Original paper

Left ventricular remodelling pattern and its relation to clinical outcomes in patients with severe aortic stenosis treated with transcatheter aortic valve implantation

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Abstract

Introduction: Left ventricular hypertrophy (LVH) is a common compensating process in the pressure overload mechanism of aortic stenosis (AS).

Aim: To identify a group of patients with a LVH pattern which may alter periprocedural and 1-year outcomes after transcatheter aortic valve implantation (TAVI).

Material and methods: Echocardiographic examinations of 226 patients with severe AS treated with TAVI between March 2010 and February 2016 were retrospectively analysed and correlated with echocardiographic parameters and clinical outcomes in the study group. Ultimately 208 patients were enrolled in the study. Based on left ventricular mass index (LVMI) and relative wall thickness (RWT) patients were divided into three categories: concentric remodelling (CR), concentric hypertrophy (CH) and eccentric hypertrophy (EH). Most of the patients with severe AS referred for TAVI were found to have CH (n = 150, 72.8%), then EH (n = 33, 16%) and CR (n = 16, 7.8%).

Results: There were no significant differences between groups in terms of periprocedural outcomes or complications. After a mean observation time of 561.8 ±239.0 days, the observed all-cause mortality rate was 19.9%. After multivariable adjustment, CR remained associated with a higher risk of mortality (HR = 4.31; 95% CI: 1.607–11.538; p = 0.004).

Conclusions: Left ventricular hypertrophy is common in patients with severe AS prior to TAVI. The LVH pattern does not affect TAVI-related complications. In patients with severe AS referred for TAVI, CR seems to be the least favourable geometry of LVH, increasing the risk of 1-year all-cause death.

Key words: mortality, left ventricular hypertrophy, left ventricular remodelling, transcatheter aortic valve implantation, severe aortic stenosis, concentric remodelling.

Introduction

Aortic stenosis (AS) is the most common cardiovascular disease besides hypertension and coronary artery disease in the adult European population [1]. During the long asymptomatic phase of AS the walls of the left ventricle (LV) are subjected to increasing pressure overload which causes gradual thickening of the muscle. Along with growing left ventricular mass (LVM) and occurrence of interstitial fibrosis, the development of diastolic and systolic dysfunction begins, slowly leading to the symptomatic phase, heart failure and death. The risk of an adverse outcome may be diminished by relieving afterload by valve replacement therapy, but the high mortality risk may persist in patients with severe left ventricular hypertrophy (LVH) [2]. The pathomechanism of this relation may be explained by slower regression of LVM after surgical aortic valve replacement (SAVR) and transcatheter aortic valve implantation (TAVI) [3–7].

Geometric changes of LV dimensions in AS are heterogeneous and fall into three categories: concentric remodelling (CR), concentric hypertrophy (CH) and eccentric hypertrophy. They differ between one another in terms of left ventricular diastolic diameter (LVDD), intraventricular septum diastolic diameter (IVSd) and posterior wall thickness (PWT), which contribute to calculating LVM. Given the known effect of LV geometry on TAVI outcomes

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[8–14] we hypothesized that different LVH patterns may affect periprocedural outcomes as well as 1-year prognosis. Some forms of hypertrophy are recognized as predictors of long-term mortality in patients with AS, and preserved ejection fraction (EF) [15], but this relation has not been confirmed in the high-risk TAVI population.

Aim

The aim of the study was to analyse the distribution of different models of LVH in the studied group, to assess the possible link between the abovementioned geometries on periprocedural outcomes, and finally to test whether any of the patterns has an effect on 1-year mortality.

Material and methods

Study design and population

The study was designed as a retrospective, single-centre, observational study with 1-year follow-up of events. Pre- and postprocedural echocardiographic examinations of 226 consecutive patients with severe AS, referred by a local Heart Team's decision for TAVI between March 2010 and March 2016, were analysed. After subtracting data of patients whose examinations were of reduced quality, and those with valve-in-valve procedures, ultimately 208 patients were enrolled in the current study. In each patient relative wall thickness (RWT) and left ventricular mass index (LVMI) were calculated, and according to the results, patients were classified into four categories: concentric hypertrophy, concentric remodelling, eccentric remodelling or normal geometry. The recorded echocardiograms of patients were examined, and accurate measurement of postprocedural values of depth of implantation and parameters describing paravalvular leak (PVL) were obtained. The 1-year follow-up echocardiograms were analysed to determine changes in LVM. Information regarding baseline characteristics and periprocedural proceedings was collected as well as follow-up data concerning outcomes and events. The study was approved by the bioethical commission of the Medical University of Warsaw.

Echocardiography

Two-dimensional Doppler transthoracic echocardiography was performed. The images were obtained in parasternal long- and short-axis views and also two- and four-chamber views. Continuous wave Doppler was used to estimate transvalvular gradients using the Bernoulli equation. Ventricular diameters and posterior and septal wall thickness were measured in two dimensions in the parasagittal view according to guideline recommendations [16]. Each included examination was assessed and besides standard parameters, postprocedural frame borders of the implanted valves were analysed as well as PVL location, volume and number. All measurements were obtained by a single, trained echocardiographer who evaluates TAVI patients on a daily basis.

Study definitions and endpoints

RWT was calculated as RWT = (2 × PWTD ÷ LVDD) and LVMI as LVMI = 0.8 × (1.04 × ((LVDD + PWTD + IVS)^3 – LVDD^3)) + 0.6 and indexed to body surface area (BSA) [17].

Patients with RWT ≤ 0.42 were divided into two categories. Those with LVMI above the cut-off values of 95 g/m^2 for women and 115 g/m^2 for men were included in the eccentric hypertrophy group and the rest were considered as normal.

The group with RWT ≥ 0.42 was also divided according to LVMI into concentric hypertrophy (LVMI ≥ 95 g/m^2 for women and 115 g/m^2 for men) and concentric remodelling [16]. The left ventricular end diastolic volume was assessed using the Teichholz formula. All clinical endpoints were defined by VARC 2 criteria [17].

Statistical analysis

In order to identify the group with the least favourable left ventricle (LV) geometry the 4 groups of patients were compared using the one-way ANOVA test with Tukey's post hoc test when appropriate. The Shapiro-Wilk test was used to confirm or reject normal distribution of each continuous variable. Categorical variables, expressed as counts and percentages, and continuous variables are expressed as means ± SD. Data concerning the number of postprocedural events and complication rate were compared using the χ^2 test or Fisher's exact test, as appropriate. All probability values reported are 2-sided and a value < 0.05 was considered to be significant. Kaplan-Meier curves and log-rank tests of the time-to-event data were used to assess the effect of LV remodelling patterns on all-cause mortality. Afterwards Cox proportional hazard analysis was performed to find possible predictors of endpoints. The proportional-hazards assumption was checked using Schoenfeld residuals. The baseline variables which differed between the predictor and the rest of the group with a p-value < 0.10 were entered in the multivariable Cox model to find independent predictors of 1-year mortality. Data were processed using the SPSS software, version 22 (IBM SPSS Statistics, New York, US) and MedCalc, version 13 (MedCalc Software, Ostend, Belgium).

Results

Differences between remodelling pattern groups

In the study population most of the patients with severe AS referred for TAVI were found to have CH

Table I. Group characteristics with electrocardiographic and echocardiographic findings

Parameter	Concentric remodelling	Eccentric hypertrophy	Concentric hypertrophy	Normal geometry	*P*-value*
Baseline:					
Age [years]	80.6 ±9.1	78.0 ±6.7	79.7 ±7.5	79.1 ±9.5	NS
Female sex, *n* (%)	10 (62.5)	11 (33.3)	82 (54.7)	4 (44.4)	0.023
BMI [kg/m^2]	30.4 ±6.2	27.2 ±4.1	26.6 ±4.4	25.8 ±3.9	NS
BSA (Du Bois) [m^2]	1.9 ±0.2	1.8 ±0.1	1.8 ±0.2	1.8 ±0.2	NS
EuroSCORE I Logistic (%)	11.1 ±5.8	22.0 ±13.3	16.7 ±11.8	23.5 ±21.2	0.044
EuroSCORE II (%)	3.6 ±2.9	6.3 ±5.0	4.1 ±3.1	5.0 ±3.5	0.001
STS (%)	3.2 ±1.9	5.4 ±4.5	3.8 ±2.4	4.4 ±2.5	0.004
Hypertension, *n* (%)	15 (93.8)	21 (63.6)	108 (72.0)	5 (55.6)	NS
Diabetes, *n* (%)	4 (25.0)	14 (42.4)	57 (38.0)	0 (0.0)	NS
eGFR < 30 ml/min, *n* (%)	2 (12.5)	5 (15.2)	13 (8.7)	1 (11.1)	NS
AF, *n* (%)	3 (18.8)	12 (36.4)	56 (37.3)	4 (44.4)	NS
COPD, *n* (%)	2 (12.5)	7 (21.2)	26 (17.3)	1 (11.1)	NS
NYHA ≥ III, *n* (%)	5 (31.3)	25 (75.8)	68 (45.3)	4 (44.4)	0.001
CCS ≥ 3, *n* (%)	3 (18.8)	10 (30.3)	23 (15.3)	2 (22.2)	NS
Myocardial infarction, *n* (%)	2 (12.5)	11 (33.3)	42 (28.0)	4 (44.4)	NS
PCI, *n* (%)	6 (37.5)	10 (30.3)	53 (35.3)	5 (55.6)	NS
CABG, *n* (%)	0 (0)	6 (18.2)	13 (8.7)	1 (11.1)	NS
Stroke/TIA, *n* (%)	2 (12.5)	3 (9.1)	23 (15.3)	0 (0.0)	NS
Permanent pacemaker, *n* (%)	1 (6.3)	5 (15.2)	26 (17.3)	1 (11.1)	NS
PAD, *n* (%)	2 (12.5)	11 (33.3)	22 (14.7)	0 (0.0)	0.006
Electrocardiography:					
QRS [ms]	104.1 ±25.1	107.4 ±43.5	102.5 ±34.6	90.8 ±61.4	NS
Any AVB, *n* (%)	1 (6.3)	2 (6.1)	10 (6.7)	2 (22.2)	NS
RBBB, *n* (%)	1 (6.3)	4 (12.1)	13 (8.7)	1 (11.1)	NS
LBBB, *n* (%)	1 (6.3)	11 (33.3)	20 (13.3)	2 (22.2)	0.004
Echocardiography:					
Moderate/severe MR, *n* (%)	0 (0)	12 (36.4)	19 (12.7)	0 (0.0)	0.001
RV [mm]	28.8 ±4.0	32.1 ±4.0	28.8 ±4.1	28.7 ±6.7	0.001
IVSd [mm]	12.8 ±1.8	12.1 ±2.4	14.5 ±2.1	9.4 ±1.9	0.001
LVDD [mm]	38.0 ±10.3	60.2 ±5.4	47.4 ±6.1	51.8 ±7.5	0.001
PWDTd [mm]	11.3 ±1.6	10.2 ±1.3	13.2 ±1.9	8.7 ±1.2	0.001
LA [mm]	39.1 ±5.9	49.4 ±5.8	43.0 ±6.5	42.0 ±6.2	0.001
EF [%]	61.2 ±4.8	36.3 ±15.0	53.0 ±15.0	54.9 ±16.5	0.001
RWT [mm]	0.8 ±1.0	0.3 ±0.1	0.6 ±0.1	0.3 ±0.1	0.001
LVM [g]	160.3 ±50.9	287.5 ±54.2	268.6 ±71.7	167.2 ±31.5	0.017
LVMI [g/m^2]	84.6 ±26.7	158.7 ±27.9	151.7 ±40.5	92.5 ±8.2	0.044
LVEDV [ml]	68.2 ±28.8	183.2 ±37.1	107.0 ±32.5	131.6 ±47.0	0.001
LVEDVI [ml/m^2]	36.0 ±15.1	100.9 ±18.0	60.4 ±18.4	73.3 ±25.8	0.001
BAV, *n* (%)	1 (6.3)	4 (12.1)	15 (10.0)	1 (11.1)	NS
Aortic annulus [mm]	22.5 ±3.0	23.9 ±5.6	22.8 ±3.3	25.1 ±2.4	NS
LVOT minimal diameter [mm]	19.2 ±3.4	21.5 ±3.1	19.4 ±4.1	21.0 ±3.2	0.006
AVA [cm^2]	0.7 ±0.2	0.9 ±0.8	0.7 ±0.3	1.2 ±1.4	NS
AVAI [cm^2/m^2]	0.4 ±0.1	0.5 ±0.5	0.4 ±0.1	0.5 ±0.1	0.024
Vmax [m/s]	4.2 ±0.5	3.8 ±0.7	4.6 ±0.7	3.6 ±1.1	0.001
PG mean [mm Hg]	51.4 ±22.1	36.6 ±14.4	51.5 ±18.5	36.0 ±15.5	0.001
PG max [mm Hg]	83.6 ±38.2	59.8 ±18.4	80.1 ±30.1	64.7 ±25.3	0.001

*PAD – peripheral artery disease, AVB – atrioventricular block, RWT – relative wall thickness, LVM – left ventricular mass, LVMI – left ventricular mass index, LVEDV – left ventricular end-diastolic volume, LVEDVI – left ventricular end-diastolic volume index, BAV – bicuspid aortic valve, AVA – aortic valve area, AVAI – aortic valve area index, NS – non-significant. *p-value determined by one-way ANOVA.*

Table II. Procedural data end pre-discharge echocardiographic findings

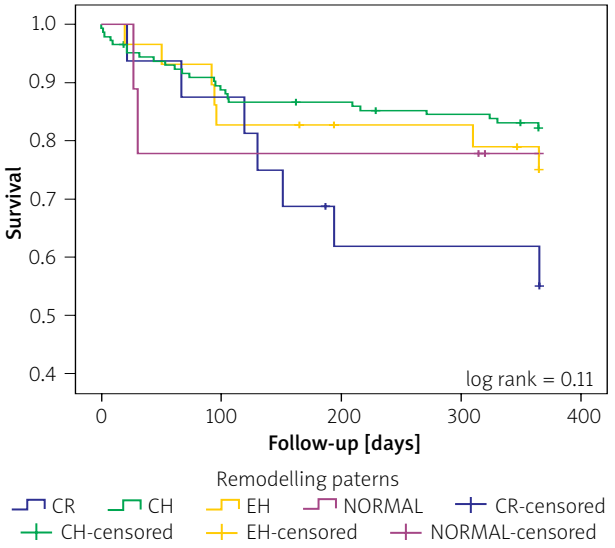
Parameter	Concentric remodelling	Eccentric hypertrophy	Concentric hypertrophy	Normal geometry	*P*-value*
Procedural data:					
Time of the procedure [min]	208.5 ±18.9	216.2 ±67.9	209.5 ±47.3	200.6 ±34.5	NS
Contrast volume [ml]	212.4 ±31.2	199.5 ±48.0	206.0 ±65.1	231.7 ±54.5	NS
Time of fluoroscopy [min]	29.6 ±8.1	28.9 ±9.0	30.7 ±11.7	31.1 ±8.6	NS
Radiation dose [mGy]	967.9 ±726.9	1208.2 ±626.0	1212.5 ±731.0	1338.6 ±524.3	NS
Cover index [mm]	16.4 ±11.9	12.6 ±12.6	15.5 ±10.2	9.9 ±7.6	NS
TF, *n* (%)	12 (75.0)	22 (66.7)	121 (80.7)	8 (88.9)	NS
CV, *n* (%)	8 (50.0)	14 (42.4)	66 (44.0)	1 (11.1)	NS
ES, *n* (%)	3 (18.8)	5 (15.2)	23 (15.3)	1 (11.1)	NS
XT, *n* (%)	3 (18.8)	2 (6.1)	26 (17.3)	0 (0.0)	NS
Lotus, *n* (%)	0 (0)	2 (6.1)	8 (5.3)	1 (11.1)	NS
EV, *n* (%)	0 (0)	5 (15.2)	9 (6.0)	3 (33.3)	NS
Predilatation, *n* (%)	13 (81.3)	18 (54.5)	111 (74.0)	3 (33.3)	NS
Postdilatation, *n* (%)	3 (18.8)	6 (18.2)	34 (22.7)	3 (33.3)	NS
Echo at discharge:					
Any central regurgitation, *n* (%)	3 (18.8)	3 (9.1)	11 (7.3)	2 (22.2)	NS
Mean PVL grade	1.5 ±1.0	1.6 ±1.0	1.4 ±1.0	1.3 ±1.0	NS
Valve frame border (mitral side) [mm]	7.3 ±2.8	7.2 ±2.9	6.5 ±2.8	8.6 ±3.3	NS
Valve frame border (IVS side) [mm]	7.9 ±2.9	7.1 ±2.8	6.4 ±2.8	7.1 ±2.2	NS
Number of PVLs	1.3 ±1.3	1.7 ±0.9	1.7 ±1.3	1.5 ±1.0	NS
AVA [cm^2]	1.7 ±0.3	1.7 ±0.3	1.7 ±0.2	1.8 ±0.3	NS
AVAI [cm^2/m^2]	1.3 ±0.6	1.5 ±0.8	1.2 ±0.7	1.2 ±0.4	NS
Vmax [m/s]	1.9 ±0.5	2.0 ±0.3	2.2 ±0.5	2.0 ±0.4	NS
PG mean [mm Hg]	10.3 ±6.2	8.3 ±3.2	10.9 ±6.0	9.4 ±4.6	NS
PG max [mm Hg]	15.1 ±9.6	16.4 ±6.1	21.1 ±14.5	17.1 ±6.8	NS

TF – transfemoral access, CV – CoreValve, ES – Edwards Sapien, XT – Edwards Sapien XT, EV – Evolut R, PVL – paravalvular regurgitation, IVS – intraventricular septum, AVA – aortic valve area, AVAI – aortic valve area index, Vmax – peak aortic jet velocity, PG – pressure gradient.

Table III. Postprocedural outcomes defined by VARC-2 criteria

Parameter	Concentric remodelling	Eccentric hypertrophy	Concentric hypertrophy	Normal geometry	Total	*P*-value
Stroke/TIA	0 (0%)	2 (6.1%)	6 (4.0%)	0 (0.0%)	8 (3.8%)	0.08
Severe PPM	1 (7.1%)	1 (3.2%)	5 (3.3%)	0 (0.0%)	7 (3.4%)	0.81
PM implantation	2 (14.3%)	5 (16.1%)	22 (15.8%)	0 (0.0%)	29 (15.2%)	0.08
PG mean > 20 mm Hg	3 (21.4%)	0 (0.0%)	8 (5.8%)	0 (0.0%)	11 (5.8%)	0.19
Moderate/severe PVL	2 (14.3%)	9 (29.0%)	28 (20.1%)	1 (14.3%)	40 (20.9%)	0.18
30-day mortality	2 (12.5%)	2 (6.1%)	11 (7.3%)	2 (22.2%)	17 (8.2%)	0.12

PPM – patient-prosthesis mismatch, PM – permanent pacemaker.



Number at risk	100 days	200 days	365 days
CR	14	10	9
CH	134	131	125
EH	28	28	26
Normal	7	7	7

Figure 1. Kaplan-Meier survival curves

CR – concentric remodelling, CH – concentric hypertrophy, EH – eccentric hypertrophy, NORMAL – normal geometry.

(*n* = 150, 72.8%), then EH (*n* = 33, 16%) and CR (*n* = 16, 7.8%). The remaining 9 (4.3%) patients were found to have normal LV geometry. Patients with EH were characterised by the lowest pre-procedural EF (36.3 ±15.0%), highest occurrence of left bundle branch block (LBBB) (33.3%), and moderate mitral regurgitation (36.4%). The predicted mortality risk by STS score and EuroSCORE II was the highest in this group (5.4 ±4.5% and 6.3 ±5.0%, respectively). The EH group was also the most symptomatic – 75.8% of patients were found to be in New York Heart Association (NYHA) class III or higher prior to the procedure.

Patients with CR were mainly women (62.5%) with the lowest mortality risk score values. The group was also characterised by the lowest minimal left ventricular outflow tract (LVOT) diameter (19.2 ±3.4 mm), left ventricle end-diastolic volume (LVEDV) (68.2 ±28.8 ml), LVM (160.3 ±50.9 g) along with the highest pre-procedural EF (61.2 ±4.8%) and mean pressure gradient (51.4 ±22.1 mm Hg).

The group with CH pattern had the highest peak aortic jet velocity (Vmax) and IVSd.

The group of severe AS patients who were found to have normal geometry was unremarkable, besides having the lowest IVSd and PWT.

All between-group differences were statistically significant at $p < 0.05$ (Table I).

Table IV. Cox proportional hazard analysis. Factors included in univariate analysis were significantly different between concentric remodelling group and others at $p < 0.1$

Parameter	Univariate				Multivariate			
	P-value	Hazard ratio (HR)	95% confidence intervals		*P*-value	Hazard ratio (HR)	95% confidence intervals	
			Lower	Upper			Lower	Upper
Weight [kg]	0.24	1.23	0.87	1.75				
BMI [kg/m^2]	0.20	0.73	0.45	1.18				
Hypertension, *n* (%)	0.86	1.09	0.41	2.92				
Severe/moderate MR	0.40	1.77	1.03	3.04				
IVSd [mm]	0.15	0.57	0.26	1.24				
LVDD [mm]	0.21	0.48	0.15	1.53				
PWDTd [mm]	0.99	0.99	0.08	11.87				
Ao [mm]	0.27	1.05	0.96	1.14				
LA [mm]	0.69	0.99	0.91	1.06				
EF (%)	0.73	1.01	0.98	1.04				
Concentric remodelling	0.05	4.68	0.99	21.9	0.01	4.31	1.61	11.54
LVM	0.25	1.04	0.97	1.11				
LVMI	0.83	0.99	0.88	1.11				
LVEDV [ml]	0.05	1.04	0.86	1.26	0.01	1.01	1.00	1.02
LVEDVI [ml/m^2]	0.12	1.05	0.82	1.35				
IVS maximal diameter [mm]	0.16	0.92	0.81	1.03				
Valve frame border (IVS side) [mm]	0.99	0.99	0.88	1.14				

BMI – body mass index, MR – mitral regurgitation, IVSd – interventricular septum diastolic diameter, LVDD – left ventricular diastolic diameter, PWDTd – posterior wall diastolic thickness, Ao – aorta, LA – left atrium, EF – ejection fraction, LVM – left ventricular mass, LVMI – left ventricular mass index, LVEDV – left ventricular end diastolic volume, LVEDVI – left ventricular end diastolic volume index.

Periprocedural outcomes

In terms of periprocedural proceedings there was a significant difference in pre-dilation number, with CR being the most frequent recipients (81.3%) (Table II). Analysing postprocedural outcomes there were no statistically significant between-group differences, with non-significantly higher rates of pacemaker implantation and stroke in EH patients (Table III).

One-year follow-up results

In 1-year observation the overall, all-cause mortality rate was 19.7%. In the Kaplan-Meier survival plot there were no statistically significant differences in terms of 1-year mortality (log rank 0.11; Figure 1). In the Cox proportional hazard model the presence of CR was a significant predictor of 1-year mortality. After including cofounding factors from baseline variables which differed between the predictor and rest of the group with a *p*-value < 0.10, CR and LVEDV were found to be independent predictors of 1-year mortality (Table IV).

Discussion

In the present study we recognized patients with CR as potentially being at increased risk of mortality after TAVI. The main findings of this observational study were: (1) the most frequent LVH pattern in patients with severe AS was CH; (2) LVH patterns did not have a significant impact on periprocedural complications; (3) CR and LVEDV are independent predictors of 1-year mortality after TAVI.

In previous studies of the effect of remodelling patterns on mortality in patients with AS, the group which was at highest risk was the CH group [7]. In our study we did not find an association between having CH and increased risk of death, but these studies were performed on different AS populations, with ours being an older group just prior to TAVI.

To the best of our knowledge this was the first study to compare periprocedural outcomes after TAVI in different LVH pattern groups. What may be surprising is that the group with the thickest IVS and smallest LVOT diameter (CR) did not have an increase in complications known to be related to those parameters i.e. pacemaker implantations (PM) and moderate/severe paravalvular leak. Although this relation may be mostly due to the relatively small sample size, this may also contribute to the high mean cover index in the CR group (16.4 ±11.9 mm).

In the present study we found a non-significantly higher PM rate in EH patients. Considering the highest percentages of right bundle branch block (RBBB) and LBBB in this group, this result supports earlier reports regarding bundle branch blocks as an important factors affecting the PM rate. Also in the EH group there was a non-significantly higher stroke rate. Whether this is just an aleatory finding or is in some way in concordance

Table V. Comparison of pre-procedural and 12-month follow-up echocardiograms of patients who survived. *P*-value assessed by Wilcoxon signed rank test

Parameter	Concentric remodelling	*P*-value	Eccentric hypertrophy	*P*-value	Concentric hypertrophy	*P*-value	Normal geometry	*P*-value
Moderate/severe MR, *n* (%)	0 (0)	2 (12.5)	0.157	12 (36.4)	9 (27.3)	0.317	19 (12.7)	9 (6.0)	0.012	0 (0.0%)	0 (0.0%)	1
RV [mm]	28.8 ±4.0	27.6 ±4.0	0.075	32.1 ±4.0	32.0 ±4.5	0.776	28.8 ±4.1	29.1 ±3.6	0.275	28.7 ±6.7	29.4 ±4.6	0.609
IVSd [mm]	12.8 ±1.8	12.3 ±1.4	0.391	12.1 ±2.4	12.3 ±1.9	0.833	14.5 ±2.1	13.3 ±2.2	0	9.4 ±1.9	10.2 ±2.1	0.2
LVDD [mm]	38.0 ±10.3	42.9 ±5.3	0.033	60.2 ±5.4	56.9 ±7.3	0	47.4 ±6.1	47.7 ±6.0	0.231	51.8 ±7.5	50.8 ±9.3	0.395
PWDTd [mm]	11.3 ±1.6	11.0 ±1.5	0.618	10.2 ±1.3	10.6 ±1.4	0.126	13.2 ±1.9	12.0 ±1.9	0	8.7 ±1.2	9.7 ±1.7	0.176
Ao [mm]	30.1 ±4.3	29.5 ±4.0	0.799	34.4 ±3.9	33.8 ±4.0	0.604	33.2 ±5.0	32.6 ±5.4	0.154	33.6 ±5.1	33.0 ±4.7	0.655
LA [mm]	39.1 ±5.9	39.8 ±5.8	0.258	49.4 ±5.8	47.8 ±6.3	0.024	43.0 ±6.5	42.7 ±6.2	0.441	42.0 ±6.2	44.3 ±6.5	0.125
EF (%)	61.2 ±4.8	61.5 ±4.6	0.799	36.3 ±15.0	41.5 ±14.6	0.004	53.0 ±15.0	55.7 ±10.5	0.073	54.9 ±16.5	55.4 ±11.2	0.854
RWT [mm]	0.8 ±1.0	0.5 ±0.1	0.047	0.3 ±0.1	0.4 ±0.1	0.005	0.6 ±0.1	0.5 ±0.1	0	0.3 ±0.1	0.4 ±0.1	0.091
LVM [g]	160.3 ±50.9	179.1 ±45.9	0.139	287.5 ±54.2	269.3 ±74.9	0.061	268.6 ±71.7	237.4 ±66.1	0	167.2 ±31.5	187.4 ±56.2	0.398
LVMI [g/m^2]	84.6 ±26.7	94.9 ±24.2	0.173	158.7 ±27.9	148.8 ±39.7	0.072	151.7 ±40.5	133.6 ±35.5	0	92.5 ±8.2	104.8 ±32.9	0.398
LVEDV [ml]	68.2 ±28.8	84.3 ±23.7	0.028	183.2 ±37.1	163.0 ±46.8	0	107.0 ±32.5	108.5 ±31.7	0.258	131.6 ±47.0	127.8 ±55.3	0.498
LVEDVI [ml/m^2]	36.0 ±15.1	44.9 ±13.3	0.028	100.9 ±18.0	89.6 ±23.8	0	60.4 ±18.4	61.3 ±17.9	0.287	73.3 ±25.8	71.1 ±31.2	0.499

MR – mitral regurgitation, RV – right ventricle, IVSd – interventricular septum diastolic diameter, LVDD – left ventricular diastolic diameter, PWDTd – posterior wall diastolic thickness, Ao – aorta, LA – left atrium, EF – ejection fraction, RWT – relative wall thickness, LVM – left ventricular mass, LVMI – left ventricular mass index, LVEDV – left ventricular end diastolic volume, LVEDVI – left ventricular end diastolic volume index.

with the low EF and its potentially thrombogenic effect needs to be confirmed in a larger, randomized trials.

While trying to explain the effect of CR on 1-year mortality, we analysed the differences between pre-procedural and 1-year echocardiograms of the remaining group (Table V). The crucial difference between CR and other groups was that it was the only group which showed a significant LVEDV and LVEDVi increase, driven by a significant increase in LVDD. This also explains the role of LVEDV on mortality found in proportional hazard analysis. Based on these results we can assume that it is not the absolute value of LVEDV that drives the mortality, but the increase of this factor.

The biggest limitation of the current study is that it was a retrospective analysis undertaken on a relatively small group of patients. There was also a lack of mid-term analysis which could enhance the insight into echocardiographic parameter changes of those patients who were still alive. Information regarding patients' baseline medications as well as biomarker values was unavailable in this study.

Conclusions

Most of the patients with severe symptomatic AS referred for TAVI already have one type of LV remodelling (95.7% of the studied group). The LV remodelling patterns have no influence on the occurrence of periprocedural complications. Concentric remodelling and increase in LVEDV are independent predictors of 1-year mortality after TAVI.

Conflict of interest

The authors declare no conflict of interest.

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