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Research Article

Predictors of Left Ventricular Mass Index One Year after Bioprosthetic Aortic Valve Replacement for Aortic Stenosis

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*Background and Aim of Study.*To evaluate predictors of residual left ventricular hypertrophy (LVH) one year after surgical aortic valve replacement (SAVR) in patients with aortic stenosis and clarify the relationship between long-term outcomes and predictors. *Methods*. We retrospectively reviewed 141 patients who underwent SAVR with a bioprosthetic valve. Left ventricular dimensions and mass index were assessed using serial transthoracic echocardiography. The difference in time course and the pattern of left ventricular mass index (LVMI) regression between patients with and without residual LVH one year after surgery were evaluated. The factors associated with LVMI one year after SAVR and the prognostic impact of these predictors on long-term outcomes were analyzed. *Results*. Although LVMI one year after surgery showed a significant decrease in patients with and without LVH, greater preoperative LVMI and lesser extent of LVMI decrease resulted in high residual LVMI at one year after SAVR in patients with LVH. The preoperative left ventricular end-diastolic dimension index ($p = 0.027$) and preoperative left atrial dimension ($p = 0.001$) were significant determinants of LVMI at one year after SAVR. A cut-off value of 30 mm/m^2 or greater for the left ventricular end-diastolic dimension index was optimal for predicting high LVMI one year after SAVR. Overall survival was significantly lower with a left ventricular end-diastolic dimension index ≥30 mm/m^2 ($p = 0.017$, Log rank). *Conclusions*. High preoperative left ventricular end-diastolic dimension index and large left atrial dimension were associated with high LVMI one year after surgical aortic valve replacement. Preoperative left ventricular end-diastolic dimension index of >30 mm/m^2 could predict adverse outcomes after surgical aortic valve replacement.

1. Introduction

Aortic stenosis (AS) causes left ventricular hypertrophy (LVH), which leads to increased LV mass (LVM) in responce to a pressure overload. Unloading the left ventricle with surgical aortic valve replacement (SAVR) promotes LVM regression. LVH is not uniform in patients with similar degrees of AS, and its regression after surgical correction varies among individuals [1, 2]. LVM regression after SAVR or transcatheter aortic valve implantation (TAVI) leads to improved clinical outcomes [3, 4], and the negative impact of persistent LVH on long-term outcomes has been reported [5, 6]. Surgical intervention for severe AS is indicated by valve narrowing, symptoms, and impaired LV ejection fraction [7, 8]. These factors do not necessarily represent the preoperative maladaptive changes of the left ventricle, including inappropriate hypertrophy, fibrosis, and dilatation [9]. Previous studies have shown that myocardial fibrosis could predict wrong reverse remodeling and worse prognosis after SAVR for aortic stenosis [6, 9–11]. In other words, the evaluation of LVM regression could add information on underlying myocardial damage that has been neglected in the current guidelines and lead to better outcomes after AVR.

This study aimed to evaluate the preoperative predictors of residual LVH one year after SAVR and the prognostic

impact of these predictors on long-term outcomes. Furthermore, we compared the difference in the degree and pattern of LVM regression between patients with and without LVH one year after SAVR.

2. Materials and Methods

2.1. Ethical Statement. The study was approved by the Institutional Ethics Committee of the Kyoto First Red Cross Hospital (ERB). The ethics committee of this retrospective study waived the requirement for individual consent.

2.2. Patients and Study Design. In this retrospective study, we performed a nonrandomized review of patients who underwent SAVR for AS using a bioprosthetic valve at our institution between January 2008 and June 2021. The exclusion criteria were concomitant mitral procedure, emergent surgery, and infective endocarditis. Baseline clinical data and procedural characteristics at SAVR were collected from medical charts and operation records. The study's primary endpoints included the time course and degree of LVM regression and the factors associated with the LVM index (LVMI) one year after SAVR. In addition, the study patients were dichotomized into two groups according to the optimal cut-off value that distinguished patients with and without LVH one year after surgery. According to the American Society of Echocardiography Recommendations, LVH was defined as an LVMI greater than 95 g/m^2 for females and greater than 115 g/m^2 for males. The secondary endpoint was the incidence of postoperative all-cause mortality within the study period.

2.3. Operative Technique. All surgeries were performed using mild hypothermic cardiopulmonary bypass with intermittent antegrade and retrograde cold blood cardioplegia. The decision to perform concomitant procedures such as coronary artery bypass grafting (CABG) or tricuspid repair was made at the discretion of the attending surgeon. In addition, the size of the prosthesis was determined by the surgeon during surgery.

2.4. Echocardiography. All patients were examined using transthoracic echocardiography preoperatively (within one month before SAVR), at 1 and 12 months postoperatively, and annually after that. The LV end-diastolic (LVEDd) and end-systolic diameters (LVESd), LV ejection fraction (EF), left atrial dimension (LAD), diastolic interventricular septal wall thickness (IVSTd), and diastolic LV posterior wall thickness (PWTd) were measured. LVEF was calculated using the modified Simpson's method. LVM was estimated by LVEDd and wall thickness at end diastole. LVM was calculated using the following formula: LVM (g) = 0.8 × 1.04[(LVEDd) + (IVSTd) + (PW Td)] [3]-LVEDd [3])] g + 0.6 [12]. LVMI was calculated from LVM and body surface area (BSA) using the formula: LVMI = LVM/BSA. Transaortic valve velocity was also measured preoperatively and at follow-up echocardiography.

2.5. Follow-up Data Collection. Postoperative clinical data, including postoperative complications and mortality, were obtained from the patient's medical records, outpatient clinics, and contact with the patient's physicians. Thirty-day mortality was defined as mortality within 30 days regardless of hospital discharge. Operative mortality was defined as mortality within 30 days or death before discharge. In addition, the incidence of postoperative all-cause mortality during the study period was evaluated.

2.6. Statistical Analysis. Categorical variables were reported as frequencies and percentages. Continuous data are expressed as mean ± SD or median with ranges. Patients were classified into two groups depending on whether they had complete regression of hypertrophy (non-LVH group), defined as LVMI <95 g/m^2 for women and <115 g/m^2 for men at one year postoperatively, or incomplete regression of hypertrophy (LVH group). Changes in echocardiographic variables over time were analyzed using a mixed-effects model for repeated measures, including factors for the group, time, and the interaction between group and time. The patient was treated as a random effect at each time point, whereas the assessment time points were treated as categorical factors. We used a univariate linear regression model with LVMI one year after AVR as the dependent variable and other measurements as independent variables. Variables considered in the univariate linear regression model included age at operation, sex, preoperative BSA, preoperative New York Heart Association (NYHA) functional class, preoperative LVEDd index, preoperative LVESd index, preoperative LVEF, preoperative LAD, preoperative LVMI, preoperative V_{max}, preoperative MR grade, preoperative TR grade, preoperative tricuspid valve pressure gradient across the tricuspid regurgitation peak gradient, and indexed prosthetic valve size. In addition, potential risk factors (p value <0.05) in the univariate analysis were included in the multivariate analysis.

Receiver operating characteristic (ROC) analysis was performed, and the area under the ROC curve was used to determine the optimal cut-off value for predicting the complete regression of LVH. The optimal cut-off value of the parameters, defined as the value that most accurately distinguished between patients with and without complete regression of hypertrophy, was examined.

The Kaplan–Meier method was used to estimate survival for the study patients, and comparisons between the two patient groups that were dichotomized according to this cut-off point were performed using the log-rank test.

Statistical analyses were performed using JMP (version 16.0; SAS Institute, Inc., Cary, USA) and SPSS (version 21.0; IBM Corp., Armonk, NY, USA). All statistical tests were two-sided, and statistical significance was set at $p < 0.05$.

3. Results

3.1. Patients' Characteristics/Operative Details. Preoperative and operative patient characteristics are listed in Tables 1 and 2, respectively. NYHA class ≥3 heart failure occurred in 29 patients (21%). Comorbidities included

Table 1: Preoperative patient characteristics.

Age (years)	77 ± 7
Male gender, *n* (%)	68 (48)
Body surface area (m^2)	1.5 ± 0.2
NYHA class, n (%)	
≤2	112 (79)
3	25 (18)
4	4 (3)
Previous cardiac surgery	1 (1)
Previous PCI	17 (12)
Hypertension	89 (63)
Hyperlipidaemia	63 (45)
Diabetes mellitus	43 (30)
Hemodialysis	29 (21)
PVD	3 (2)
CVD	7 (5)
Atrial fibrillation	23 (16)
Transthoracic echocardiographic data	
LVEDd (mm)	44 ± 7
LVEDs (mm)	28 ± 7
LVEDd index (mm/m^2)	30 ± 4
LVEDs index (mm/m^2)	19 ± 4
LVEF (%)	66 ± 13
LAD (mm)	42 ± 7
LVM	212 ± 73
LVMI (g/m^2)	141 ± 48
V Max (m/sec)	4.7 ± 0.9
MR grade	0.8 ± 0.7
TR grade	0.8 ± 0.9
TRPG (mmHg)	29 ± 13

Data are presented as numbers (percentages) or means ± standard deviation. CVD: cerebral vascular disease; LAD: left atrial dimension; LVEDd: left ventricular end-diastolic diameter; LVEDs: left ventricular end-systolic diameter; LVEF: left ventricular ejection fraction; LVMI: left ventricular mass index; MR: mitral regurgitation; NYHA : New York heart association; PCI: percutaneous coronary intervention; PVD: peripheral vascular disease; TR: tricuspid regurgitation; TRPG: tricuspid valve pressure gradient across the tricuspid regurgitation peak gradient; V_{max}: maximum velocity and the aortic valve maximum jet velocity.

hypertension in 89 (63%) patients, diabetes mellitus in 43 (30%), current hemodialysis in 29 (21%), peripheral vascular disease in 3 (2%), and atrial fibrillation in 23 (16%).

Concomitant procedures were performed in 63 patients (45%), including intervention for the tricuspid valve in 8 patients (6%), coronary artery bypass grafting in 46 (33%), and Maze procedure in 23 (16%). SAVR was usually performed via a median full sternotomy, while a minimally invasive cardiac surgery approach was selected in 3 patients (2%). The mean prosthesis size was 22.0 ± 2.2 mm, and the median prosthesis size was 21 mm. The prosthesis size distribution is presented in Table 2. After 21 mm, the frequencies of the 23- and 25-mm devices were similar.

3.2. Clinical Outcomes. The 30-day and in-hospital mortality rates were 1.4% ($n = 2$) and 2.8% ($n = 4$), respectively. Causes of death included sepsis ($n = 1$), lung hemorrhage ($n = 1$), nonobstructive mesenchymal ischemia ($n = 1$), and pneumonia ($n = 1$). During the follow-up period of 43 ± 36 months, another 26 patients died, including seven who died of cardiac conditions (myocardial infarction, 1 case; heart failure, 1 case; prosthetic valve endocarditis, 2 cases; and sudden death, 3 cases), 17 of noncardiac conditions (pneumonia, 6 cases; sepsis, 4 cases; acute pancreatitis, 1 case; intracranial hemorrhage, 3 cases; and cancer, 3 cases), and 2 of unknown causes. The 5- and 10-year survival rates were 74% and 62%, respectively (Figure 1(a)).

Table 2: Operative details.

AVR prosthetic valve size	
19 mm	26 (18)
21 mm	54 (38)
23 mm	29 (21)
25 mm	28 (20)
27 mm	4 (3)
Indexed prosthetic valve size (mm/m^2)	14.7 ± 1.5
CABG	46 (33)
TAP	8 (6)
Maze/PVI	23 (16)
CPB time (min)	196 ± 68
ACC time (min)	120 ± 33
MICS	3 (2)

Data are presented as numbers (percentages) or means ± standard deviation. ACC: aortic cross-clamp; AVR: aortic valve replacement; CABG : Coronary artery bypass grafting; CPB: cardiopulmonary bypass; MICS: minimally invasive cardiac surgery; PVI: pulmonary vein isolation; TAP: tricuspid valve annuloplasty.

3.3. Ventricular and Atrial Remodeling. Among the 141 patients, 12 died within one year of SAVR. A total of 93 patients underwent LVM evaluation one year after AVR. Overall, a significant reduction in LVMI was observed early after SAVR (preoperative versus postoperative: LVMI, 141 ± 44 vs. 102 ± 30 ml/m^2; $p < 0.001$); the reduction continued over time but not beyond one year after SAVR. Thus, the one-year follow-up time point was chosen for our analysis to identify the determinants of the LVM regression. We examined the extent of changes in LVMI between patients with and without LVH one year after AVR. The LVH group consisted of 43 patients (46%), and the non-LVH group consisted of 50 patients (54%). LVMI one year after SAVR significantly decreased in both groups (time effect: $p < 0.001$). Preoperative LVMI was significantly lower in the non-LVH group; these differences extend to one year after SAVR (group effect: $p = 0.002$) (Figure 2(a)). A significant decrease was observed in IVSTd and PWTd in both groups one year after SAVR (time effect IVSTd : $p < 0.001$; PWTd : $p < 0.001$) (Figures 2(c) and 2(d)), and a significant decrease in LVEDd (time effect: $p < 0.001$) (Figure 2(b)). Patients in the non-LVH group demonstrated a greater decrease in IVSTd and PWTd (interaction effect: IVSTd, $p = 0.018$; PWTd, $p = 0.008$), whereas the decrease in LVEDd was similar (interaction effect: $p = 0.038$). As a result, a greater extent of LVMI decrease was seen in patients without hypertrophy, although the difference between the groups did not reach statistical significance (interaction effect: $p = 0.058$). LAD was consistently higher in both preoperative and one-year follow-ups in patients with hypertrophy (group effect: $p = 0.002$), and a significant decrease in LAD was observed in both groups to the same degree (time effect: $p = .003$; interaction effect: $p = 0.295$) (Figure 2(e)).

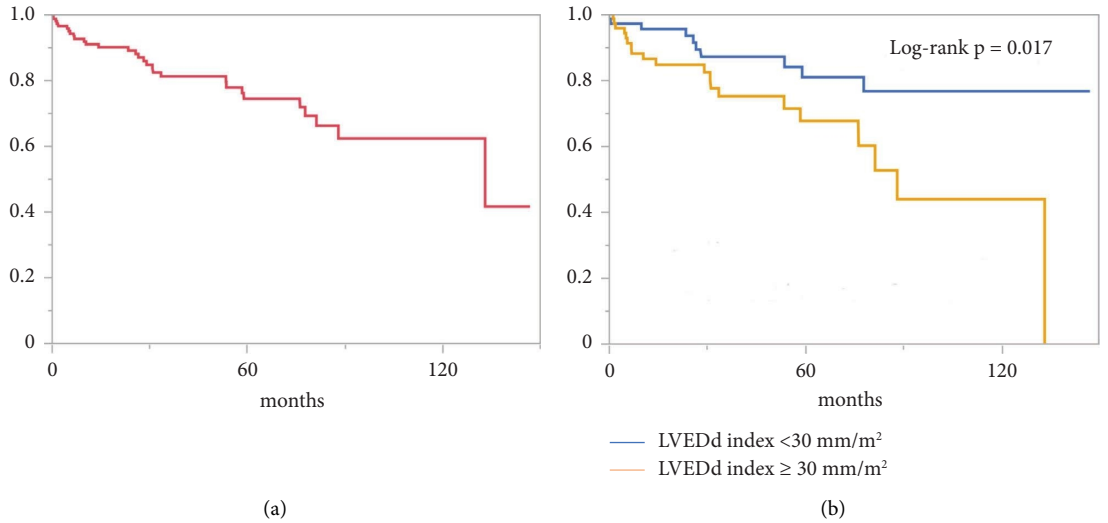

Figure 1: Kaplan–Meier curve showing freedom from all-cause death in all patients (a), divided by preoperative left ventricular end-diastolic diameter (LVEDd) index; (b) Group 1, preoperative LVEDd index <30 mm/m^2, and Group 2, preoperative LVEDd index ≥30 mm/m^2.

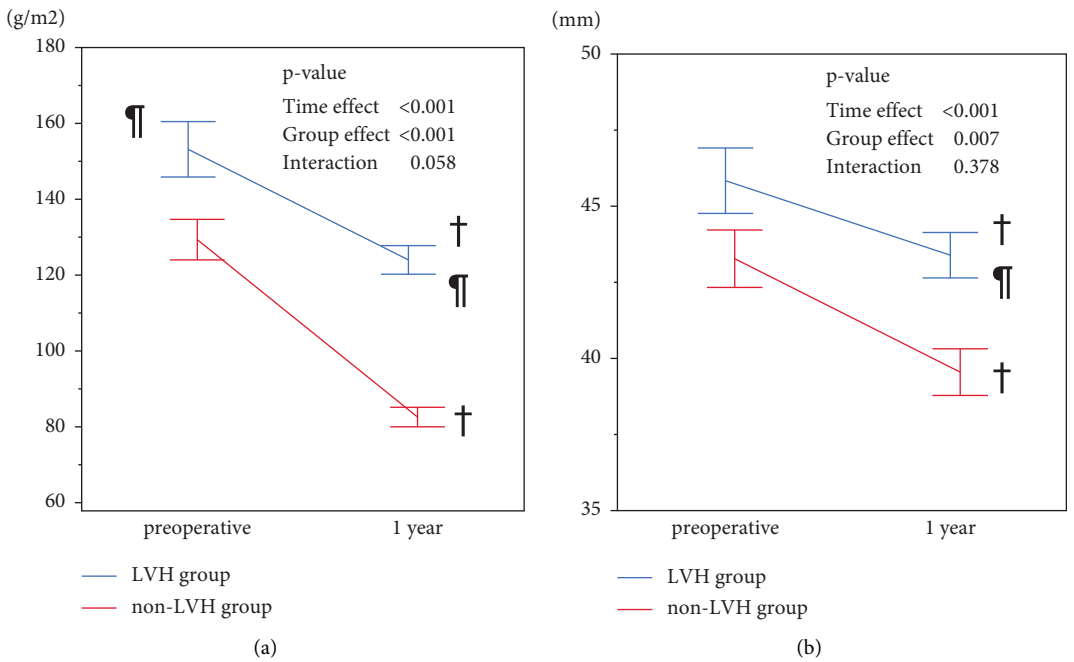

Figure 2: Continued.

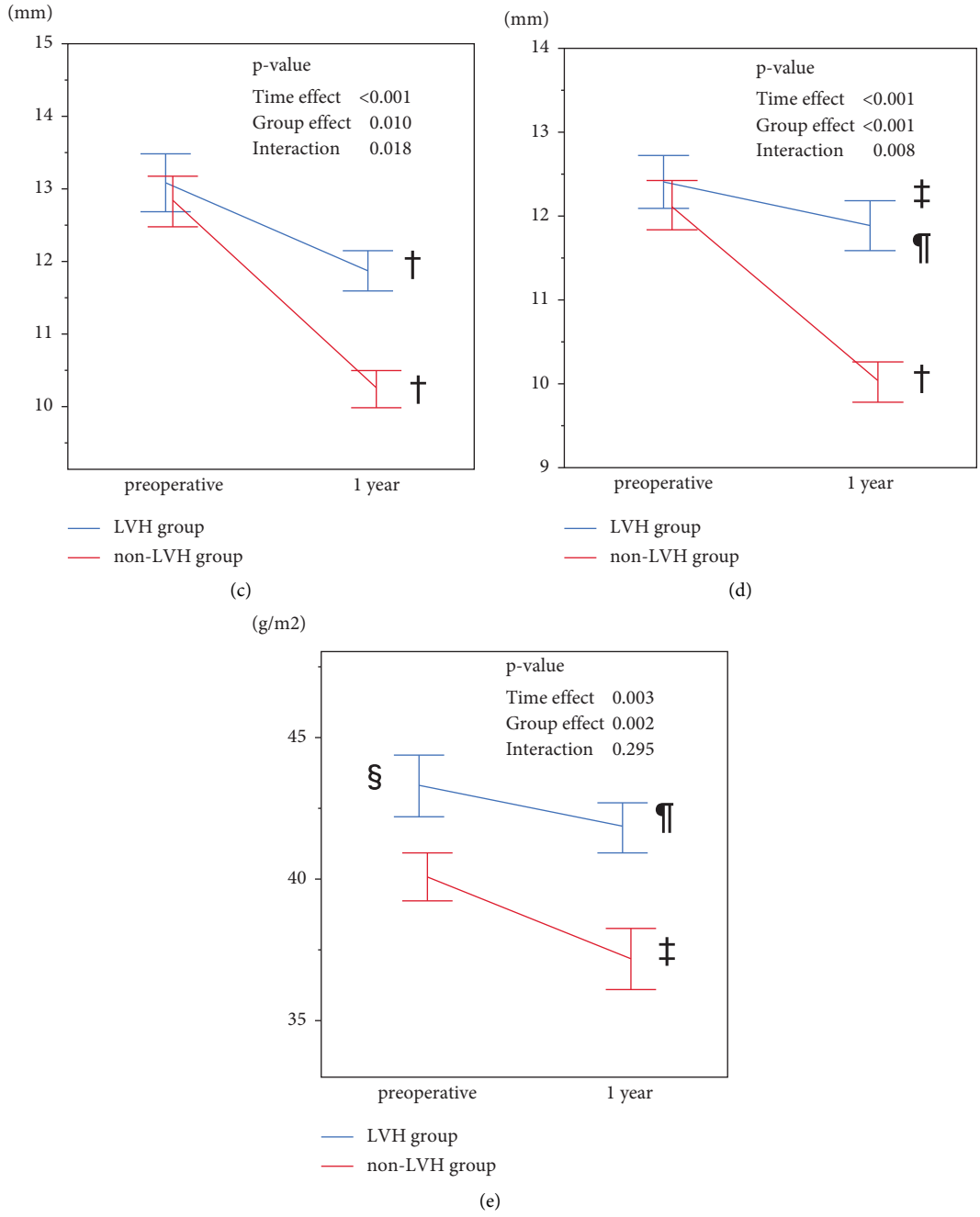

FIGURE 2: Longitudinal changes in the left ventricular mass index (LVM index, Figure 2(a), left ventricular end-diastolic diameter (LVEDd, Figure 2(b), diastolic interventricular septal wall thickness (IVSTd, Figure 2(c), diastolic left ventricular posterior wall thickness (PWTd, Figure 2(d), and left atrial diameter (LAD, Figure 2(e), divided by whether patients had complete regression of hypertrophy (non-LVH group) or not (LVH group). Error bars represent standard error. $^{\dagger}p < 0.01$ versus preop, $^{\ddagger}p < 0.05$ versus preop, $^{\P}p < 0.01$ versus non-LVH group, and $^{\S}p < 0.05$ versus non-LVH group.

3.4. Predictors of LVMI at One Year after AVR. Univariate linear regression analysis showed that preoperative LVEDd index, preoperative LVMI, preoperative MR grade, preoperative LAD, and indexed prosthetic valve size were significant determinants of LVMI one year after SAVR (Table 3). Multivariate analysis based on stepwise multiple linear regression analysis was implemented, and the final model, which included two variables (preoperative LVEDd index and preoperative LAD), was derived to discover the significant determinants of LVMI at one year after SAVR. Multivariate linear regression analysis showed that the preoperative LVEDd index (β coefficient, 0.29, $p = 0.027$) and preoperative LAD (β coefficient, 0.35, $p = 0.001$) were significant determinants of % LVMI one year after SAVR (Table 3). Receiver operating characteristic curve analysis revealed that a cut-off value of 30 mm/m^2 or

Table 3: Univariate and multivariate predictors for LVM index at one-year.

	β	p value	β	p value
Age at operation	0.133	0.209		
Male sex	−2.3	0.713		
Body surface area	−0.02	0.862		
Preop NYHA	0.03	0.772		
Preop LVEDd index	0.448	<0.001	2.53	<0.001
Preop LVEDs index	0.36	<0.001		
Preop LVEF	−0.198	0.06		
Preop LAD	0.365	<0.001	1.363	0.001
Preop LVMI	0.359	<0.001		
Preop V max	−0.09	0.398		
Preop MR grade	0.248	0.018		
Preop TR grade	0.244	0.19		
Preop TRPG	0.244	0.052		
Indexed valve size	0.217	0.038		

LAD: left atrial dimension; LVEDd: left ventricular end-diastolic diameter; LVEDs: left ventricular end-systolic diameter; LVEF: left ventricular ejection fraction; LVMI: left ventricular mass index; MR: mitral regurgitation; NYHA : New York heart association; TR: tricuspid regurgitation; TRPG: tricuspid valve pressure gradient across the tricuspid regurgitation peak gradient; V_{max}: maximum velocity and the aortic valve maximum jet velocity.

greater LVEDd index was optimal for predicting high LVMI one year after SAVR, with a sensitivity and specificity of 72% and 74%, respectively (Figure 3). The area under the curve was 0.72 (95% CI, 0.62 to 0.83).

Kaplan–Meier curve analysis showed that overall survival was significantly lower in patients with an LVEDd index ≥30 mm/m^2 than in patients with an LVEDd index <30 mm/m^2 (log-rank: $p = 0.017$; Figure 1(b)).

4. Discussion

The main finding of this study was that approximately half of the patients had residual LVH one year after successful SAVR. The greater extent of LVMI decrease was mainly driven by the decline in IVSTd and PWTd in patients without hypertrophy. A preoperative LVEDd index of >30 mm/m^2 was associated with a high LVMI. In addition, this cut-off point may be a valuable predictor of postoperative all-cause mortality.

Pressure overload in patients with aortic stenosis increases the LV mass, a measure of ventricular hypertrophy in AS. SAVR relieved pressure overload, and the left ventricular mass decreased immediately after surgery. Previous studies have examined the extent of myocardial reverse remodeling after afterload reduction and showed that residual LVH after SAVR or TAVI negatively impacts long-term survival and rehospitalization for heart failure [1–4].

The degree of postoperative LV reverse remodeling may reflect the underlying myocardial damage, which is difficult to estimate from preoperative parameters alone [6, 9–11]. Izumi et al. [5] studied the importance of LV reverse remodeling and evaluated it by follow-up echocardiography. They concluded that echocardiographic parameters one year after SAVR are more significant predictors of long-term outcomes than preoperative parameters.

Kadkhodayan et al. [13] reported that the immediate reduction in LVM calculated after SAVR is likely due to the LVM formula in which LVEDd is a significant component rather than the actual regression of LVM. They also

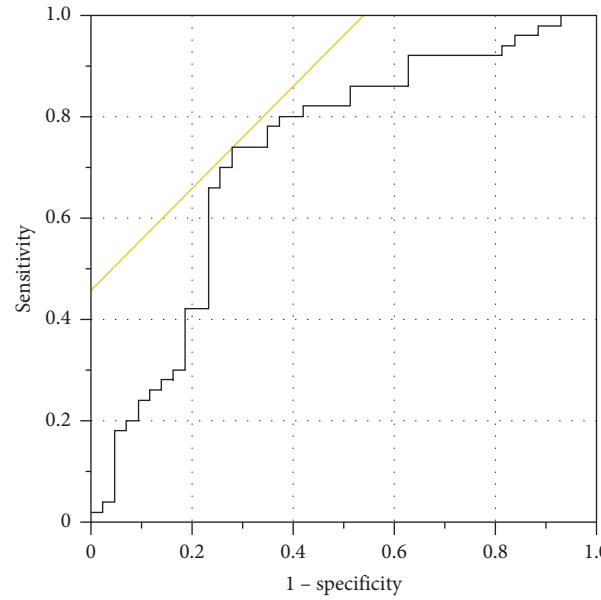

Figure 3: Receiver-operating characteristic curve A receiver-operating characteristic curve (ROC) of the preoperative left ventricular end-diastolic diameter index used to predict residual hypertrophy one-year after surgical aortic valve replacement (SAVR) for aortic stenosis (AS).

mentioned that the more substantial reduction in LVM immediately after SAVR was due to a smaller postoperative LVEDd and was associated with significantly reduced stroke volume, which led to a higher 30-day mortality trend in patients with a more significant LVM reduction. This might be one of the pitfalls of this formula, for its over-dependency on LVEDd, and the difference in LVM regression between patients with and without hypertrophy one year after SAVR could be solely explained by the difference in LVEDd. In our study, we showed that the decrease in IVSTd and PWTd was more prominent in patients without hypertrophy than in those with hypertrophy at one year, while the decline in LVEDd at one year did not differ between the patients in

each group. Therefore, the more significant change in LVMI in patients without hypertrophy was not solely dependent on the decrease in LVEDd, but rather on the decline in IVSTd and PWTd. A previous study suggested that only a high preoperative LVM and not the extent of LVM regression determine the clinical outcomes. Weissler-Snir et al. [14] reported that patients with a higher baseline LVM demonstrated more significant LVM regression six months after transcatheter aortic valve implantation. An improvement in NYHA functional class was observed similarly in the entire spectrum of LVMI regression.

In our data, the absolute LVMI decreased by the same extent in both groups; however, in patients with excessive preoperative hypertrophy, the LVMI remained more significant than the upper limit of normal. These data, including ours, suggest that residual LVH after the intervention is strongly associated with worse clinical outcomes than the degree of LVM regression. There seems to be a threshold for myocardial hypertrophy beyond which histological and functional normalization becomes impossible. Once this threshold has been reached, LVM regression occurs insufficiently, which may result in poor outcomes, even after successful SAVR. Our data suggest that a preoperative LVEDd index of >30 mm/m predicts high LVMI one year after SAVR and all-cause mortality; thus, it could be, at least in part, the preoperative indicator of severe myocardial damage in AS patients.

Patients with AS also have left ventricular diastolic dysfunction [6, 15–17]. Hess et al. [16] reported that moderate-to-severe diastolic dysfunction in patients with aortic stenosis was an independent predictor of late mortality after valve replacement. Previous reports have shown a close relationship between left atrial (LA) dilatation and LVH in AS patients. Preoperative LA dilatation, reflecting long-standing disease and diastolic dysfunction severity, was associated with more significant LVH and adverse outcomes after SAVR. Beach et al. [18] suggested that the LA diameter did not decrease after SAVR, even after LV reverse remodeling. They also concluded that a preoperative dilated left atrium reduced long-term survival, independent of symptom status. Izumi et al. [(5)] reported that the freedom rate from a composite of cardiac death or hospitalization due to heart failure was higher in patients with postoperative LAD ≥40 mm than in those with LAD <40 mm. Their findings were consistent with our present study, in which preoperative LA dilatation predicted lesser LVM regression after SAVR. Preoperative diastolic dysfunction, reflected by the left atrial diameter, may affect postoperative LVM regression after AVR.

Current guidelines for managing AS decided on intervention indications with valve severity, symptoms, and reduced LVEF as the primary gatekeepers [7, 8]. They do not focus on LV remodeling parameters, such as LVEDd, LAD, and LVMI. These factors could indicate irreversible myocardial damage [19], adding a residual risk to patients after SAVR.

Our study had several limitations. First, this was a retrospective study conducted at a single institution and may be susceptible to various sources of bias. For example, the number of patients was small, the study group was heterogeneous, and additional procedures may have modified the effects of SAVR. We excluded patients who had undergone concomitant mitral valve surgery to minimize these effects. In addition, we only included patients who received bioprosthetic valves. Second, serial echocardiographic examinations were available for a limited number of patients, as follow-up echocardiography was not routinely performed at our institution. Third, we did not have accurate information about the patient's medications and standard disease status, such as hypertension. For example, LVM regression can be influenced by systemic hypertension and postoperative medication use.

5. Conclusion

A high preoperative LVEDd index and large LA diameter were associated with a high LVMI one year after SAVR. In addition, a preoperative LVEDd index of >30 mm/m^2 could predict worse outcomes after SAVR. Considering LV remodeling parameters such as LVEDd index, LAD, and LVMI to indicate valve intervention could improve the outcomes after SAVR in patients with AS.

Data Availability

The Excel data used to support the findings of this study are available from the corresponding author upon request.

Ethical Approval

The study was approved by the Institutional Ethics Committee of the Kyoto First Red Cross Hospital.

Consent

The Ethics Committee of the Kyoto First Red Cross Hospital waived the requirement for individual patient consent.

Conflicts of Interest

The authors declare that there are no conflicts of interest.

Authors' Contributions

Sachiko Yamazaki conceptualized the study, curated data, performed formal analysis, collected methodology, and wrote original draft; Kazunari Okawa curated data; Keisuke Shunto curated data; Katsuhiko Oka curated data; Koki Ikemoto curated data; Akiyuki Takahashi conceptualized the study, curated data, collected methodology, and performed supervision and surgery.

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