

Impact of Left Ventricular Mass Index on Early Outcomes After Aortic Valve Replacement with Sutureless Bioprosthetic Valve: A Comparison of Two Decades



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ABSTRACT

Introduction: The objective of the present study was to elucidate whether high left ventricular mass index (LVMI) affects early outcomes after sutureless bioprosthetic aortic valve replacement (AVR) in aortic stenosis (AS).

Patients and Methods: Postoperative early outcomes of 60 high-risk patients with aortic valve stenosis after replacement with sutureless bioprosthetic valve were retrospectively analyzed. Patients were grouped into two depending on LVMI. Left ventricular (LV) mass was calculated using the Devereux formula and indexed to the body surface area. High LVMI was defined as LVMI > 134 g/m² for males and LVMI > 100 g/m² for females. Early outcomes of surgery were compared between the normal and high LVMI patient groups.

Results: Preoperative patient characteristics were similar between the groups. Early mortality was 8.3%. There was no statistically significant difference between the groups with respect to postoperative early complication rates and mortality. LVMI decreased from 114.7 ± 13.7 g/m² at baseline to 109 ± 32.2 g/m² at follow-up in group I (p= 0.60) and from 192.5 ± 31.9 g/m² at baseline to 117.9 ± 25.2 g/m² in group II (p< 0.001).

Conclusion: The impact of high LVMI on morbidity and mortality after AVR with sutureless bioprosthetic valve was not deleterious in patients with isolated AS. Significant reduction in LVMI at 6 months is encouraging for these high-risk patients with severe LV hypertrophy; however, long-term follow-up is required.

Key Words: Aortic stenosis; left ventricular hypertrophy; heart valve prosthesis

Sol Ventrikül Kitle İndeksinin Dikişsiz Biyoprotez Kapak ile Aort Kapak Replasmanı Sonrası Erken Dönem Sonuçlara Etkisi; İki On Yıllık Karşılaştırılması

ÖZET

Giriş: Çalışmanın amacı artmış sol ventrikül kitle indeksinin, aort darlığı nedeniyle dikişsiz biyoprotez kapak ile aort kapak replasmanı sonrası erken dönem sonuçlara etkisinin araştırılmasıdır.

Hastalar ve Yöntem: Aort darlığı nedeniyle dikişsiz biyoprotez kapak ile aort kapak replasmanı uygulanan 60 yüksek riskli hastanın operasyon sonrası erken dönem sonuçları retrospektif olarak analiz edilmiştir. Hastalar sol ventrikül kitle indeksi değerlerine göre iki gruba ayrılmıştır. Sol ventrikül kitle indeksi Devereux formula ile hesaplanıp, vücut yüzey alanı ile indekslenmiştir. Erkek hastalar için > 134 g/m², kadın hastalar için > 100 g/m² yüksek olarak tanımlanmıştır. Cerrahi sonrası erken dönem sonuçlar iki hasta grubunda karşılaştırmalı olarak değerlendirilmiştir.

Bulgular: Operasyon öncesi hasta özellikleri gruplar arasında benzer bulunmuştur. Erken mortalite %8,3'tür. Operasyon sonrası erken dönem mortalite ve morbidite açısından gruplar arasında anlamlı fark tespit edilmiştir. Sol ventrikül kitle indeksi birinci grupta 114.7 ± 13.7 g/m² den 109 ± 32.2 g/m² ye (p= 0.60), yüksek kitle indeksi olan ikinci grup hastalarında 192.5 ± 31.9 g/m² bazal değerinden 117.9 ± 25.2 g/m² ye gerilemiştir (p< 0.001).

Sonuç: İzole aort darlığında operasyon öncesi artmış sol ventrikül kitle indeksinin, dikişsiz biyoprotez kapak ile aort kapak replasmanı sonrası erken dönem morbidite ve mortalite üzerine olumsuz etkisi tespit edilmiştir. Ciddi sol ventrikül hipertrofisi olan yüksek riskli bu hasta grubunda altı aylık takipte sol ventrikül kitle indeksinde anlamlı azalma görülmesi cesaret verici olmakla birlikte uzun dönem takip gerektirmektedir.

Anahtar Kelimeler: Aort kapak stenozu; sol ventrikül hipertrofisi; kalp kapak protezleri

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INTRODUCTION

Progression of aortic valve stenosis and clinical findings varies between patients, but left ventricular hypertrophy (LVH) is the main pathophysiological adaptive mechanism in all cases. LVH is an important risk factor for long-term morbidity and mortality⁽¹⁾. Aortic valve replacement (AVR) is the gold standard treatment for aortic stenosis (AS). After AVR, LVH regresses with postoperative remodeling. Regression of LVH is one of the most important parameters that have an impact on long-term survival and incidence of adverse events⁽²⁾. Postoperative regression of LVH after replacement of the stenotic valve depends on various factors including preoperative demographics, hemodynamic parameters, and the type of the prosthesis⁽³⁾. Sutureless aortic bioprosthesis has been used with promising clinical and hemodynamic performance with relatively shorter surgical times in high-risk patients than conventional valves^(4,5). Although the decrease of echocardiographic parameters related with left ventricular (LV) mass has been reported before, there is not enough study in the literature related with the effect of sutureless prosthesis on LV mass regression⁽⁶⁾.

The objective of the present study was to document the degree of regression of LVH and to clarify whether high left ventricular mass index (LVMI) had an impact on early mortality, morbidity, and regression of LV mass after surgery with sutureless aortic bioprosthesis for isolated aortic valve stenosis.

A similar pioneering investigation from the same clinic, with absolutely the same inclusion criteria but using mechanical prosthetic valves for replacement, had been completed before and demonstrated that higher LVMI was independently related with increased early morbidity, prolonged hospitalization, and increased in-hospital mortality. Therefore, the secondary aim of the present study was to inquire, compare, and discuss our previous outcomes with this new series.

PATIENTS and METHODS

This was a retrospective observational cohort study. Patients undergoing sutureless aortic bioprosthesis implantation in our hospital between May 2012-September 2017 were included in the study. The study protocol was approved by the Maltepe University review board (2016/900/10). The hospital registry database was searched for patients undergoing heart valve replacement surgery. Inclusion criteria were sutureless-biological aortic valve implantation due to isolated aortic valve stenosis. Exclusion criteria were emergent surgical interventions, surgeries for combined AS and insufficiency or infective endocarditis, concomitant procedures, additional valve replacement, or coronary artery interventions.

Patient Population

A total of 60 high-risk patients were included in the study. The mean age of the patients was 74.2 ± 5.6 years. Male patients comprised 38.3% (n= 23) of the whole patient group. All patients had two or more co-morbidities. Echocardiograms were performed before surgery, intraoperatively, after surgery before discharge, and at every 6 months during follow-up. LVMI was calculated using the Devereux formula and was indexed to the body surface area⁽⁷⁾.

$$LVMI (g/m^2) = 1.04 \times [(LVIDD + PWTD + IVSTD)^3 - (LVIDD)^3] - 13.6 / BSA$$
, where LVIDD is the LV internal diameter in diastole, PWTD is the posterior wall thickness in diastole, IVSTD is the interventricular septum thickness in diastole, and BSA is the body surface area. LVMI > 131 g/m² for males and LVMI > 100 g/m² for females were defined as high depending on the criteria used in clinical practice^(8,9). Sixty patients who were included in the study were divided into two groups depending on LVMI value: 23 patients with LVMI < 131 g/m² for male and < 100 g/m² for female patients were defined as group I and 37 patients with LVMI > 131 g/m² for male and > 100 g/m² for female patients were defined as group II. Patient demographics and echocardiographic findings are recorded to reveal baseline characteristics and shown in Table 1.

Surgical Procedure

Median sternotomy was performed in 40 (66.7%) patients, mini J sternotomy was performed in 13 (21.7%) patients, and right thoracotomy was performed in 7 (11.7%) patients depending on the surgeon's preference and patient's anatomy. Cardiopulmonary bypass (CPB) was instituted, and antegrade blood cardioplegia was used for myocardial protection. Transverse aortotomy was performed 2 cm above the sinotubular junction to provide a place for the frame of the sutureless bioprosthesis. Native valve was excised, and bioprosthesis was implanted with its special delivery system after sizing the annulus. The balloon was inserted into the valve and expanded with 4 atm pressure for 30s, followed by 37°C flush of sterile saline for fixing the stent to the aortic wall. Aortotomy was closed, and after weaning from CPB, bioprosthesis was evaluated by transesophageal echocardiography for position, function, gradients, and paravalvular leakage. Operative data are shown in Table 2.

Follow-up

Clinical and echocardiographic data were collected from hospital control visits at 6 months postoperatively. The National Death Notification System was used to confirm patients who died during follow-up.

Table 1. Patient baseline clinical and echocardiographic data

Variables	Group I (n= 23)	Group II (n= 37)	All patients (n= 60)	p
Age (day), mean ± SD	72.6 ± 4.4	75.2 ± 6.1	74.2 ± 5.6	0.08
Male gender, n (%)	11 (47.8)	12 (32.4)	23 (38.3)	0.23
BMI (kg/m ²)	29.3 ± 3.5	28.5 ± 4.9	28.8 ± 4.4	0.49
BSA (m ²), mean ± SD	1.8 ± 0.2	1.8 ± 0.1	1.8 ± 0.1	0.1
Hypertension, n (%)	13 (56.5)	19 (51.4)	32 (53.3)	0.70
Diabetes, n (%)	10 (43.5)	8 (21.6)	18 (30.0)	0.07
CAD, n (%)	3 (13.0)	9 (24.3)	12 (20.0)	0.29
PAD, n (%)	4 (17.4)	10 (27.0)	14 (23.3)	0.39
COPD, n (%)	8 (34.8)	10 (27.0)	18 (30.0)	0.52
CVD, n (%)	3 (13.0)	5 (13.5)	8 (13.3)	0.96
Preoperative PM, n (%)	0	1 (2.7)	1 (1.7)	0.62
Echocardiographic findings				
EF (%), mean ± SD	54.8 ± 12.5	59.9 ± 9.2	57.9 ± 10.8	0.13
LAD (cm), mean ± SD	4.1 ± 0.7	4.2 ± 0.6	4.1 ± 0.6	0.78
LVIDS (cm), mean ± SD	3.1 ± 0.9	3.1 ± 0.6	3.1 ± 0.7	0.37
LVIDD (cm), mean ± SD	4.3 ± 0.3	4.7 ± 0.4	4.6 ± 0.4	< 0.001*
PWTD (cm), mean ± SD	1.2 ± 0.1	1.4 ± 0.2	1.3 ± 0.2	< 0.001*
IVSTD (cm), mean ± SD	1.2 ± 0.2	1.5 ± 0.2	1.4 ± 0.2	< 0.001*
Aortic peak PG (mmHg)	64.8 ± 19.1	81.3 ± 21.2	75.0 ± 21.8	0.003*
Aortic mean PG (mmHg)	40.5 ± 12.2	49.5 ± 12.9	46.1 ± 13.3	0.009*
LVMI (g/m ²), mean ± SD	114.7 ± 13.7	192.5 ± 31.9	162.7 ± 46.3	< 0.001*

*Statistically significant parameter.

BMI: Body mass index, BSA: Body surface area, CAD: Coronary artery disease, PAD: Peripheral arterial disease, COPD: Chronic obstructive pulmonary disease, CVD: Cerebrovascular disease, PM: Pacemaker, EF: Ejection fraction, LAD: Left atrial diameter, LVIDS: Left ventricular internal diameter in systole, LVIDD: Left ventricular internal diameter in diastole, PWTD: Posterior wall thickness in diastole, IVSTD: Interventricular septum thickness in diastole, PG: Pressure gradient, LVMI: Left ventricular mass index, SD: Standard deviation.

Statistical Analysis

Statistical analysis was performed using the SPSS (Statistical Package for the Social Sciences) software (SPSS Inc., Chicago, IL, USA). Continuous data with normal distribution were expressed as mean and standard deviation, whereas continuous data with non-normal distribution were expressed as median and range. Categorical variables were presented as frequency and percentage.

Independent sample t-test was used for normally distributed variables, whereas Mann-Whitney U test was used for non-normally distributed variables. Pearson chi-square test was used for categorical variables between the two groups. In the dependent group comparison, paired sample t-test was used for normally distributed variables, and Wilcoxon test was used for non-normally distributed variables. Univariate and multivariate

logistic regression analyses were performed to determine the predictive factors for adverse outcomes. A p value < 0.05 was defined as statistically significant.

RESULTS

There was no statistically significant difference between the groups according to preoperative demographic findings except for the parameters of LVIDD, IVSTD, PWTD, LVMI, aortic valvular peak, and mean gradients. These parameters were significantly higher in group II (p < 0.001 for each variable). Patients in the group with high LVMI were slightly older. There was no significant difference between the groups with respect to operative data including the aortic cross-clamp (ACC) and total perfusion times.

Early (≤ 30 days) all-cause mortality was 8.3% with five patients. Four patients died due to multiorgan failure follow-

Table 2. Operative data

Variables	Group I (n= 23)	Group II (n= 37)	All patients (n= 60)	P
Surgical approach, n (%)				0.30
Median sternotomy	18 (78.3)	22 (59.5)	40 (66.7)	
Mini J sternotomy	3 (13.0)	10 (27.0)	13 (21.7)	
Right thoracotomy	2 (8.7)	5 (13.5)	7 (11.7)	
Valve size, n (%)				0.64
Small	9 (39.1)	15 (40.5)	24 (40.0)	
Medium	5 (21.7)	12 (32.4)	17 (28.3)	
Large	6 (26.1)	8 (21.6)	14 (23.3)	
X-large	3 (13.0)	2 (5.4)	5 (8.3)	
CPB time (min), mean ± SD	91.0 ± 48.3	81.2 ± 29.0	85.0 ± 37.5	0.99
ACC time (min), mean ± SD	56.9 ± 37.4	47.3 ± 21.7	51.0 ± 28.8	0.48

CPB: Cardiopulmonary bypass, ACC: Aortic cross-clamp, SD: Standard deviation.

Table 3. Early postoperative events

Variables	Group I (n= 23)	Group II (n= 37)	All patients (n= 60)	p
Left ventricle failure, n (%)	2 (8.7)	6 (16.2)	8 (13.3)	0.41
IABP, n (%)	1 (4.3)	1 (2.7)	2 (3.3)	0.62
ECMO, n (%)	1 (4.3)	0	1 (1.7)	0.38
Paravalvular leak, n (%)	1 (4.3)	0	1 (1.7)	0.38
Reoperation for bleeding, n (%)	2 (8.7)	1 (2.7)	3 (5.0)	0.08
Atrial fibrillation, n (%)	5 (21.7)	9 (24.3)	14 (23.3)	0.82
Conduction abnormalities, n (%)	0	5 (13.5)	5 (8.3)	0.07
CVE, n (%)	3 (13.0)	2 (5.4)	5 (8.3)	0.30
Infection, n (%)	4 (17.4)	4 (10.8)	8 (13.3)	0.47
Acute renal failure, n (%)	2 (8.7)	2 (5.4)	4 (6.7)	0.62
Early mortality, n (%)	2 (8.7)	3 (8.1)	5 (8.3)	0.64
LOS of ICU (day), median (ranges)	3 (1-57)	2 (1-11)	2 (1-57)	0.10
LOS of hospital (day), mean ± SD	17.5 ± 14.2	12.6 ± 8.7	14.5 ± 11.3	0.16

IABP: Intra-aortic balloon pump, ECMO: Extracorporeal membrane oxygenator, CVE: Cerebrovascular event, ICU: Intensive care unit, LOS: Length of stay, SD: Standard deviation.

ing low cardiac out-put syndrome, and one patient died due to respiratory failure and infection followed by sepsis. One patient from each group required intra-aortic balloon pump support due to postoperative LV failure, and the same patient from group I further required extracorporeal membrane oxygenator support after surgery. Rhythm disturbance in the form of atrial fibrillation developed in nine patients in group II and five patients in group I, and normal sinus rhythm was sustained with medical treatment in all patients. Five patients in group II had temporary

conduction abnormalities, but none of them required permanent pacemaker. Two patients from each group required short-term dialysis for acute renal failure. There was only one patient in group I with mild paravalvular leakage at follow-up. There was no statistically significant difference between the groups with respect to postoperative early complication rates. Similarly, the length of stay in the intensive care unit and total hospitalization times were not different between the groups. Postoperative outcomes regarding to groups are shown in Table 3.

Table 4. Preoperative and postoperative echocardiographic data

Variables	Group I			Group II		
	Preoperative TTE	Preoperative TTE	p	Preoperative TTE	Preoperative TTE	p
EF (%), mean ± SD	54.8 ± 12.5	54.8 ± 9.8	0.87	59.9 ± 9.2	57.2 ± 8.9	0.07
LVIDS (cm), mean ± SD	3.1 ± 0.9	3.1 ± 1.0	0.94	3.1 ± 0.6	2.9 ± 0.5	0.001*
LVIDD (cm), mean ± SD	4.3 ± 0.3	4.7 ± 0.7	0.01*	4.7 ± 0.4	4.6 ± 0.4	0.15
LAD (cm), mean ± SD	4.1 ± 0.7	3.9 ± 0.5	0.02*	4.2 ± 0.6	3.9 ± 0.5	0.001*
PWTD (cm), mean ± SD	1.2 ± 0.1	1.2 ± 0.1	0.75	1.4 ± 0.2	1.2 ± 0.2	<0.001*
IVSTD (cm), mean ± SD	1.2 ± 0.2	1.2 ± 0.1	0.81	1.5 ± 0.2	1.2 ± 0.2	<0.001*
LVMI (g/m ²), mean ± SD	114.7 ± 13.7	109.7 ± 32.2	0.60	192.5 ± 31.9	117.9 ± 25.2	<0.001*
Aortic peak PG (mmHg)	64.8 ± 19.1	24.1 ± 8.6	<0.001*	81.3 ± 21.2	23.1 ± 8.5	<0.001*
Aortic mean PG (mmHg)	40.5 ± 12.2	13.0 ± 4.5	<0.001*	49.5 ± 12.9	12.1 ± 5.5	<0.001*

* Statistically significant parameter.

EF: Ejection fraction, LAD: Left atrial diameter, LVIDS: Left ventricular internal diameter in systole, LVIDD: Left ventricular internal diameter in diastole, PWTD: Posterior wall thickness in diastole, IVSTD: Interventricular septum thickness in diastole, LVMI: Left ventricular mass index, PG: Pressure gradient, SD: Standard deviation, TTE: Transthoracic echocardiography.

Postoperative echocardiography was performed at discharge and at 6 months of follow-up. Transaortic gradients decreased significantly in both groups as expected. LVMI decreased from 114.7 ± 13.7 g/m² at baseline to 109 ± 32.2 g/m² at follow-up in group I ($p=0.60$) and from 192.5 ± 31.9 g/m² to 117.9 ± 25.2 g/m² in group II ($p<0.001$). This decrease was statistically important in group II but not in group I. LV internal diameter, interventricular septum thickness, and LV posterior thickness decreased significantly after surgery in group II but not in group I. Improvement in these echocardiographic parameters was accompanied with clinical improvement of patients with New York Heart Association class I or II symptoms in both groups. Changes in echocardiographic variables are shown in Table 4.

Univariable and multivariable Cox regression analyses were used to examine the relationship between LVMI and postoperative adverse events and mortality. Multivariate analysis for early mortality demonstrated only advanced age to be an independent risk factor.

DISCUSSION

Increased LVMI was found to be independently associated with increased cardiovascular morbidity and mortality in patients with non-severe AS followed up during the progress of their disease⁽¹⁰⁾. Concentric LVH with especially increased posterior wall thickness was demonstrated to be associated with increased postoperative mortality after AVR for AS^(11,12). In contrast, a recent study by Minamino-Muta documented that high LVMI did not have any adverse impact on morbidity and mortality in surgically treated patients with AS⁽¹³⁾. There

are several studies in the literature showing the association of high LVMI with increased morbidity, such as congestive heart failure, low cardiac output syndrome, arrhythmias, and mortality after surgery^(11,14,15). Sutureless aortic bioprosthetic valves have been shown to be safe and effective after they had been introduced into clinical practice⁽¹⁶⁻¹⁹⁾. These valves were accepted as feasible alternatives to conventional surgery for high-risk patients with AS. Therefore, we aimed to analyze high LVMI effect on early morbidity and mortality after implantation of sutureless valves. Early mortality was slightly higher in the present study than in several previous studies^(6,19). Patients in the present study were the first group of patients who had sutureless valve implantation in our hospital, and all patients had high-risk profile with prolonged exposure to AS for years and the outcomes probably reflected the learning curve of the new technology in addition to the inherent risks of the patients. Groups were comparable with regard to preoperative and intraoperative data except for excessive LVH in group II. Postoperative LV failure and arrhythmias in group II were almost twice those of group I, but probably since the sample size was small, it did not achieve statistical significance. There were no significant between-group differences in the incidence of adverse events and deaths. Age, chronic obstructive lung disease, and preoperative mean aortic gradient were found as independent risk factors for cardiac adverse events, and age was the only factor affecting mortality. Preoperative high LVMI was not found to be an independent risk factor for early morbidity and mortality. These outcomes were not consistent with our initial analysis of AVR with mechanical valves, in which preoperative high LVMI was an independent risk factor

Table 5. Univariate and multivariate analyses for early mortality

Variables	Univariate			Multivariate		
	OR	95% CI lower-upper	p	OR	95% CI lower-upper	p
Gender	0.93	0.14-6.01	0.94			
Age (day)	1.23	1.02-1.48	0.03	1.29	1.04-1.58	0.02
BMI	1.0	0.81-1.23	0.99			
BSA	0.34	0-295.0	0.75			
Hypertension	0.56	0.09-3.59	0.54			
Diabetes	1.63	0.25-10.67	0.61			
CAD	3.0	0.44-20.38	0.26			
COPD	4.0	0.61-26.35	0.15			
CVD	0	0	0.99			
Preoperative PM	0	0	1.0			
EF	0.98	0.91-1.05	0.53			
LVMI	1.0	0.98-1.02	0.99			
Aortic peak PG	0.99	0.95-1.03	0.62			
Aortic mean PG	0.98	0.91-1.06	0.61			
Cross-clamp time	1.0	0.97-1.03	0.91			
CPB time	1.0	0.99-1.03	0.27			

BMI: Body mass index, BSA: Basal surface area, CAD: Coronary artery disease, CI: Confidence interval, COPD: Chronic obstructive pulmonary disease, CPB: Cardiopulmonary bypass, CVD: Cerebrovascular disease, EF: Ejection fraction, LVMI: Left ventricular mass index, OR: Odds ratio, PG: Pressure gradient, PM: Pacemaker.

Table 6. Univariate and multivariate analyses for cardiac adverse event

Variables	Univariate			Multivariate		
	OR	95% CI lower-upper	p	OR	95% CI lower-upper	p
Gender	1.43	0.49-4.19	0.52			
Age	1.44	1.02-1.28	0.02	1.17	1.03-1.32	0.01
BMI	0.95	0.84-1.07	0.36			
BSA	0.44	0.01-19.59	0.67			
Hypertension	1.4	0.49-3.97	0.53			
Diabetes	0.67	0.21-2.12	0.49			
COPD	2.5	0.81-7.74	0.11	4.13	1.10-15.49	0.04
CVD	1.6	0.36-7.13	0.54			
Preoperative PM	0	0	1.0			
EF	1.01	0.06-1.06	0.81			
LVMI	1.01	0.99-1.02	0.06			
Aortic peak PG	1.03	0.99-1.05	0.06			
Aortic mean PG	1.04	0.99-1.08	0.08	1.05	1.0-1.11	0.05
Cross-clamp time	1.0	0.99-1.02	0.75			
CPB time	1.0	0.99-1.02	0.61			

BMI: Body mass index, BSA: Basal surface area, CI: Confidence interval, COPD: Chronic obstructive pulmonary disease, CPB: Cardiopulmonary bypass, CVD: Cerebrovascular disease, EF: Ejection fraction, LVMI: Left ventricular mass index, OR: Odds ratio, PG: Pressure gradient, PM: Pacemaker.
Cardiac adverse event: The presence of new onset atrial fibrillation, malignant arrhythmia, left ventricle failure, and early mortality.

of early mortality in addition to age, ACC time, and CPB time⁽²⁰⁾. Probably the reason of this difference was the shorter ACC and CPB times, which was an obvious advantage of the sutureless valves in these hypertrophic ventricles.

One of the therapeutic aims of valve replacement is to provide regression of LV mass since it is closely associated with long-term outcomes and survival. Regression of LVH after surgery is a long process affected by many factors⁽²¹⁻²⁴⁾. Lund, et al. reported the preoperative risk profile of patients to be related with regression of LVH after 10 years of follow-up of AVR for AS⁽²²⁾. Baseline LVMI was shown to be an independent predictor of LV mass regression after replacement with stentless valves⁽²⁵⁾. Lim, et al. reported that baseline LVMI is found to be the single variable that influenced LV mass regression after AVR with stentless valves or homografts⁽²⁶⁾. Satisfactory LV mass regression has been documented in our previous series of our patients with AS after conventional AVR with mechanical valves⁽²⁰⁾. A recent meta-analysis comparing mechanical and tissue valves stated higher patient-prosthesis mismatch and less reduction in ventricular mass with tissue valves in patients with small aortic roots⁽²⁷⁾. Since the current guidelines do not recommend mechanical valves in elderly patients, sutureless valves may be an alternative in this elderly group of patients, with their satisfactory hemodynamic results, adequate effective orifice area, and ventricular mass reduction^(28,29). Concistre, et al. reported a significant regression of LV mass even in high-risk patients with 3f Enable[®] valves (sutureless aortic bioprosthesis, Medtronic; ATS Medical, Minneapolis, MN, USA)⁽²¹⁾. Santarpino, et al. reported a significant mass regression with Perceval S[®] (sutureless aortic bioprosthesis; Sorin Group, Saluggia, Italy) at 1 year of follow-up⁽⁶⁾. This study also supports that replacement of the stenotic valve with sutureless aortic bioprosthesis provides satisfactory regression of LV mass in patients with high preoperative LVMI at 6 months of follow-up. This was an encouraging result but needs to be followed up since regression is an ongoing process. Long-term results of our patients are planned to be followed up and documented for the course of regression of LVH and its effect on long-term survival.

Although high LVMI was not found to be an independent factor affecting postoperative morbidity and mortality, baseline LVMI has been shown to have an effect on regression of LV mass after surgery; therefore, we can propose that early intervention may provide better reverse remodeling of the left ventricle and has a positive prognostic effect. In conclusion, sutureless bioprosthesis can be a safe alternative approach for valve replacement in elderly high-risk patients with severe LVH, but long-term follow-up is necessary.

CONFLICT of INTEREST

The authors declare that there is no conflict of interest regarding the publication of this article.

AUTHORSHIP CONTRIBUTIONS

Concept/Design: AA, BÇ

Analysis/Interpretation: MD, EÇ

Data Acquisition: BÇ, EÇ

Writing: AA, BÇ

Critical Revision: AA, AT

Final Approval: All of authors.

REFERENCES

1. Kupari M, Turto H, Lommi J. Left ventricular hypertrophy in aortic valve stenosis: preventive or promotive of systolic dysfunction and heart failure? *Eur Hear J* 2005;26:1790-6.
2. Thomson HL, O'Brien MF, Almeida AA, Tesar PJ, Davison MB, Bursleftov DJ. Hemodynamics and left ventricular mass regression: a comparison of the stentless, stented and mechanical aortic valve replacement. *Eur J Cardiothorac Surg* 1998;13:572-5.
3. Villa E, Troise G, Cirillo M, Brunelli F, Tomba MD, Mhagna Z, et al. Factors affecting left ventricular remodeling after valve replacement for aortic stenosis. An overview. *Cardiovasc Ultrasound* 2006;4:25.
4. Phan K, Tsai YC, Niranjana N, Bouchard D, Carrel TP, Dapunt OE, et al. Sutureless aortic valve replacement: a systematic review and meta-analysis. *Ann Cardiothorac Surg* 2015;4:100-11.
5. Shrestha M, Fischlein T, Meurise B, Flameng W, Carrel T, Madonna F, et al. European multicentre experience with the sutureless Perceval valve: clinical and haemodynamic outcomes upto 5 years in over 700 patient. *Eur J Cardiothorac Surg* 2016;49:234-41.
6. Santarpino G, Pfeiffer S, Pollari F, Concistrè G, Vogt F, Fischlein T. Left ventricular mass regression after sutureless implantation of the Perceval S aortic valve bioprosthesis: preliminary results. *Interactive Cardio Vasc and Thorac Surg* 2014;18:38-42.
7. Devereux RB, Reichek N. Echocardiographic determination of left ventricular mass in man: anatomic validation of the method. *Circulation* 1977;55:613-8.
8. Levy D, Savage DD, Garrison RJ, Anderson KM, Kannel WB, Castelli WP. Echocardiographic criteria for left ventricular hypertrophy: the Framingham Heart Study. *Am J Cardiol* 1987;59:956-60.
9. Foppa M, Duncan BB, Rohde LEP. Echocardiography-based left ventricular mass estimation. How should we define hypertrophy? *Cardiovascular Ultrasound* 2005;3:17.
10. Gerds E, Rossebo AB, Pedersen TR, Cioffi G, Lonnebakk MT, Cramariuc D, et al. Relation of left ventricular mass to prognosis in initially asymptomatic mild to moderate aortic valve stenosis. *Circ Cardiovasc Imaging* 2015;8:e003644.
11. Orsinelli DA, Aurigemma GP, Battista S, Krendel S, Gaasch WH. Left ventricular hypertrophy and mortality after aortic valve replacement for aortic stenosis. *JACC* 1993;22:1679-83.
12. Duncan AI, Lowe BS, Garcia MJ, Xu M, Gillinov AM, Mihaljevic T, et al. Influence of concentric left ventricular remodeling on early mortality after aortic valve replacement. *Ann Thorac Surg* 2008;85:2030-9.
13. Minamino-Muta E, Kato T, Morimoto T, Taniguchi T, Inoko M, Haruna T, et al. Impact of the left ventricular mass index on the outcomes of severe aortic stenosis. *Heart* 2017;103:1992-9.
14. Mehta RH, Bruckman D, Das S, Tsai T, Russman P, Karavşte D, et al. Implications of increased left ventricular mass index on in-hospital outcomes in patients undergoing aortic valve surgery. *J Thorac Cardiovasc Surg* 2001;122:919-28.

15. Fuster RG, Argudo JA, Albarova OG, Sos FH, Lopez SC, Sorli MJ, et al. Left ventricular mass index in aortic valve surgery: a new index for early valve replacement? *Eur Cardiothorac Surg* 2003;23:696-702.
16. Folliguet TA, Laborde F, Zannis K, Ghorayeb G, Haverich A, Shrestha M. Sutureless Perceval aortic valve replacement: results of two European centers. *Ann Thorac Surg* 2012;93:1483-8.
17. Borger MA, Dohmen P, Misfeld M, Mohr FW. Current trends in aortic valve replacement: development of the rapid deployment Edwards Intuity valve system. *Exp Rev Med Devices* 2013;10:461-70.
18. Kocher AA, Laufer G, Haverich A, Shrestka M, Walther T, Misfeld M, et al. One-year outcomes of the Surgical Aortic Valve (TRITON) trial: a prospective multicenter study of rapid-deployment aortic valve replacement with the Edward Intuity Valve System. *J Thorac Cardiovasc Surg* 2013;145:110-5.
19. Concistre G, Chiaramonti F, Santarpino G, Pfeiffer S, Marchi F, Vogt F, et al. Left ventricular mass regression after two alternative sutureless aortic bioprostheses. *Innovations* 2015;10:114-9.
20. Antal Dönmez A. Aort darlığı nedeniyle uygulanan aort kapak replasmanında sol ventrikül kitle indeksinin erken morbidite ve mortalite üzerine etkisi. T.C. Sağlık Bakanlığı Koşuyolu Kalp Eğitim ve Araştırma Hastanesi, İstanbul, Türkiye, 2003.
21. Concistre G, Miceli A, Marchi F, Chiaramonti F, Glauber M, Solinas M. Regression of left ventricular mass after implantation of the sutureless 3f Enable aortic bioprosthesis. *Tex Heart Inst J* 2015;42:117-23.
22. Lund O, Emmertsen K, Dorup I, Jensen FT, Flo C. Regression of left ventricular hypertrophy during 10 years after valve replacement for aortic stenosis is related to the preoperative risk profile. *Eur Heart J* 2003;24:1437-46.
23. Tasca G, Brunelli F, Cirillo M, Tomba MD, Mhagna Z, Troise G, et al. Impact of the improvement of valve area achieved with aortic valve replacement on regression of left ventricular hypertrophy in patients with pure aortic stenosis. *Ann Thorac Surg* 2005;79:1291-6.
24. Rubens FD, Gee YY, Ngu JMC, Chen L, Burwash IG. Effect of pericardial valve choice on outcomes and left ventricular mass regression in patients with left ventricular hypertrophy. *J Thorac Cardiovasc Surg* 2016;152:1291-8.
25. Del Rizzo DF, Abdoh A, Cartier P, Doty D, Westaby S. Factors affecting left ventricular mass regression after aortic valve replacement with stentless valves. *Semin Thorac Cardiovasc Surg* 1999;11(4 Suppl 1):114-20.
26. Lim E, Ali A, Theodorou P, Sousa I, Ashrafian H, Chamageorgakis T, et al. Longitudinal study of the profile and predictors of left ventricular mass regression after stentless aortic valve replacement. *Ann Thorac Surg* 2008;85:2026-9.
27. Moscarelli M, Fattouch K, Speziale G, Nasso G, Santarpino G, Gaudino M, et al. A meta-analysis of the performance of small tissue versus mechanical aortic valve prostheses. *Eur J Cardiothorac Surg* 2019.
28. Belluschi I, Moriggia S, Giacomini A, Del Forno B, Di Sanzo S, Blasio A, et al. Can Perseval sutureless valve reduce rate of patient prosthesis mismatch? *Eur J Cardiothorac Surg* 2017;51:1093-9.
29. Haverich A, Wahlers TC, Borger MA, Shrestha M, Kocher AA, Walther T, et al. Three-year hemodynamic performance, left ventricular mass regression, and prosthetic-patient mismatch after rapid deployment aortic valve replacement in 287 patients. *J thorac Cardiovasc Surg* 2014;148:2854-61.