more reduced LV longitudinal strain than cited studies, even if our patients had all preserved LVEF and no other cardiological comorbidities.

This study has some important limitations. First, the small number of patients and its retrospective nature limit its strength. However, this issue reflects the rarity of patients with severe aortic stenosis who underwent TAVI and without any other cardiovascular disease. Second, although performed by experienced echocardiographists, LV mass measurement and epicardial/endocardial border tracing for 2D strain analysis might be affected by potential errors. Third, we lack a control group. Fourth, the small number of patients prevented us from carrying out a multivariate approach. Furthermore, larger controlled studies are needed to confirm our findings.

CONCLUSION

In conclusion, baseline global longitudinal strain seems to predict LVMR in patients with pure aortic stenosis undergoing TAVI. The assessment of global longitudinal strain, in addition to other echocardiographic parameters, may be helpful in detecting patients undergoing TAVI who are unlikely to benefit from it. Future studies are needed to confirm our results.

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