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Effect of Prosthesis Orifice Area on Left Ventricular Reverse Remodeling After Surgical Aortic Valve Replacement

Marián Homola, Tomáš Toporcer, Tomáš Lopuchovský, Anton Bereš, Michal Trebišovský, Štefan Lukačín, Adrián Kolesár

Department of Heart Surgery, East Slovak Institute for Cardiovascular Diseases and Medical Faculty, Pavol Jozef Šafárik University, Košice, Slovakia

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SÚHRN

Cieľ: Cieľom štúdie bolo zhodnotiť, či veľkosť chlopňovej protézy po chirurgickej náhrade aortálnej chlopne (SAVR) ovplyvňuje rozsah reverznej remodelácie ľavej komory, a to aj pri neprítomnosti nezhody medzi pacientom a protézou.
Materiál a metódy: Do štúdie bolo zaradených 216 pacientov, ktorí v období od januára 2021 do decembra 2024 podstúpili izolovanú primárnu SAVR pre aortálnu stenózu s použitím jedného modelu chlopňovej protézy a ktorí nemali predoperačne ani pooperačne prítomnú fibriláciu predsiení. Echokardiografické parametre boli hodnotené predoperačne a jeden rok po operácii. Zmeny remodelácie ľavej komory boli analyzované vo vzťahu k indexovanej geometrickej ploche ústia protézy (iGOA).
Výsledky: Pri jednoročnej kontrole bolo zaznamenané významné zlepšenie takmer všetkých echokardiografických parametrov. Väčšia iGOA bola významne spojená s echokardiograficky zaznamenaným väčším nárastom indexovanej plochy aortálnej protézy (iAVA) (B = 0,321; 95% CI 0,134–0,509; *p* = 0,004) a s výraznejším zmenšením indexovaného end-systolického a end-diastolického rozmeru ľavej komory (iLVeSD a iLVeDD) (B = –6,578; 95% CI –13,071 až –0,086; *p* = 0,047 a B = –2,48; 95% CI –4,815 až –0,144; *p* = 0,038). Pri väčšej chlopňovej protéze sa pozoroval aj štatisticky nevýznamný trend k výraznejšiemu zlepšeniu ejekčnej frakcie ľavej komory (LVEF), k väčšiemu poklesu indexovanej hmotnosti ľavej komory (LVMi) a indexovanému objemu ľavej predsiene (LAVi).
Záver: Použitie väčšej chlopňovej protézy pri SAVR je spojené s výraznejšou reverznou remodeláciou ľavej komory, a to aj pri neprítomnosti nezhody medzi pacientom a protézou. Štatisticky významný vplyv na krátkodobé prežívanie sa nepreukázal.

Keywords:
Aortic stenosis
Indexed geometric orifice area
Reverse left ventricle remodeling
Surgical aortic valve replacement

ABSTRACT

Aim: This study aimed to evaluate whether valve prosthesis size after surgical aortic valve replacement (SAVR), even in the absence of patient–prosthesis mismatch, influences the magnitude of reverse left ventricular remodeling.
Material and methods: A total of 216 patients undergoing isolated SAVR for native AS with a single prosthetic valve model and without preoperative or postoperative atrial fibrillation between January 2021 and December 2024 were included. Echocardiographic parameters were assessed preoperatively and at 1-year follow-up. Changes in left ventricular remodeling were analyzed in relation to indexed geometric orifice area (iGOA).
Results: At 1-year follow-up, significant improvement was observed in nearly all echocardiographic parameters. Larger iGOA was significantly associated with an increase in indexed aortic valve area difference (B = 0.321, 95% CI 0.134–0.509; *p* = 0.004) and greater reductions in indexed left ventricular end-systolic and end-diastolic diameters (iLVeSD and iLVeDD) (B = –6.578, 95% CI –13.071 to –0.086, *p* = 0.047; B = –2.48, 95% CI –4.815 to –0.144, *p* = 0.038; respectively). A nonsignificant trend toward greater improvement in left ventricle ejection fraction (LVEF) and greater reductions in left ventricle mass index (LVMi) and left atrium volume index (LAVi) was also observed with larger valve prosthesis.
Conclusion: Larger prosthetic valve size during SAVR is associated with more pronounced reverse left ventricular remodeling, even in the absence of patient–prosthesis mismatch. No statistically significant effect on short-term survival was observed.

Address: Doc. MUDr. Tomáš Toporcer, PhD., Department of Heart Surgery, East Slovak Institute for Cardiovascular Diseases, Ondavská 8, 04001 Košice, Slovakia, e-mail: topyto@gmail.com
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Introduction

Aortic stenosis (AS) represents the most prevalent valvular heart disease in Western countries.[1] The incidence of this valvular disease increases with age, affecting approximately 2% of individuals older than 65 years and up to 12% of the population older than 75 years.[2,3] According to current European Association for Cardio-Thoracic Surgery (EACTS) guidelines, valve intervention (either surgical or transcatheter) is primarily indicated at an advanced stage of the disease.[4] However, increasing evidence suggests that significant myocardial alterations occur already in the preclinical stages of AS, with increased afterload triggering left ventricular remodeling before the onset of clinical symptoms.[5,6] Sustained pressure overload and altered hemodynamics lead to the classical development of left ventricular (LV) hypertrophy.[5,7,8] Impaired myocardial perfusion due to supply–demand mismatch promotes myocyte loss and fibrosis, with irreversible replacement fibrosis and partially reversible diffuse fibrosis.[3] LV remodeling leads to deterioration of both systolic and diastolic LV function.[5]

Aortic valve (AV) replacement (AVR) remains the gold standard of treatment for symptomatic severe AS. It is the most frequently performed valvular intervention, with approximately 280,000 procedures performed worldwide each year.[1] AVR effectively eliminates left ventricular outflow obstruction. This results in a significant reduction of the transvalvular pressure gradient. The decrease in afterload is expected to initiate reverse left ventricular remodeling (RLVR).[5,6,8,9] The extent of this reverse remodeling appears to depend on the degree of afterload reduction and on the presence of potentially reversible myocardial changes prior to the intervention. However, echocardiographic follow-up at 1 year after surgical or transcatheter AV replacement in the PARTNER 2 trial demonstrated improvement in the stage of cardiac damage in 16% of patients, no change in 58%, and worsening in 27%.[3] This suggests that reverse remodeling is a complex process dependent on multiple factors, including changes at the subcellular level.[7]

An important determinant of outcomes after AVR is the size of the implanted prosthetic valve, as reflected by the postoperative indexed aortic valve area (iAVA). Numerous studies have reported worse outcomes when the iAVA after AVR is <0.65 cm²/m², a condition defined as severe patient–prosthesis mismatch (PPM).[10–14] However, the PARTNER studies did not demonstrate impaired survival in patients with measured moderate PPM, defined by an measured iAVA between 0.65 and 0.85 cm²/m².[13] Moreover, some studies have reported a lower incidence of reoperation in patients with moderate PPM compared not only with those with severe PPM but also with patients without PPM (iAVA >0.85 cm²/m²).[12] In contrast, our previous study demonstrated preferentially better survival in patients with larger iAVA of a bioprosthetic valve compared with those with iAVA values at the borderline between moderate PPM and no PPM.[15] It is therefore notable that further increases in iAVA beyond the thresholds defining PPM are associated with more favorable myocardial adaptation and more pronounced RLVR. Therefore, this study aimed to determine whether prosthetic valve size after surgical AVR, even in the absence of standard PPM criteria, influences the magnitude of RLVR.

Material and methods

This retrospective observational study included all patients who underwent surgical aortic valve replacement at our department between January 2021 and December 2024. Patients who underwent concomitant cardiac surgical procedures were excluded. Patients undergoing redo aortic valve surgery and those in whom surgery was not indicated for native aortic valve stenosis were excluded subsequently. Data regarding the type of implanted prosthetic valve and the presence of preoperative and postoperative atrial fibrillation were obtained through a review of medical records. To ensure homogeneity of the hemodynamic properties of the implanted valve, the most frequently used prosthetic valve model was identified. All patients who received a prosthetic valve other than this most commonly implanted model were subsequently excluded. Furthermore, to achieve homogeneity in left ventricular response to post-operative hemodynamics following aortic valve replacement, patients with preoperative or postoperative permanent or persistent atrial fibrillation were excluded. Ultimately, only patients who underwent isolated surgical aortic valve replacement with a single prosthetic valve model for native aortic valve stenosis and without preoperative or postoperative permanent or persistent atrial fibrillation were included in the final analysis.

The following preoperative and operative data were obtained for all patients through a review of medical records: age at the time of surgery, use of a minimally invasive approach via partial upper ministernotomy (PUMS), cardiopulmonary bypass (CPB) time, aortic cross-clamp time, need for postoperative surgical re-exploration, size of the implanted prosthetic valve (mm), length of hospital stay, body mass index (BMI), and the presence of diabetes mellitus, hepatopathy, chronic obstructive pulmonary disease (COPD), and a history of stroke. Based on the size of the implanted prosthetic valve and the patient's body surface area (BSA) at the time of the surgery, the indexed geometric orifice area (iGOA) was calculated for each patient. Echocardiographic (ECHO) parameters were obtained at two time points: preoperatively and at 1-year follow-up. The following ECHO variables were analyzed: left atrial (LA) diameter, left ventricular ejection fraction (LVEF), mean transaortic gradient, peak transaortic gradient, peak aortic jet velocity, interventricular septal diameter (IVSD), indexed left atrial volume (LAVi), the E/e′ ratio (the e′ value was calculated as the average of medial and lateral e′ velocities), ECHO derived indexed aortic valve area (iAVA), indexed left ventricular end-systolic diameter (iLVeSD), and indexed left ventricular end-diastolic diameter (iLVeDD). Finally, left ventricular mass index (LVMi) was calculated both preoperatively and at follow-up. Changes in ECHO parameters were expressed as the difference (Δ) between values obtained at 1-year follow-up and those measured preoperatively. Patient survival was determined using publicly available data based on the patients' health insurance records.

Results are presented as the number of positive cases/total number of patients (percentage) for qualitative variables and as mean ± standard deviation for quantitative variables. Differences between preoperative and postoperative ECHO parameters were assessed using a paired Student's t-test. The association between iGOA and changes in ECHO parameters was evaluated using linear regression analysis. The impact of iGOA on patient survival was assessed using Cox proportional hazards regression analysis. Statistical analyses were performed using Microsoft Excel and IBM SPSS Statistics, version 20, with a significance level set at p <0.05.

Results

A total of 910 patients who underwent AVR between January 2021 and December 2024 were initially identified. After exclusion of patients who underwent concomitant cardiac surgery, those undergoing redo aortic valve surgery, and those operated on for indications other than aortic stenosis, 327 patients remained. The most frequently implanted prosthetic valve model was used in 238 patients from this cohort. Among these, permanent or persistent atrial fibrillation (AF) was identified preoperatively or postoperatively in 22 patients (**Fig. 1**). Consequently, the final statistical analysis included 216 patients in whom preoperative and operative characteristics, as well as ECHO parameters obtained preoperatively and at 1-year follow-up, were evaluated. In this cohort, changes in ECHO parameters and patient survival were further analyzed in relation to the iGOA.

Preoperative and operative characteristics of the study population are summarized in **Table 1**. When changes in ECHO parameters were evaluated (1 year after surgery versus preoperatively), statistically significant differences were observed in 10 of the 11 assessed variables. A significant reduction in left atrial diameter of 1.44 mm was noted, along with an increase in LVEF of 2.44%. Mean and peak transaortic pressure gradients decreased significantly by 37 mmHg and 61 mmHg, respectively, accompanied by a reduction in peak aortic jet velocity of 2.11 m/s. Interven-

Table 1 – Preoperative parameters and comparison of echocardiographic measurements obtained preoperatively and 1 year after aortic valve replacement

Clinical parameter	Preoperatively		
Age	66.54 ± 7.36		
PUMS	76/216 (35%)		
CPB time (min)	85.18 ± 22.27		
Clamp time (min)	69.39 ± 16.25		
Surg. revision	13/216 (6%)		
Prothesis size (mm)	22.29 ± 1.67		
Hospitalization (days)	10.56 ± 4.8		
BMI (m^2/kg)	31.58 ± 17.88		
Diabetes mellitus	62/216 (29%)		
Hepatopathy	30/216 (14%)		
Stroke history	13/216 (6%)		
COPD	28/216 (13%)		
	Preoperatively	**1 year after surgery**	***p***
LA diameter (mm)	**42.44 ± 6.32**	**41.00 ± 6.02**	**<0.0001**
LVEF (%)	**55.02 ± 9.4**	**57.46 ± 6.24**	**<0.0001**
Mean grad. AV (mmHg)	**52.26 ± 19.25**	**14.80±5.19**	**<0.0001**
Peak grad. AV (mmHg)	**87.46 ± 28.59**	**26.39 ± 9.58**	**<0.0001**
V_{max} AV (m/s)	**4.61 ± 0.73**	**2.5 ± 0.46**	**<0.0001**
IVSD (mm)	**13.95 ± 2.35**	**12.35 ± 2.01**	**<0.0001**
LAVi (mL/m^2)	39.91 ± 13.45	38.17 ± 10.69	0.4519
E/avrg e'	**10.15 ± 3.78**	**9.21 ± 3.26**	**0.0064**
iAVA (cm^2/m^2)	**0.37 ± 0.1**	**0.78 ± 0.19**	**<0.0001**
iLVeSD (mm/$m^{2)}$	**17.96 ± 4.93**	**15.91 ± 3.19**	**0.0035**
iLVeDD (mm/m^2)	**25.23 ± 3.63**	**24.78 ± 2.77**	**<0.0001**
LVMi (g/m^2)	130.27 ± 41.06	99.06 ± 22.66	<0.0001

AV – aortic valve; BMI – body mass index; CPB – cardiopulmonary bypass; iAVA – indexed aortic valve area; iLVeDD – indexed left ventricular end-diastolic diameter; iLVeSD – indexed left ventricular end-systolic diameter; IVSD – interventricular septal diameter; LA – left atrium; LAVi – indexed left atrial volume; LVEF – left ventricular ejection fraction; PUMS – partial upper ministernotomy; V_{max} AV – peak aortic jet velocity.

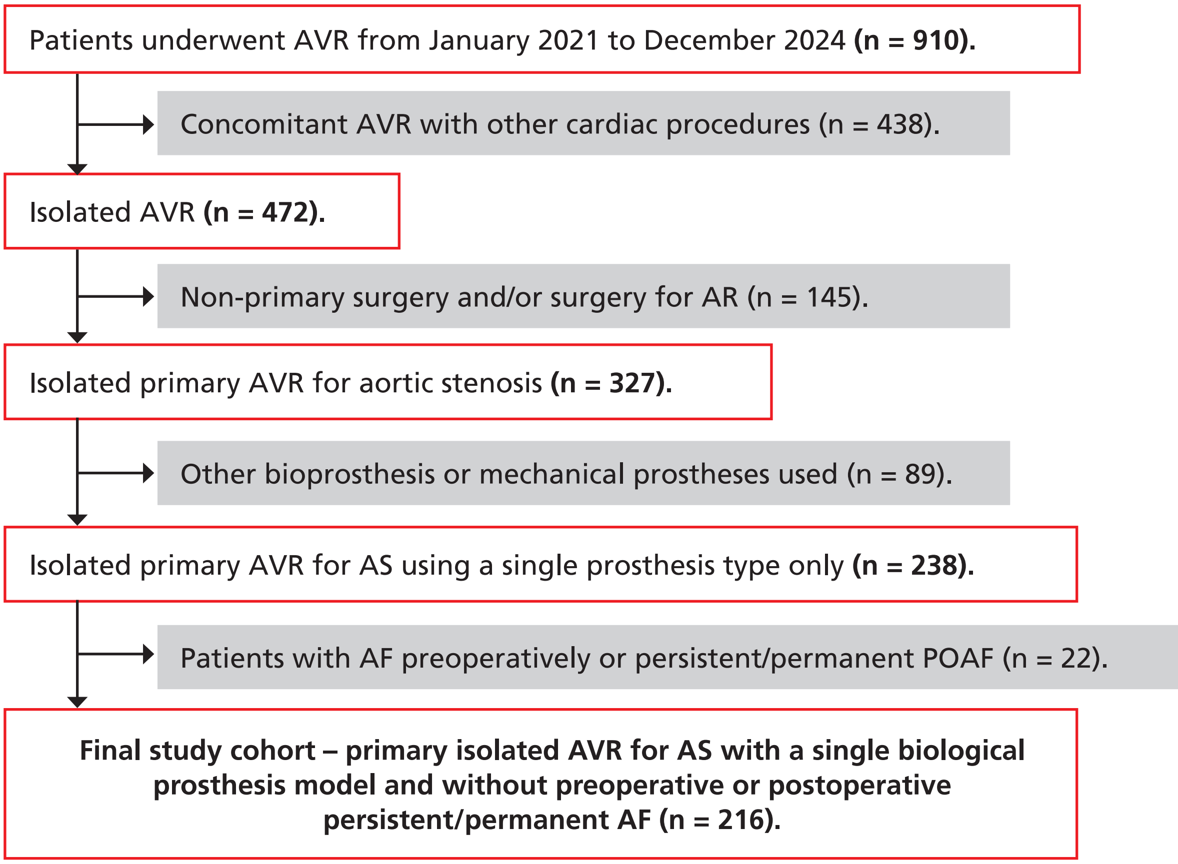

Fig. 1 – Patient flow diagram showing cohort selection with inclusion and exclusion criteria. AF – atrial fibrillation; AR – aortic regurgitation; AS – aortic stenosis; AVR – aortic valve replacement; POAF – postoperative atrial fibrillation.

tricular septal thickness decreased by 1.6 mm, and the E/e′ ratio declined by 0.89, calculated as the average of medial and lateral e′ velocities. The iAVA more than doubled compared with preoperative values, increasing by 0.41 cm²/m². Indexed left ventricular end-systolic and end-diastolic diameters decreased by 2.02 and 0.45 mm/m², respectively. A significant reduction in LVMi was observed, decreasing from 130.27 ± 41.06 g/m² preoperatively to 99.06 ± 22.66 g/m² at 1-year follow-up, corresponding to a mean reduction of 29.33 g/m² (p <0.0001). No statistically significant change was observed in LAVi, which showed a nonsignificant reduction of 1.74 mL/m² (**Table 1**).

Assessment of the impact of iGOA on changes in ECHO parameters identified three variables whose changes were significantly associated with prosthetic valve size, as reflected by iGOA. Larger iGOA was associated with

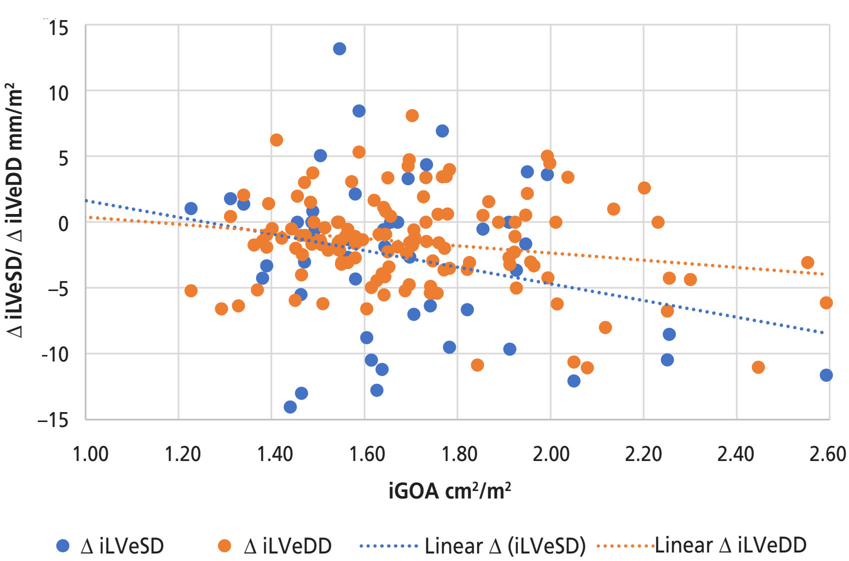

Fig. 2 – Scatter plot with a linear regression line showing the association between valve prosthesis iGOA and the 1-year change in ΔiLVeSD, and ΔiLVeDD. iGOA – indexed geometric orifice area; Δ iLVeDD – 1-year change in indexed left ventricular end-diastolic diameter; Δ iLVeSD – 1-year change in indexed left ventricular end-systolic diameter.

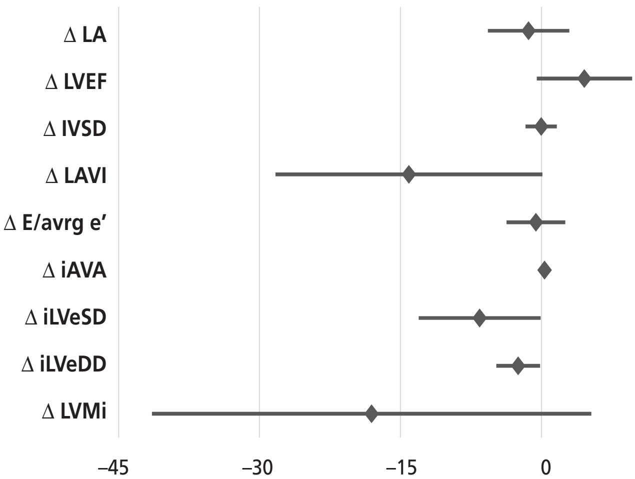

Fig. 3 – Graphical representation of the linear regression between prosthesis iGOA and 1-year changes in left atrial and left ventricular echocardiographic parameters.

a significantly greater increase in iAVA (B = 0.321, 95% CI 0.134–0.509; *p* = 0.004) and with significantly greater reductions in iLVeSD and iLVeDD (B = –6.578, 95% CI –13.071 to –0.086, *p* = 0.047; B = –2.48, 95% CI –4.815 to –0.144, *p* = 0.038; respectively) (**Table 2, Figs 2, 3**). Borderline associations were observed between iGOA and increase in LVEF (B = 4.566, 95% CI –0.513 to 9.645, *p* = 0.078), as well as reduction in LAVi (B = –14.114, 95% CI –28.317 to –0.089, *p* = 0.051). A numerically greater, but not statistically significant, reduction in LVMi was also observed in patients receiving a valve prosthesis with larger iGOA (B = –18.083; 95% CI –41.461 to 5.296; *p* = 0.128) (**Table 2, Figs 3, 4**). No significant association was found between iGOA and changes in LA diameter, IVSD or the E/e′ ratio (**Table 2, Figs 3, 4**). Cox regression analysis did not demonstrate a statistically significant effect of prosthetic valve size, as defined by iGOA, on short-term patient survival (HT = 1.138, 95% CI 0.012–1.649, *p* = 0.118) (**Fig. 5**).

Discussion

Imaging modalities for the assessment of reverse left ventricular remodeling

Transthoracic ECHO represents the gold standard for the diagnosis of aortic stenosis as well as for the assessment of the compensatory left ventricular response to this condition. Moreover, it is a well-established modality for evaluating the extent of RLVR following AVR.[5] In addition to standard parameters, which were also included in the present study, left ventricle global longitudinal strain has recently been incorporated among parameters used to identify and quantity RLVR.[6,16] Another modality for the assessment of reverse remodeling is cardiac magnetic resonance imaging (MRI).[8,17] The presence of reverse remodeling is defined by a >15% reduction in end-diastolic volume, a >15% decrease in indexed left ventricular mass, a >10% reduction in geometric remodeling (LV mass / end-diastolic volume ratio), or a >10% increase in left ventricular ejection fraction (LVMi).[18] LVMi has also been evaluated in studies using ECHO, where it is mathematically derived from IVSD, LVeDD, and posterior wall thickness.[6] The same calculation method was applied in the present study. The literature reports a reduction in LVMi from 123.6 ± 32.1 to 109.7 ± 28.9 g/m² three months after TAVI.[6] Notably, our results demonstrate a slightly more pronounced reduction (mean decrease of 29.33 g/m²) one year after surgical AVR. Cardiac magnetic resonance imaging allows differentiation of left ventricular mass regression into cellular and extracellular components. Using this approach, a predominantly cellular regression of 18.7% and a smaller contribution from extracellular mass regression of 7.2% have been documented two months after aortic valve replacement.[17]

Echocardiographic markers of reverse remodeling

The first group of parameters that directly quantify improvement in hemodynamic conditions after aortic valve replacement includes aortic valve area (AVA), transaortic pressure gradients, and blood flow velocity across the prosthesis. An increase in AVA has been documented following both

Table 2 – Linear regression analysis of the association between iGOA of implanted valve prosthesis and changes in left atrial and left ventricular ECHO parameters (1-year postoperative minus preoperative)

	Coef.	95% CI		*p*
		Lower	Upper	
Δ LA diameter (mM)	–1.376	–5.703	2.951	0.53
Δ LVEF (%)	4.566	–0.513	9.645	0.078
Δ IVSD (mM)	–0.033	–1.694	1.629	0.969
Δ LAVi (mL/m²)	–14.114	–28.317	0.089	0.051
Δ E/avrg e´	–0.592	–3.721	2.537	0.708
Δ iAVA (cm²/m²)	**0.321**	**0.134**	**0.509**	**0.001**
Δ iLVeSD (mm/m²)	**–6.578**	**–13.071**	**–0.086**	**0.047**
Δ iLVeDD (mm/m²)	**–2.48**	**–4.815**	**–0.144**	**0.038**
Δ LVMi (g/m2)	–18.083	–41.461	–5.296	0.128

iAVA – indexed aortic valve area; iLVeDD – indexed left ventricular end-diastolic diameter; iLVeSD – indexed left ventricular end-systolic diameter; IVSD – interventricular septal diameter; LA – left atrium; LAVi – indexed left atrial volume; LVEF – left ventricular ejection fraction.

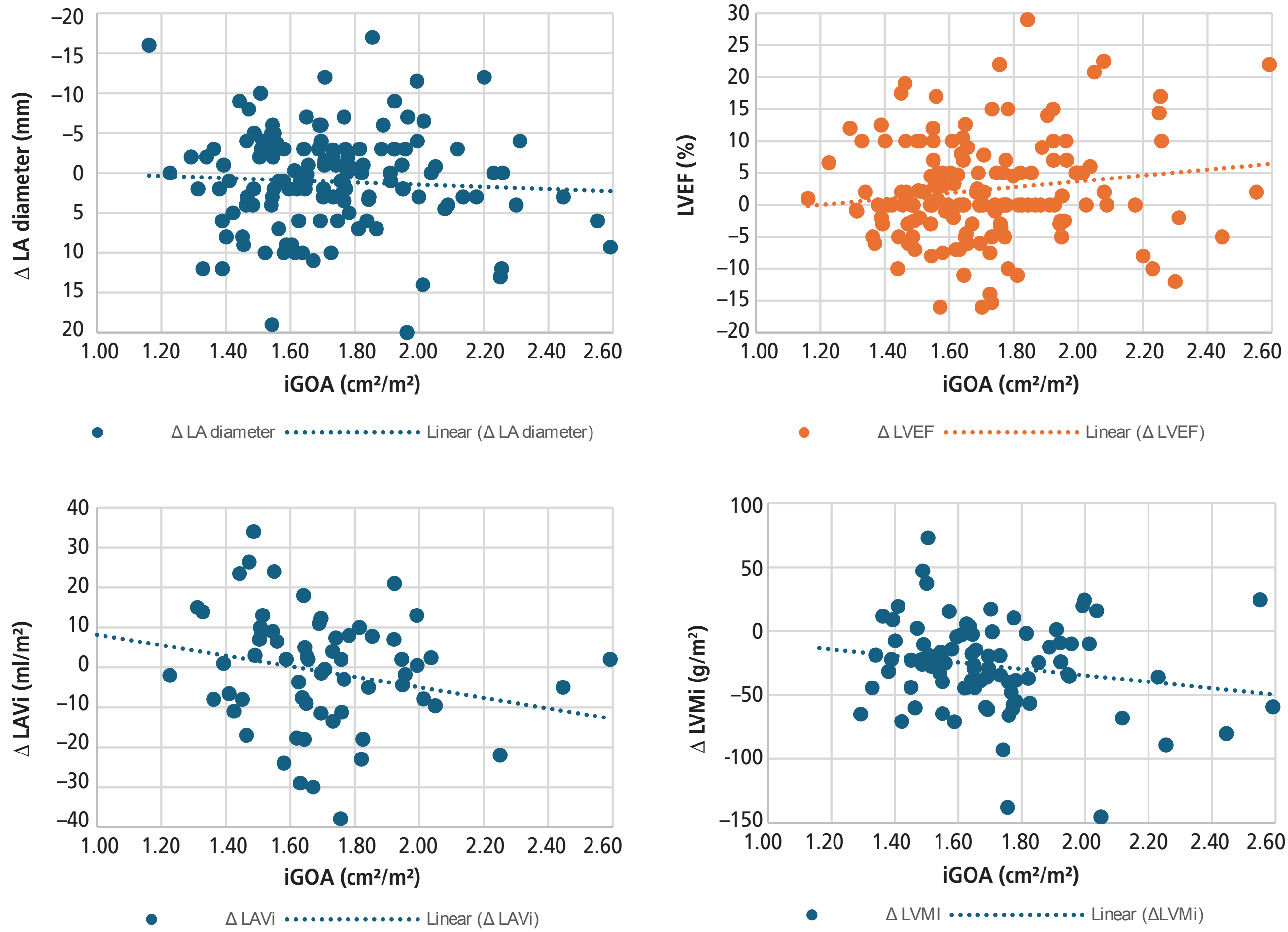

Fig. 4 – Scatter plots with linear regression lines showing the associations between prosthesis iGOA and 1-year changes in ΔLA diameter, ΔLVEF, ΔLAVI, and ΔLVMi. iGOA – indexed geometric orifice area; ΔLVMi – 1-year change in left ventricular mass index; ΔLA – 1-year change in left atrium; ΔLAVi – 1-year change in indexed left atrial volume; ΔLVEF – 1-year change in left ventricular ejection fraction.

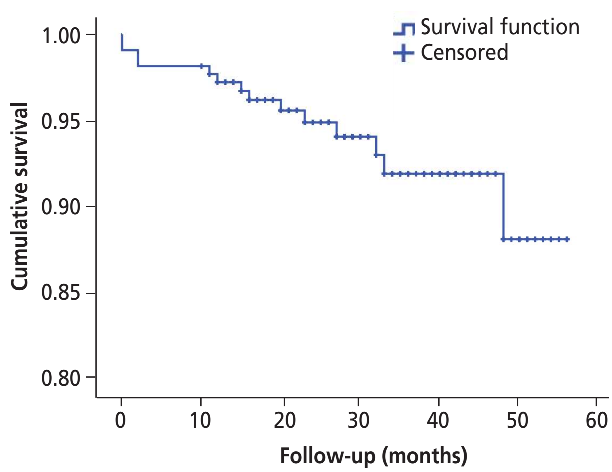

Fig. 5 – Kaplan–Meier analysis of survival with Cox regression analysis of the effect of iGOA on survival (HR = 1.138, 95% CI 0.012–1.649, *p* = 0.118).

surgical aortic valve replacement (SAVR) and transcatheter aortic valve implantation (TAVI). A meta-analysis including data from 27 studies reported a mean AVA increase of 0.99 cm² after TAVI and 1.19 cm² after SAVR, which is consistent with our findings of an increase in iAVA of 0.41 cm²/m² at a mean body surface area (BSA) of 1.98 ± 0.21 m² (unpublished data).[5] The above-mentioned meta-analysis further reported a mean reduction in the transaortic mean pressure gradient of 38.23 mmHg, which closely corresponds to the reduction of 37 mmHg observed in our study.[5] Even smaller studies focusing on reverse remodeling after transcatheter or surgical AVR have documented an increase in aortic valve area of 0.9–1.11 cm² and reductions in mean and peak transaortic gradients of 35–46 and 33–69 mmHg, respectively.[6,19,20] These findings are consistent with the results observed in our study.

Secondary parameters reflecting reverse left ventricular remodeling most commonly include LVEF and LVeDD. This pattern of remodeling is specifically associated with changes related to aortic stenosis and differs from the remodeling observed in other aortic valve pathologies.[21] A meta-analysis including 37 studies reported a mean increase in LVEF of 2.35% after AVR, independent of the time interval from surgery within a range of one month to one year, as well as a mean reduction in LVeDD of 1.78 mm, with the most pronounced decrease documented at six months after the procedure.[5] In the present study, a significant increase in LVEF of 2.44% and a reduction in indexed left ventricular end-diastolic diameter of 0.45 mm/m² at a mean BSA of 1.98 ± 0.21 m² were observed. Thus, our findings are consistent with the results of the meta-analysis, despite a slightly less pronounced reduction in LVeDD. In contrast, compared with the present study, smaller studies assessing patients as early as the first three months after TAVI did not document a positive effect on improvements in LVEF or LVeSD and LVeDD.[6,20] One of these studies also documented a reduction in IVSD of 1.4 mm, which was similar to that observed in our study.[6] Other parameters evaluated in some studies include left ventricular volume, for which a significant reduction has also been reported following AVR.[5] More recent studies have increasingly evaluated left ventricular global longitudinal strain, the change of which after aortic valve replacement appears to be a more sensitive predictor of long-term patient survival compared with LVEF.[6,16] Assessment of left ventricular hypertrophy and its reverse remodeling may be further influenced by comorbidities, particularly arterial hypertension, the effects of which on the left ventricle resemble those observed in aortic stenosis.[6] Some studies have differentiated left ventricular remodeling according to etiology, demonstrating that AS predominantly leads to a shift of the LV left–right axis with a decrease in short-axis eccentricity, whereas hypertension is more commonly associated with septal thickening. Furthermore, it has been documented that the left–right axis shift is not reversible following AVR.[2] Such complex assessments require prospective inclusion of these parameters, which was not feasible in the present retrospective analysis. Another group of parameters includes echocardiographic measures directly quantifying diastolic dysfunction. Among the most objective parameters of left ventricular diastolic dysfunction is the E/e′ ratio, with more recent data suggesting that averaged e′ provides greater diagnostic accuracy than assessment of septal or lateral e′ alone. In our study, this approach confirmed a favorable effect of aortic valve replacement on the E/e′ ratio, whereas not all previous studies have reported similar findings.[6]

Echocardiographic changes reflecting reverse left ventricular remodeling can be detected as early as one month after aortic valve replacement.[5] However, the temporal evolution of several ECHO parameters during the first year after surgery may be inconsistent. According to a meta-analysis focused on left ventricular remodeling after aortic valve replacement, a more pronounced reduction in LVM and LVeDD has been documented at six months compared with measurements obtained at three and twelve months following the procedure.[5] On the other hand, other authors have suggested that changes observed three months after TAVI may already be very similar to the final extent of reverse remodeling observed one year after the intervention.[6] However, their study did not demonstrate reductions in LVeSD and LVeDD, nor improvements in LVEF or the E/e′ ratio three months after TAVI.[6] In contrast, our study, which assessed ECHO parameters one year after SAVR, demonstrated a positive effect of surgical intervention across these parameters.

Prosthesis size and reverse remodeling

Patient–prosthesis mismatch (PPM) was first described in 1978 by Rahimtoola and represents an imbalance between the hemodynamic performance of the implanted prosthetic valve and the patient's cardiac output requirements. As outlined in the Introduction, severe PPM is typically defined by an indexed effective orifice area of less than 0.65 cm²/m².[10,11] In the presence of patient–prosthesis mismatch, the prosthetic valve functions normally but provides a relatively small effective orifice area in relation to the patient's body surface area, and this condition may occur after both surgical and transcatheter aortic valve replacement.[22] PARTNER studies that also evaluated patient–prosthesis mismatch identified only ECHO defined severe PPM as a risk factor for a higher incidence of primary endpoints, including death and hospitalization for heart failure. A statistically significant difference was demonstrated not only in comparison with no PPM (severe PPM vs. no PPM, p <0.001) but also in comparison with moderate PPM (severe PPM vs. moderate PPM, p = 0.02).[13] One smaller study even reported a higher incidence of reoperation in patients without PPM compared with those with moderate PPM (10-year cumulative incidence of reoperation: 8.3% for no PPM vs. 6.7% for moderate PPM).[12] Previous studies have more frequently focused on the impact of PPM on postoperative outcomes rather than on the effect of absolute prosthetic valve size or iGOA beyond the threshold of PPM. In contrast to the aforementioned lower risk of reoperation observed in patients with moderate PPM compared with those with larger aortic valve area, functional outcomes appear less favorable. A smaller increase in LVEF has been reported in patients with PPM compared with those without PPM one year after TAVI (1.9 ± 21.3% vs. 8.2 ± 30.1%, p = 0.045). In addition, a statistically significant effect on left ventricle global longitudinal strain has been documented.[16] The presence of patient–prosthesis mismatch is associated with worse survival after aortic valve replacement (hazard ratio 3.56, 95% CI 1.37–9.25; p = 0.009), a finding that is widely accepted and well documented in the scientific literature.[14] In the present cohort, however, only 4 of 216 patients exhibited a postoperative aortic valve area measured by ECHO ≤0.85 cm², and thus 98% of patients did not meet the criteria for PPM (unpublished data). Consequently, the present study focuses on evaluating the effect of iGOA above the PPM threshold. Direct comparison with studies primarily addressing PPM may therefore be limited and should be interpreted with caution. In our previous study, we demonstrated that implantation of a larger bioprosthetic valve was associated with better survival compared with a smaller bioprosthetic valve, even beyond the thresholds defining patient–prosthesis mismatch. In contrast, this association was not observed for mechanical prostheses.[15] The literature indicates that patient–prosthesis mismatch is associated with a 2.5-fold increase in the risk of structural valve deterioration.[11,23] This may subsequently adversely affect patient survival even in the absence of an impact on early postoperative reverse left ventricular remodeling. It therefore remained unclear whether smaller prosthetic valves lead to insufficient reverse myocardial remodeling or whether they are more prone to structural valve deterioration with subsequent recurrence of aortic stenosis. Our results demonstrate that, in the absence of PPM, larger indexed geometric orifice area of the implanted prosthesis is associated with greater improvement in parameters of RLVR, particularly LVeSD and LVeDD, and borderline improvement in LVEF, compared with smaller iGOA. Based on this favorable remodeling effect, a subsequent beneficial impact on the incidence of heart failure and long-term survival may be anticipated, despite the absence of a detectable effect on short-term survival in the present study.

Conclusion

ECHO assessment performed one year after AVR for AS demonstrated a statistically significant improvement in iAVA, mean and peak transaortic pressure gradients, and a reduction in aortic valve jet velocity. The observed changes further indicate the presence of reverse left ventricular remodeling, documented by a statistically significant increase in LVEF and reductions in IVSD, iLVeSD, iLVeDD, and LVMi. Improvements in left atrial parameters were also observed, reflected by a reduction in left atrial diameter, along with improved left ventricular diastolic filling as indicated by a statistically significant decrease in the E/e′ ratio.

The present study confirms a beneficial effect of implantation of a larger valve prosthesis on more pronounced improvements in several left heart parameters, particularly iLVeSD and iLVeDD, with borderline but not statistically significant effects on LVEF and LAVi. Similarly, a nonsignificant trend toward greater LVMi reduction was observed with larger iGOA. However, no positive effect of larger prosthetic valve implantation on short-term patient survival was demonstrated.

Conflict of interest

The authors declare that there are no financial or personal relationships that could inappropriately influence (bias) the work reported in this manuscript. The authors confirm that they have no conflicts of interest relevant to the content of this article.

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Ethical statement

The processing of the retrospective study was approved by the Ethics Committee in compliance with the principles of the Declaration of Helsinki and ICH Guidelines for Good Clinical Practice and applicable regulatory requirements (protocol code: A2082025 and date of approval: August 22. 2025).

Informed consent

During hospitalization, patients provided written consent for the healthcare received. According to the recommendation of the Ethics Committee, due to the retrospective nature of the study, no further patient consents were required.

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