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ORIGINAL RESEARCH

Outcomes of Worsened Left Ventricular Ejection Fraction After Transcatheter Aortic Valve Replacement

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Background: Left ventricular ejection fraction (LVEF) worsening after transcatheter aortic valve replacement (TAVR) was common in clinical practice. However, the effect of acute worsening LVEF is unclear.

Methods: All consecutive patients who underwent TAVR between January 2016 and May 2022 were analyzed. Patients were divided into worsened LVEF and non-worsened LVEF according to whether or not they had an LVEF decline of ≥5% at discharge. Survival at follow-up was compared between two groups. Logistic regression analysis was used to determine independent predictors of worsening LVEF.

Results: A total of 439 patients were included in the analysis, and 112 (25.5%) patients had worsened LVEF. Worsened LVEF was more common in patients with LVEF ≥50%. After multivariable logistic analysis, only baseline LVEF was associated with worsening LVEF [OR=1.06 (95% CI: 1.04–1.08), P < 0.001]. The decline in LVEF recovered to the baseline after one month. There were no significant differences in survival between patients with and without worsened LVEF (Log rank P = 0.48).

Conclusion: Acute worsening of LVEF after TAVR was not uncommon but did not affect survival. It could recover to baseline levels after one month. Routine post-TAVR echocardiography should focus on other metrics rather than acute LVEF changes.

Keywords: aortic stenosis, transcatheter aortic valve replacement, left ventricular ejection fraction

Introduction

Left ventricular dysfunction was common in aortic stenosis (AS) due to increased pressure load and left ventricular remodeling. Reduced left ventricular ejection fraction (LVEF) was associated with poor prognosis in patients with severe AS who were treated conservatively or surgically.[1] In current guidelines, patients with severe AS and LVEF less than 50% was defined as Class I indication for aortic valve replacement, and transcatheter aortic valve replacement (TAVR) has been established as a procedure for patients with severe stenosis in high surgical risk.[2] Previous studies indicated that left ventricular systolic function improved after TAVR in patients with reduced LVEF.[3,4] A decline of LVEF after aortic valve replacement was also seen in clinical practice.[5] The role of LVEF in patients with AS treated by TAVR was conflicting.[6–10] There was limited data on the change of LVEF after TAVR in varying LVEF at baseline. The impact of LVEF reduction after TAVR on the outcome is not clear. Accordingly, the present study focused on acute LVEF deterioration after TAVR and its predictors and examined the impact of acute LVEF deterioration on survival.

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Materials and Methods

We analyzed data of all patients who underwent TAVR at our institution from January 2016 through May 2022. Patients were excluded from the study if data were missing on baseline or discharge LVEF. We retrospectively analyzed data that were prospectively collected in our institutional registry database. All patients were present with severe symptomatic aortic valve stenosis, and they were considered at high surgical risk by the heart team, which consisted of a cardiac surgeon, an interventional cardiologist, and an anesthesiologist. Patients gave their informed consent for the procedure and for data collection. This study was approved by the Research Ethics Committee of Guangdong Provincial People's Hospital (No. GDREC2019384H). Written informed consent was obtained from all participants for this registry. Our study complied with the Declaration of Helsinki.

All patients underwent comprehensive 2-dimensional and Doppler echocardiographic evaluation according to the guideline pre-operation and before discharge.[11] Mean aortic pressure gradients were determined using the simplified Bernoulli equation. LVEF was calculated using Simpson's biplane method. We assessed the difference between post-operative (discharge) LVEF and preoperative LVEF ($\Delta EF = LVEF_{pre\text{-}procedure} - LVEF_{post\text{-}procedure}$). As previous study, we used a 5% LVEF variation as the cutoff for study group creation.[5] A 5% cutoff was chosen as it reflects meaningful short-term variations while minimizing noise. Patients were divided into two groups according to the LVEF variation: worsened LVEF ($\Delta EF \geq 5\%$) and no worsened LVEF ($\Delta EF < 5\%$).

The details of TAVR procedures in our institution have been previously described.[12] The heart team had a consensus on the vascular access and valve size selection. Self-expanding valve such as VenusA, VenusA-Pro, VenusA-Plus valve (Venus Medtech), Taurusone valve (Peijia Medical), ScienCrown valve (Lepu medical) and Vitaflow (Microport); balloon-expanding valve such as Edwards Sapien (Edwards Lifesciences) were implanted.

The primary outcome measure in the present analysis was 30-day mortality after TAVR. Other outcome measures included cardiovascular death, major vascular complications, pacemaker implantation, new-onset atrial fibrillation and major bleeding at 30-day after TAVR and mortality, stroke, permanent pacemaker and rehospitalization for heart failure at follow-up. All the events were defined according to the Valve Academic Research Consortium definitions 3.

Statistical Analysis

Categorical variables are presented as numbers and percentages and were compared using the chi-square test or the Fisher exact test. Continuous variables are expressed as mean ± SD or median (interquartile range). Continuous variables were compared using one-way analysis of variance or Kruskal–Wallis tests based on their distributions. Univariable and multivariable logistic regression models were used to evaluate the factors that were associated with worsened LVEF. Odds ratios (ORs) and 95% confidence intervals (CIs) were reported. Cumulative incidence was estimated by the Kaplan–Meier method, and differences were assessed using the Log rank test. Multivariable Cox proportional regression analysis was used to evaluate the association of worsened LVEF with all-cause mortality. Hazard ratios (HRs) and 95% CI were reported. All tests were two-tailed. A value of p less than 0.05 was considered significant.

Results

A total of 439 patients with severe AS treated by TAVR were included in the final analysis. There were 112 patients (25.5%) with worsened LVEF and 381 patients (74.5%) without worsened LVEF. Baseline demographic and clinical characteristics of the overall population and according to ΔEF were presented in Table 1. Mean age was 72.5 ± 7 years, and 190 patients (43.3%) were female. In the entire cohort, the median Society of Thoracic Surgeon's Predicted Risk of Mortality Score (STS score) was 2.45% (IQR: 1.50–4.39). The baseline patient's characteristics were generally similar between the groups with and without worsened LVEF, except for higher prevalence rate of prior cardiac valve surgery in patients with worsened LVEF. Patients with worsened LVEF seemed to have lower levels of NT-pro BNP than that in patients without worsened LVEF, although it did not reach a statistically significant difference ($P = 0.058$) (Table 1). The characteristics of the procedure were similar in the groups with and without worsened LVEF (Table 1). Before procedure, LVEF was significantly greater [64% (IQR 55.8–70%) vs 59% (IQR 40.1–65%); $P < 0.001$] and the incidence of pulmonary hypertension were significantly lower (11.6 vs 23.9%, $P = 0.006$) in the group with worsened LVEF than in

Table 1 Baseline Demographical and Clinical Characteristics of the Overall Population and According to Change of Left Ventricular Ejection Fraction

Variables	Total (n=439)	Worsened LVEF (n=112)	No-Worsened LVEF (n=327)	P value
Age, y	72.5±7	73.5±6.4	72.2±7.1	0.089
Female	190(43.3)	48(42.9)	142(43.4)	0.917
Body mass index, kg/m2	22.7(20.3,25.4)	22.3(20.1,25.0)	22.8(20.4,25.4))	0.519
Hypertension	224(51)	62(55.4)	162(49.5)	0.288
Diabetes mellitus	101(23)	22(19.6)	79(24.2)	0.327
Peripheral artery disease	64(14.6)	12(10.7)	52(15.9)	0.179
Chronic obstructive pulmonary disease	22(5)	7(6.3)	15(4.6)	0.486
Prior coronary artery disease	156(35.5)	41(36.6)	115(35.2)	0.784
Prior MI	33(7.5)	7(6.3)	26(8)	0.556
Prior percutaneous coronary intervention	76(17.3)	19(17)	57(17.4)	0.91
Prior stroke	30(6.8)	9(8)	21(6.4)	0.559
Prior pacemaker implantation	4(0.9)	1(0.9)	3(0.9)	1
Atrial fibrillation	71(16.2)	21(18.8)	50(15.3)	0.391
Prior cardiac valve surgery	16(3.6)	9(8)	7(2.1)	0.008
Anemia	148(33.7)	39(34.8)	109(33.3)	0.774
STS score, %	2.45(1.50,4.39)	2.55(1.53,4.2)	2.39(1.47,4.44)	0.892
Bicuspid aortic valve	251(57.2)	70(62.5)	181(55.4)	0.187
NT-proBNP	2027.5(629.6,6861.5)	1503.5(596.8,5437)	2077(646.2,7387.5)	0.058
TnT, pg/mL	26.1(16,64)	26.9(14.9,64.8)	25.9(16.1,63.4)	0.713
Creatinine, mg/L	1.0(0.79,1.27)	1.0(0.8,1.35)	1.0(0.79,1.25)	0.9
Procedure detail				
Transfemoral access	418(95.7)	104(92.9)	314(96.6)	0.096
TAV in TAV	44(10)	11(9.8)	33(10.1)	0.934
Concomitant PCI	42(9.8)	7(6.3)	36(11)	0.144
Valve type				0.158
Self-expandable	428(97.5)	107(95.5)	321(98.2)	
Balloon-expandable	11(2.5)	5(4.5)	6(1.8)	
Device brand				0.162
VenusA/VenusA-Pro/VenusA-Plus	402(91.6)	99(88.4)	303(92.7)	
Taurusone	3(0.7)	1(0.9)	2(0.6)	
ScienCrown	6(1.4)	0	6(1.8)	
Vitaflow	17(3.9)	7(6.3)	10(3.1)	
Sapien	11(2.5)	5(4.5)	6(1.8)	

Notes: Data are presented as mean±SD, median [25–75% interquartile range] and n (%).
Abbreviations: MI, myocardial infarction; STS score, Society of Thoracic Surgeons' score; TnT, troponin T; TAV in TAV, more than one valve prosthesis was implant at same procedure; PCI, percutaneous coronary intervention.

the group without worsened LVEF, while moderate or severe aortic regurgitation and mitral regurgitation, mean gradient and peak velocity were not significantly different between the two groups (Table 2).

Before discharge, the median LVEF was significantly lower in the group with worsened LVEF than in the group without worsened LVEF (53 vs 61%, $P<0.001$). LVEF was significantly lower before discharge compared to LVEF at baseline in the group with worsened LVEF (53 vs 64%, $P<0.001$), while LVEF was significantly greater before discharge than that at baseline in the group without worsened LVEF (61 vs 57%, $P<0.001$). The prevalence rates of moderate or severe aortic regurgitation, mitral regurgitation, tricuspid regurgitation, and pulmonary hypertension were not significantly difference between the 2 groups (Table 2). Univariate regression analysis showed that prior cardiac valve surgery and higher baseline LVEF were associated with LVEF worsened. However, after multivariate regression analysis, only higher baseline LVEF was independent predictors for LVEF worsening after TAVR (OR:1.06, 95% CI:1.04–1.08; P<0.001) (Table 3).

Table 2 Echocardiographic Parameters at Baseline, Discharge and 1 month After Procedure

Variables	Pre-TAVR			
	Total (n=439)	Worsened LVEF (n=112)	No-worsened LVEF (n=327)	P value
LVEF, %	59(42,66)	64.5(56.3,70)	57(39,64)	<0.001
Peak velocity, m/s	4.8(4.3,5.3)	4.7(4.2,5.4)	4.9(4.3,5.3)	0.397
Mean gradient, mm Hg	55(43,67)	53(44,65)	56.5(43,69)	0.288
Aortic regurgitation ≥ moderate	205(46.7)	60(53.6)	145(44.3)	0.091
Mitral regurgitation ≥ moderate	169(38.5)	35(31.3)	134(41)	0.068
Tricuspid regurgitation ≥ moderate	104(23.7)	21(18.8)	83(25.4)	0.154
Pulmonary hypertension ≥ moderate	91(20.7)	13(11.6)	78(23.9)	0.006
Before discharge				
LVEF, %	60(45,65)	53(40.3, 61)	61(46,67)	<0.001
Aortic regurgitation ≥ moderate	43(9.8)	10(8.9)	33(10.1)	0.721
Mitral regurgitation ≥ moderate	106(24.1)	32(28.6)	74(22.6)	0.205
Tricuspid regurgitation ≥ moderate	83(18.9)	21(18.8)	62(19)	0.961
Pulmonary hypertension ≥ moderate	23(5.2)	7(6.3)	16(4.9)	0.578
One month after TAVR				
LVEF, %	62(52,68)	61(50,66)	63(52,68)	0.073
Aortic regurgitation ≥ moderate	41(12.1)	10(11.0)	31(12.5)	0.705
Mitral regurgitation ≥ moderate	56(16.6)	16(17.6)	40(16.2)	0.761
Tricuspid regurgitation ≥ moderate	51(15)	19(20.9)	32(12.9)	0.069
Pulmonary hypertension ≥ moderate	18(5.4)	5(5.6)	13(5.3)	1

Notes: Data are presented as mean±SD, median (25–75% interquartile range) and n (%).
Abbreviation: LVEF, left ventricular ejection fraction.

Table 3 Association of Pre-Operation Characteristics with LVEF Declination

	Univariate OR (95% CI)	P value	Multivariate OR (95% CI)	P value
Age	1.03(0.99–1.07)	0.093	–	–
Male	0.97(0.60–1.58)	0.908	–	–
Diabetes mellitus	0.66(0.36–1.20)	0.175	–	–
Prior coronary artery disease	1.29(0.79–2.11)	0.311	–	–
Prior percutaneous coronary intervention	1.33(0.73–2.44)	0.354	–	–
Atrial fibrillation	1.23(0.66–2.31)	0.517	–	–
Prior cardiac valve surgery	4.05(1.25–13.1)	0.02	3.34(0.97–11.57)	0.057
Baseline LVEF	1.06(1.04–1.09)	<0.001	1.06(1.04–1.08)	<0.001

The distribution of different group of LVEF before TAVR is shown in Figure 1. About 81% of patients in group of worsened LVEF have a LVEF≥ 50%, while only 60% patients in group without worsened LVEF. The distribution of LVEF before procedure was significant between two groups ($P < 0.001$). Three hundred and thirty-nine patients (80.4%) completed echocardiographic assessment one month after the procedure. The change of LVEF over time in the whole population and the two groups are shown in Figure 2. LVEF regression in patients with worsened LVEF is still worse than LVEF at baseline. LVEF in patients without worsened LVEF remained stable at one month compared with baseline LVEF. There was no significant difference in LVEF at one month between the group with worsened LVEF and the group without worsened LVEF [61(IQR:51–66) vs 63(IQR:52–68), $P = 0.073$].

Sixteen patients (3.6%) died within 30 days, and the rate of mortality was comparable between the two groups (5.4 vs 3.1%, $P = 0.255$). The 30-day clinical outcomes were generally similar between the groups with and without worsened LVEF, except for higher prevalence rates of new-onset atrial fibrillation in the group with worsened LVEF (16.1 vs 8.9%, $P = 0.033$) (Table 4). Thirty-one (7.5%) patients died for all cause in the whole cohort during a median follow-up of 21

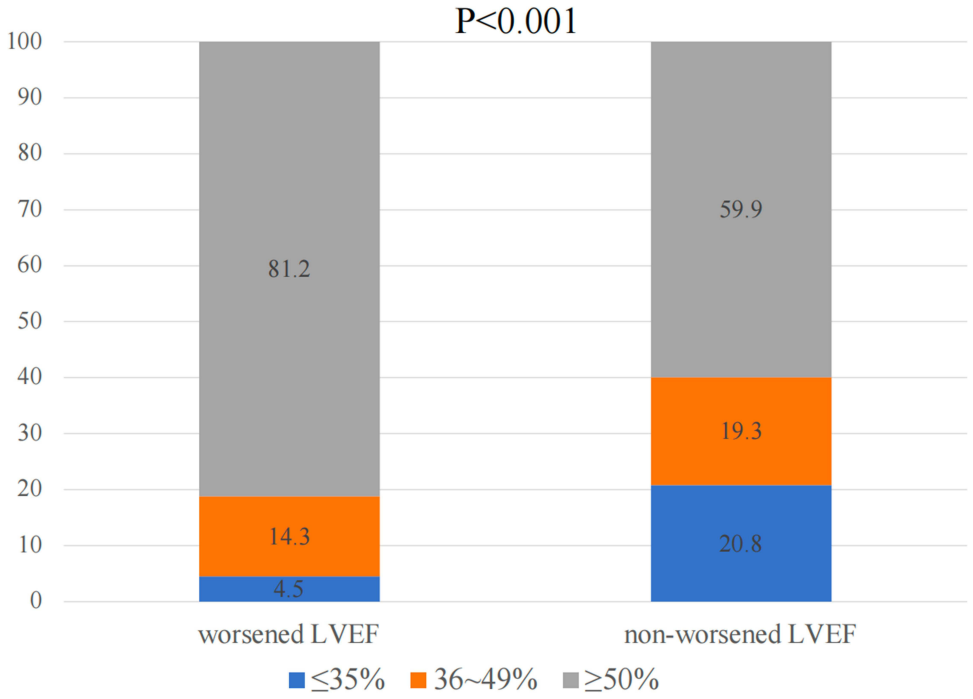

Figure 1 The distribution of different group of LVEF before TAVR.

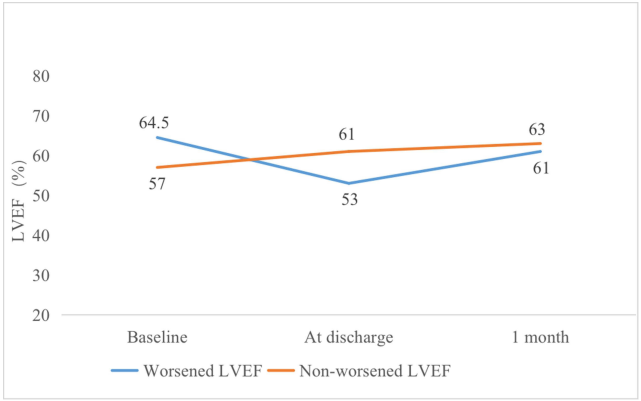

Figure 2 The change of LVEF over time according to LVEF.

months. Figure 3 shows the Kaplan–Meier unadjusted survival curve for patients with or without worsened LVEF. Cumulative mortality rate was comparable between patients with and without worsened LVEF (4.8 vs 8.4%, P = 0.225). The rate of stroke, pacemaker, and hospitalization for heart failure at follow-up was not the difference between the two groups.

Discussion

The main findings of this study are that (1) About a quarter of patients with AS had LVEF worsening after TAVR; (2) patients with better baseline LVEF had higher probability to decline LVEF after TAVR but regression at one month; and (3) LVEF worsening did not affect survival at follow-up.

The impact of LVEF on prognosis after TAVR in patients with AS is controversial.[8–10] To the best of our knowledge, this is the first study with large samples to report the early worsening LVEF after TAVR. In the present study of patients

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Table 4 30-Day and Follow-up Clinical Outcomes

Variable	Total	Worsened LVEF (n=132)	No-worsened LVEF (n=381)	P value
30-day outcome				
All-cause mortality	16(3.6)	6(5.4)	10(3.1)	0.255
Hospitalization for heart failure	9(2.1)	3(3.7)	6(1.8)	0.699
Stroke	9(2.1)	1(0.9)	8(2.4)	0.459
Life-threatening or major bleeding	42(9.6)	9(8)	33(10.1)	0.523
Major vascular complications	24(5.5)	4(3.6)	20(6.1)	0.307
New-onset atrial fibrillation	47(10.7)	18(16.1)	29(8.9)	0.033
Pacemaker implantation	27(6.2)	4(3.6)	23(7)	0.188
MACE	28(6.4)	7(6.3)	21(6.4)	0.949
Follow up outcome				
All-cause mortality	31(7.5)	5(4.8)	26(8.4)	0.225
Stroke	5(1.2)	1(1.0)	4(1.3)	1
Permanent Pacemaker implantation	6(1.4)	0	6(1.9)	0.344
Hospitalization for heart failure	10(2.4)	4(3.8)	6(1.9)	0.28

Abbreviations: MACE, major adverse cardiovascular event, composite of death, stroke and heart failure rehospitalization.

undergoing TAVR, 25.5% developed worsened LVEF defined as an absolute decline in LVEF ≥5% from baseline to discharge. Our results are in agreement with a relatively small retrospective study that included 122 patients with severe AS who underwent transapical TAVR, of which 22% patients had a LVEF reduction of more than 5% before discharge.[5]

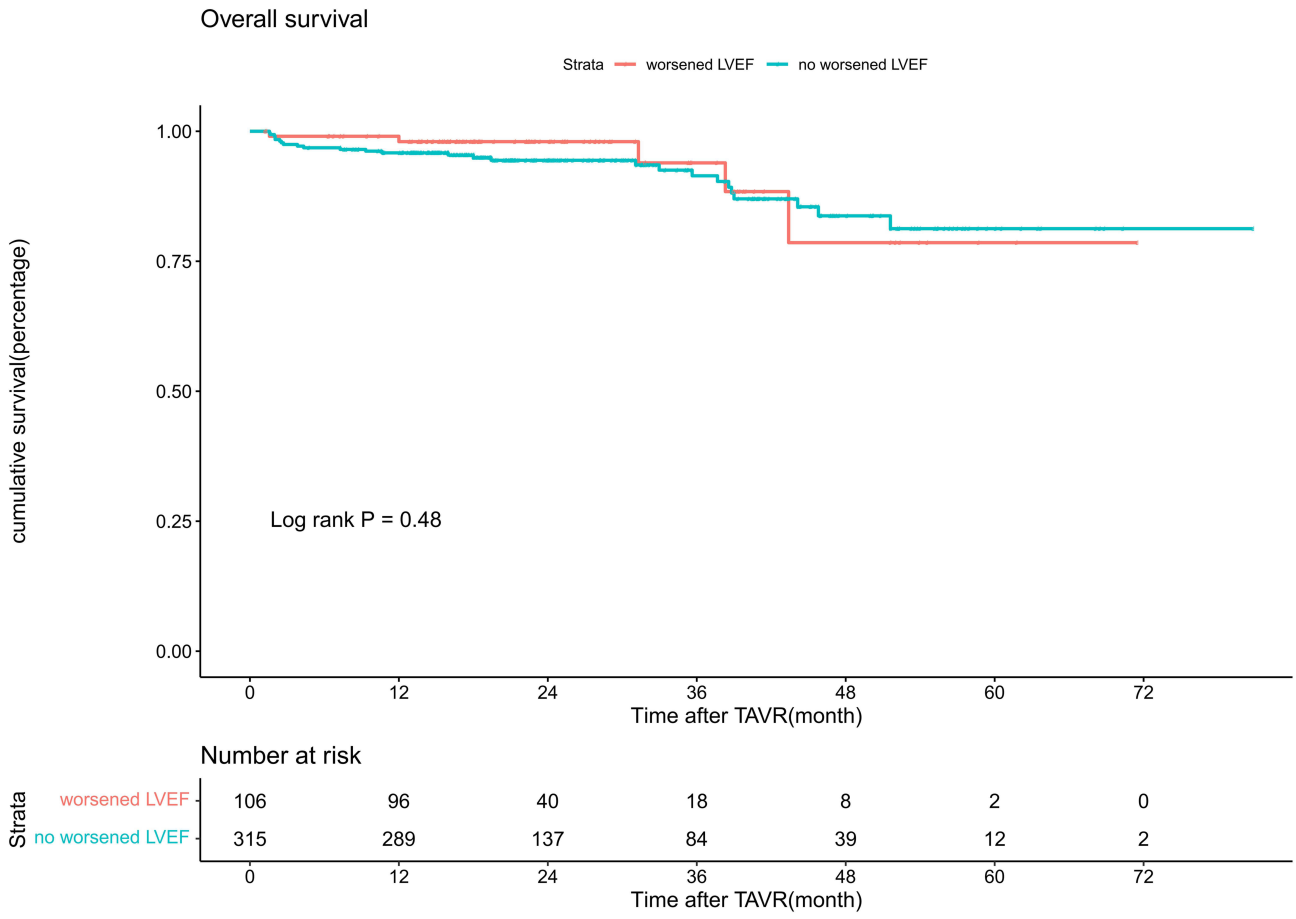

Figure 3 Kaplan–Meier curve in terms of change of LVEF after TAVR.

Left ventricular systolic dysfunction in patients with AS may be caused by ventricular overload, LV hypertrophy and myocardial fibrosis.[13,14] In theory, LVEF would improve early due to the reduction in left ventricular afterload after TAVR. A number of possible mechanisms may account for LVEF decline. Acute myocardial injury during procedure caused by pre- or post-dilation or guide wire injury may lead to myocardial dysfunction. In the acute phase after TAVR, left ventricular remodeling had limited effect on LVEF change before discharge.[5] The change in LVEF after TAVR may be a combination of acute improvement in cardiac function due to reduction in afterload and cardiac dysfunction due to acute myocardial injury.

We observed that LVEF worsening at discharge had no impact on survival compared with patients without worsened LVEF. Our results were consistent with a previous study showing that patients with reduced LVEF did not experience worse survival compared to those with unchanged or improved LVEF following transapical aortic valve implantation.[5] Previous studies showed that pre-operation pulmonary hypertension[15,16] and mitral regurgitation[17] were associated with worse prognosis after TAVR. At baseline, mitral regurgitation and pulmonary hypertension more than moderate are less common in patients with worsened LVEF. It may be one of potential reasons that patients with worsened LVEF did not show worse survival. The other potential reason is LVEF in patients with worsened LVEF regress to the baseline level at one month. A recent report by Kolte et al[4] in a cohort study reported early LVEF improvement within one month after TAVR is associate with less all cause and cardiac death. Similarly, Dauerman et al reported that Early LVEF recovery at 30 days, which was defined as an absolute increase of ≥10% in EF after TAVR, is associated with improved clinical outcomes.[18] Acute LVEF worsening before discharge may be a stress response for procedure and recover in the short term. Therefore, worsened LVEF before discharge has no impact on long-term prognosis. However, it needs more large cohort studies to confirm.

The present study indicates that patients with better pre-operative LVEF have more chance to decline and better pre-operative LVEF is an independent predictor of LVEF worsening. Levi and colleagues reported that higher baseline LVEF, male sex and diabetes were associated with LVEF decline after cardiac intervention therapy within one year.[19] Similarly, Chen et al showed in a large registry study that increased left ventricular end-diastolic dimension, LVEF, high-density lipoprotein cholesterol and atrial fibrillation can predict worsening LVEF.[20] The above studies evaluated the difference between preoperative LVEF and LVEF at follow-up. And these predictors in previous studies may predict chronic declination of LVEF but not acute worsening. However, only higher baseline LVEF is associated with LVEF worsening after procedure in our study. However, only limited parameters were included in the analysis in the present study. Further research is needed to explore other potential risk factors for worsening LVEF. As mentioned above, the change in LVEF after TAVR was a combination of acute improvement in cardiac function and myocardial damage. We hypothesized that patients with better baseline LVEF would have preserved systolic function at baseline and less acute improvement after TAVR. Therefore, patients with a better LVEF at baseline were more likely to have an acute worsening of LVEF after TAVR. We measure LVEF before discharge not at follow-up because we indicate to assess the impact of acute LVEF worsening on prognosis. The results of the present study show that an acute worsening of LVEF after TAVR has little impact on the prognosis. Therefore, routine post-TAVR echocardiography should focus on other metrics, such as mitral regurgitation, pulmonary hypertension and even left ventricular global longitudinal strain, rather than transient LVEF change. Future studies should focus on investigating other potential echocardiographic parameters that may predict prognosis.

Interestingly, we also found that LVEF would recover at one month near the level before procedure in patients with worsened LVEF. The regression of LVEF is likely to be multifactorial. Inflammation reduction, LV remodeling and aortic remodeling may contribute to the recovery. Absence of previous myocardial infarction and higher aortic gradients were identified as predictors of early LVEF recovery in previous study.[18]

Limitations

Several limitations should be noted in our study. First, this is a single center and retrospective study with its natural limitations. The sample size is relatively small, and the follow-up time is not long enough; larger studies with longer follow-up are needed in the future. Second, there is no consensus definition of worsened LVEF after TAVR. A 5% decrease in LVEF may not be the best cut-off. However, this may be an appropriate cut-off as a 5% change may reflect

small changes in the acute post-procedure period after the procedure and reduce the impact of potential measure variability between pre- and post-TAVR. The interobserver variability of LVEF measures is unavoidable. Third, the underlying mechanisms of worsened LVEF are not well understood in our studies. Further studies are needed to investigate potential mechanisms.

Conclusion

Acute worsening of LVEF after TAVR was not uncommon and recovered to baseline at one month. Acute worsening of LVEF had no impact on survival; routine post-TAVR echocardiography should focus on other metrics rather than acute LVEF changes.

Data Sharing Statement

The data underlying this article will be shared on reasonable request to the corresponding author.

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Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

Disclosure

The authors declare that there is no conflict interest.

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