Predictors and Outcomes of Persistent Tricuspid Regurgitation After Transcatheter Aortic Valve Implantation

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Persistent tricuspid regurgitation (TR) after transcatheter aortic valve implantation (TAVI) has been reported to increase mortality. The aim of this study was to investigate clinical and echocardiographic determinants and outcome of persistent TR after TAVI. We reviewed 1,085 patients who underwent TAVI. Among them, 100 patients who had ≥moderate TR without organic dysfunction of the tricuspid valve apparatus were studied. Preprocedural and follow-up transthoracic echocardiography after TAVI were analyzed. After TAVI, patients were divided into persistent TR group and improved TR group. Clinical event was defined as all-cause mortality and readmission for heart failure within 1,000 days. Fifty-three (53%) patients had persistent TR, whereas 47 (47%) patients had improved TR. Risk of clinical event was significantly higher in the persistent TR group compared with the improved TR group. Atrial fibrillation (AF) and tricuspid annular dimension (TAD; p <0.05 for all) were independent predictors of persistent TR. Receiver operating characteristic curve showed the optimal cut-off value of TAD for predicting persistent TR was 37 mm. The combination of AF and TAD ≥37 mm was associated with persistent TR (p <0.001). In conclusion, AF and dilated TAD before TAVI predicted persistent TR which was associated with higher all-cause mortality and readmission for heart failure. **(Am J Cardiol 2019;124:772−780)**

Significant tricuspid regurgitation (TR) commonly accompanies left-sided heart valve disease,[1,2] and is believed to be improved if left-sided heart disease is treated.[3] However, TR is not necessarily improved with correction of left-sided heart disease.[4] Moreover, preprocedural significant TR coexisting with left-sided heart disease has been associated with co-morbidity and mortality.[5,6] In patients with transcatheter aortic valve implantation (TAVI), it is previously reported that the prevalence of ≥moderate TR is around 15% to 25%.[5,7] Recently, McCarthy et al reported that significant TR before TAVI was associated with increased mortality and readmission after TAVI.[8] Furthermore, Schwartz et al reported that persistence of moderate or greater TR at 6 months after TAVI is associated with poor survival even when adjusted for clinical and right ventricular parameters.[9] Therefore, both significant TR concomitant with aortic stenosis and persistent TR after TAVI have been spotlighted lately. However, the preprocedural determinants and outcome of persistent TR after TAVI have not been fully elucidated. The aim of this study is to investigate the clinical and echocardiographic preprocedural determinants and outcome of persistent TR after TAVI.

Methods

We retrospectively reviewed 1,085 patients who underwent TAVI for aortic stenosis at our institution from January 2014 to January 2017. Patients underwent comprehensive clinical evaluation by the designated heart team in our institute and considered appropriate to undergo TAVI as suggested by the guidelines.[10] Among them, 145 patients were found to have ≥moderate TR without organic dysfunction of the tricuspid valve (TV) apparatus before procedure from the transthoracic echocardiographic report. We excluded patients with no preprocedural echocardiographic images within 6 months before TAVI (n = 4), inadequate preprocedural echocardiographic images (n = 1), no follow-up echocardiography after 30 days after TAVI (n = 38), and patients who had undergone TAVI previously (n = 2). The remaining 100 patients who underwent preprocedural and follow-up transthoracic echocardiography after TAVI were eligible for this study (Figure 1). After TAVI, we divided the patients into 2 groups on the basis of persistent TR or improved TR; persistent TR group, defined as those with ≥moderate TR at follow-up, and improved TR group, defined as patients with ≤mild TR at follow-up. This study was approved by the Cedars-Sinai Institutional Review Board.

Clinical and laboratory data were collected from medical records. Laboratory data at baseline and follow-up were collected at the same date as transthoracic echocardiography, respectively. Percent change in B-type natriuretic peptide (BNP) from baseline was calculated as (BNP at follow-up − BNP at baseline)/BNP at baseline × 100 (%). In-hospital complications were assessed according to Valve Academic Research Consortium 2 criteria.[11]

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See page 779 for disclosure information.

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0002-9149/
https://doi.org/10.1016/j.amjcard.2019.05.066

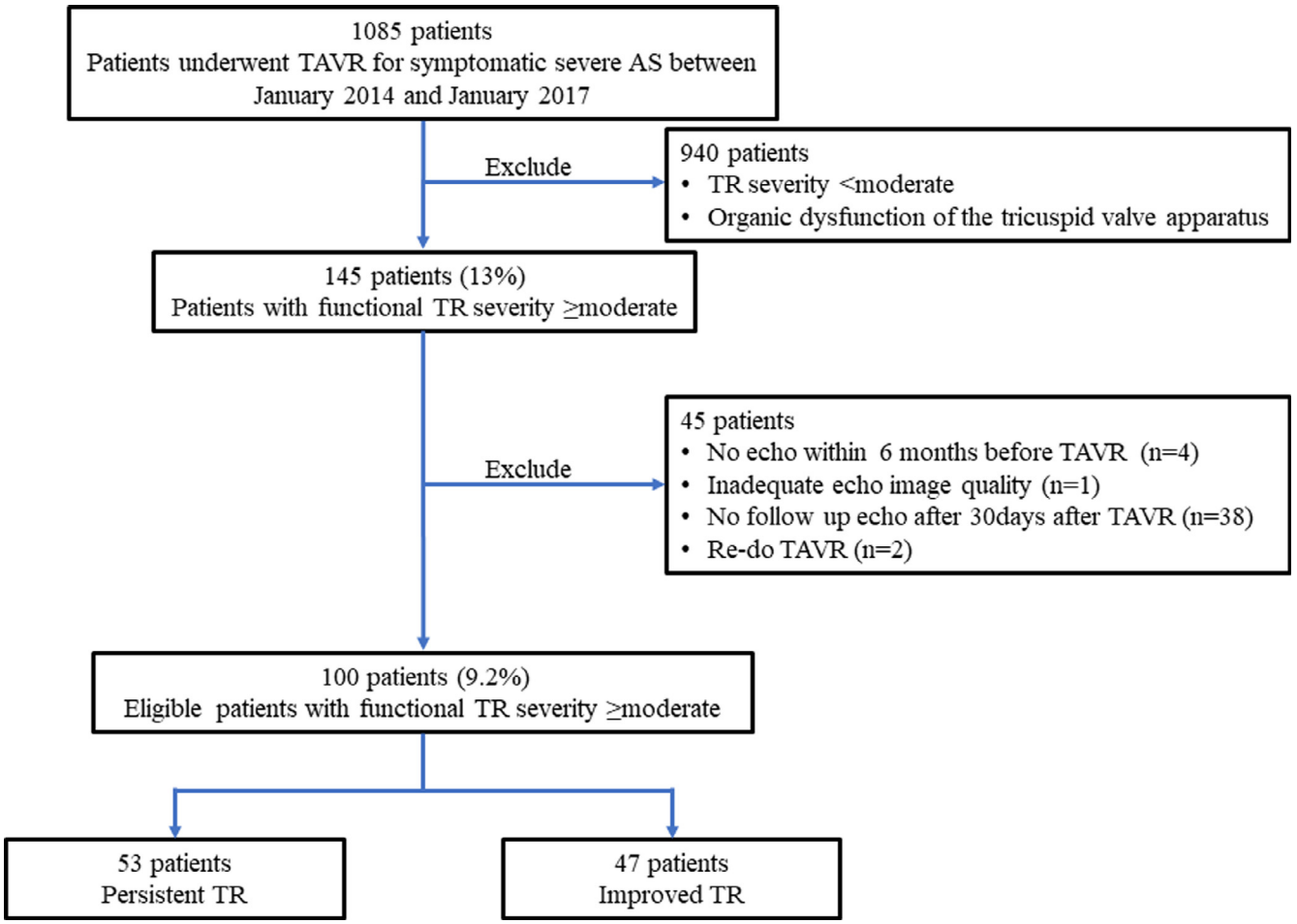

Figure 1. Flowchart of the study population.
AS = aortic stenosis; TAVI = transcatheter aortic valve implantation; TR, tricuspid regurgitation.

Comprehensive transthoracic echocardiography examination was performed before and at follow-up after TAVI according to the current guideline.[12,13] The iE33 ultrasound system (Philips Medical Systems, Andover, Massachusetts) equipped with S5-1 phased array transducer was used. In the patients with AF, all measurements were averages of at least 5 cardiac cycles. Aortic valve area was measured using the continuity equation. Minimal tethering height of tricuspid leaflet was defined as distance of the line vertically descended from the coaptation of the leaflets to the annulus line at mid-systole. Tricuspid annular dimension (TAD) at early diastole was measured from the apical 4-chamber view. TR severity was graded as none, mild, moderate, and severe by the integrative method according to the American Society of Echocardiography guideline.[14,15] The vena contracta width was measured from the apical 4-chamber and parasternal right ventricular inflow views during held end expiration. The averaged vena contracta width from these 2 roughly orthogonal views was calculated to account for its noncircular and ellipsoidal shape.[16]

Clinical event was defined as all-cause mortality and rehospitalization for heart failure within 1,000 days after TAVI. In-hospital death was excluded from the analysis.

Continuous variables are presented as mean ± standard deviation or as median (interquartile range). Student's *t* test or Wilcoxon rank sum test as appropriate were used for continuous variables. Paired *t* test was used to compare systolic pulmonary artery pressure before and after TAVI. Wilcoxon sign-rank test was used to compare BNP before and after TAVI. Categorical data were presented as numbers/percentages and compared between groups using the chi-square test or the Fisher exact test. Odds ratio (OR) was calculated in univariate and adjusted OR were calculated in multivariate logistic regression model with 95% confidence interval (CI). Age, AF, left ventricular ejection fraction, E/e', e', systolic pulmonary artery pressure, right ventricular end-diastolic area, right atrial area, TV tethering height, TAD, mitral regurgitation /mitral stenosis ≥moderate, and severe TR at baseline were respectively analyzed in univariate logistic regression analysis of persistent TR after TAVI. Variables with p value <0.05 in univariate logistic regression were included in the multivariate logistic regression to identify independently associated parameters with persistent TR after TAVI. Receiver operating characteristics curves were generated to determine the optimal cut-off value of TAD for predicting persistent TR after TAVI. Kaplan-Meier analyses were performed using the log-rank test to evaluate the clinical event. For time-to-event outcome, crude hazard ratio (HR) was calculated in univariate and adjusted HR were calculated in multivariate Cox-regression models

with 95% CI including Society of Thoracic Surgeons Predicted Risk of Mortality, tricuspid annular plane systolic excursion, right ventricular fractional area change, systolic pulmonary artery pressure, access site, frailty (inability to walk), in-hospital complications, and severe TR at baseline known to affect patient outcomes after TAVI.[8,17–20] Reproducibility of right heart echocardiographic measurements, as described by absolute difference ± standard deviation and intraclass correlation, was evaluated in 40 data sets 1 month after the initial measurement by the first author for intraobserver variability and by a second observer for interobserver variability. The second observer selected the same data set and frame that the first observer had used and measured independently. All 2-sided p values <0.05 were considered statistically significant. All data were statistically analyzed using the SPSS software package, version 23.0 (SPSS, Chicago, Illinois).

Results

The clinical and echocardiographic characteristics of patients with ≥moderate TR before TAVI are summarized in Table S1 (in the Data Supplement).

Follow-up period of transthoracic echocardiography after TAVI was a median of 364 [112 to 552] days. Changes in TR severity after TAVI in patients with ≥moderate TR are shown in Figure 2. At baseline, TR was graded as severe in 17 patients (17%), and moderate in 83 patients (83%). After TAVI, the TR severity persisted in 53 patients (53%) whereas it was improved in 47 patients (47%). Baseline clinical and echocardiographic characteristics of persistent TR group and improved TR group are shown in Table 1. Compared with improved TR group, the prevalence of AF, Society of Thoracic Surgeons Predicted Risk of Mortality, and the prevalence of severe TR were significantly higher in persistent TR group. TR jet area, vena contracta width, right ventricular end-diastolic area, right atrial area, and TAD were significantly larger in persistent TR group than improved TR group. There was no significant difference in in-hospital complications (Table S2 in the Data supplement). Representative cases of improved and persistent TR after TAVI are shown in Figure 3.

There was also no significant difference in BNP level between persistent TR group and improved TR group at baseline (Table 1). However, BNP level after TAVI showed significant differences between the 2 groups. Figure S1 (in the Data Supplement) shows the change in BNP level after TAVI. In persistent TR group, there was no significant difference in BNP level between before and after TAVI (395 [221 to 857] vs 461 [247 to 789] pg/ml, p = 0.58) whereas BNP level significantly decreased after TAVI in improved TR group (661 [174 to 1,350] vs 223 [123 to 402] pg/ml, p = 0.005). Percent increase in BNP in the persistent TR group was greater than that in the improved TR group after TAVI (2 [−29 to 57] vs −36 [−75 to 17] %, p = 0.003).

In addition, there was no significant difference in systolic pulmonary artery pressure before TAVI between the 2 groups (Table 1). Systolic pulmonary artery pressure significantly decreased after TAVI in improved TR group whereas it did not in the persistent TR group (Figure 4). As a result, there was a significant difference in systolic pulmonary artery pressure after TAVI between the 2 groups (54 ± 18 vs 40 ± 14 mm Hg, p <0.001). However, there were no significant differences in the aortic valve pressure gradients

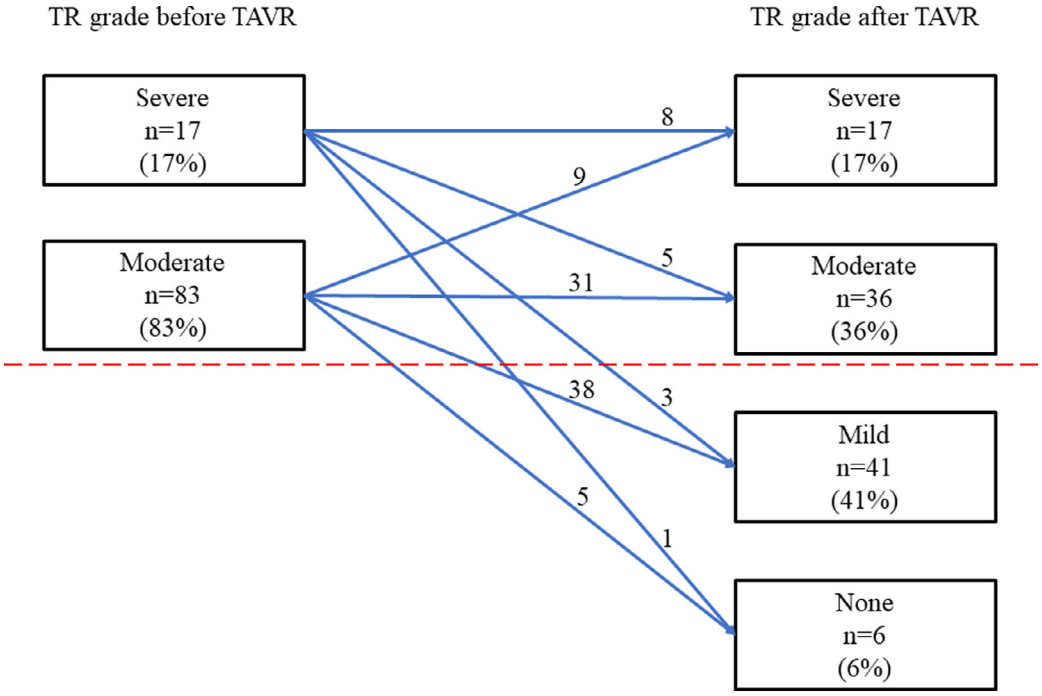

Figure 2. Changes in TR severity after TAVI.
Of the 100 patients, TR was graded as severe in 17 patients (17%), and moderate in 83 patients (83%) at baseline. After TAVI, the TR severity was improved in 47 patients (47%) whereas it persisted in 53 patients (53%)
TR = tricuspid regurgitation; TAVI = transcatheter aortic valve implantation.

Table 1
Comparison of clinical and echocardiographic characteristics between persistent TR group and improved TR group after TAVR

Variable	Tricuspid regurgitation		p value
	Persistent (n = 53)	Improved (n = 47)	
Clinical characteristics			
Age (years)	84 ± 8	82 ± 8	0.33
Men	28 (53%)	24 (51%)	0.86
Body surface area (m)	1.80 ± 0.24	1.86 ± 0.21	0.13
Systolic artery pressure (mm Hg)	130 ± 20	130 ± 17	0.94
Diastolic artery pressure (mm Hg)	67 ± 13	65 ± 14	0.42
Heart rate (bpm)	76 ± 12	78 ± 15	0.68
Atrial fibrillation	39 (74%)	18 (38%)	**<0.001**
New York Heart Association class III/IV	40 (76%)	39 (83%)	0.36
STS-PROM (%)	8.2 [5.7–11.4]	6.3 [4.7–9.7]	**0.020**
Inability to walk	2 (4%)	2 (4%)	0.90
Current smoker	5 (9%)	1 (2%)	0.13
Hypertension	44 (83%)	38 (81%)	0.78
Dyslipidemia	35 (66%)	36 (77%)	0.25
Diabetes mellitus	11 (21%)	11 (23%)	0.75
Coronary artery disease	25 (47%)	24 (51%)	0.70
Post coronary artery bypass graft	19 (36%)	12 (26%)	0.27
Post percutaneous coronary intervention	8 (15%)	11 (23%)	0.29
Hemodialysis	3 (6%)	2 (4%)	0.75
Chronic obstructive pulmonary disease	10 (19%)	4 (9%)	0.14
Pacemaker	19 (36%)	11 (23%)	0.18
Hemoglobin (g/dl)	12 ± 2	12 ± 2	0.66
Creatinine (mg/dl)	1.8 ± 1.3	1.3 ± 0.9	0.06
B-type natriuretic peptide (pg/ml)	395 [221–857]	661 [174–1,350]	0.08
Medications			
Anticoagulant agent	28 (53%)	18 (38%)	0.15
Antiplatelet agent	20 (38%)	22 (47%)	0.36
ACE inhibitors/ARBs	17 (32%)	15 (32%)	0.99
Beta blockers	23 (43%)	21 (45%)	0.90
Calcium channel blockers	9 (17%)	7 (15%)	0.78
Diuretics	34 (64%)	33 (70%)	0.52
Access site			
Transfemoral access	52 (98%)	45 (96%)	0.49
Non-transfemoral access	1 (2%)	2 (4%)	0.49
Transthoracic echocardiography			
Left ventricular end-diastolic volume (ml)	110 ± 51	121 ± 51	0.26
Left ventricular end-systolic volume (ml)	57 ± 42	67 ± 44	0.14
Left ventricular ejection fraction (%)	53 ± 14	49 ± 15	0.17
Stroke volume (ml)	60 ± 13	63 ± 17	0.34
Stroke volume index (ml/m)	33 ± 9	33 ± 9	0.97
Left atrial volume index (ml/m)	54 ± 23	51 ± 24	0.49
e' (cm/s)	8.4 ± 2.3	7.9 ± 2.4	0.38
E/e'	16 ± 7	18 ± 10	0.25
E wave (cm/s)	123 ± 40	128 ± 40	0.55
Aortic valve area (cm)	0.67 ± 0.19	0.68 ± 0.21	0.71
Aortic valve area index (cm/m)	0.37 ± 0.10	0.36 ± 0.13	0.61
Peak transaortic valve velocity (cm/s)	410 ± 66	423 ± 74	0.35
Peak transaortic pressure gradient (mm Hg)	70 ± 22	76 ± 22	0.15
Mean transaortic pressure gradient (mm Hg)	41 ± 14	45 ± 14	0.15
Systolic pulmonary artery pressure (mm Hg)	56 ± 17	55 ± 16	0.87
Right ventricular end-diastolic area (cm)	22 ± 8	19 ± 7	**0.046**
Right ventricular end-systolic area (cm)	13 ± 7	10 ± 6	0.066
Right ventricular fractional area change (%)	43 ± 14	45 ± 14	0.47
Right atrial area (cm)	26 ± 8	22 ± 9	**0.045**
TAPSE (mm)	13 ± 6	14 ± 5	0.41
Tricuspid valve tethering height (mm)	4.1 ± 2.9	4.6 ± 2.5	0.41
Tricuspid annular dimension (mm)	40 ± 6	35 ± 6	**<0.001**
Tricuspid regurgitation severity			**0.033**
Moderate	40 (76%)	43 (91%)	
Severe	13 (24%)	4 (9%)	

(*continued*)

Table 1 (*Continued*)

Variable	Tricuspid regurgitation		p value
	Persistent (n = 53)	Improved (n = 47)	
Tricuspid regurgitation jet area (cm^2)	8.6 ± 3.8	6.7 ± 3.4	**0.010**
Vena contracta (cm)	0.72 ± 0.27	0.61 ± 0.20	**0.036**
Mitral regurgitation moderate	15 (28%)	18 (38%)	0.29
Mitral stenosis moderate	2 (4%)	1 (2%)	0.63

ACE = angiotensin-converting enzyme; ARBs = angiotensin II receptor blockers; STS-PROM = Society of Thoracic Surgeons Predicted Risk of Mortality; TAPSE = tricuspid annular plane systolic excursion.

Bold p values indicate statistically significant (p <0.05).

Value are mean ± SD or median [interquartile range].

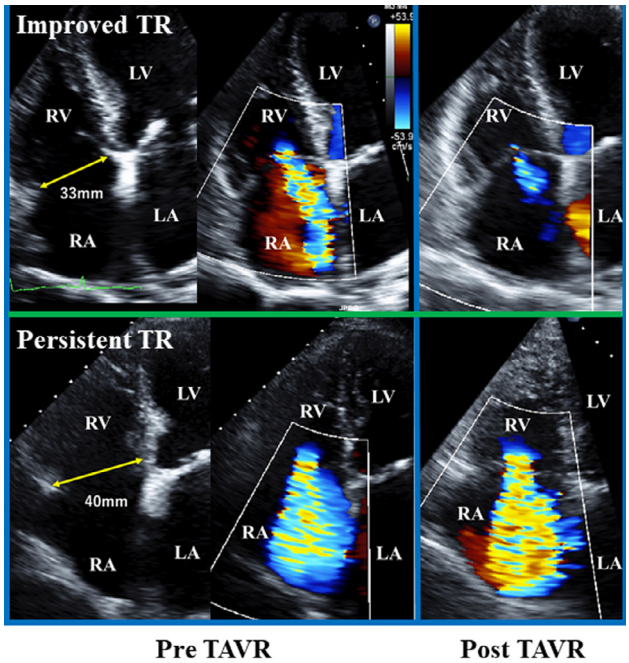

Figure 3. Representative 2 cases with improved TR and persistent TR after TAVI.

Upper panel showed the representative case of improved TR group demonstrating preprocedural TAD of 33 mm. The severity of TR improved after TAVI. Lower panel showed the representative case of persistent TR group demonstrating preprocedural TAD of 40 mm. The severity of TR did not improve after TAVI.

LA = left atrium; LV = left ventricle; RA = right atrium; RV = right ventricle; TAD = tricuspid annular dimension; TAVI = transcatheter aortic valve implantation; TR = tricuspid regurgitation.

and mitral regurgitation/mitral stenosis severity after TAVI (Table S3 in the Data Supplement).

The median follow-up for time-to-event outcome was 439 [207 to 785] days among all 100 patients. Twenty-five (25%) patients had clinical event within 1,000 days (8 patients died and 17 patients needed rehospitalization for heart failure). Kaplan-Meier estimates showed significantly higher rate of clinical event in persistent TR group as compared with improved TR group within 1,000 days (34% vs 19%, p = 0.014; Figure 5). Crude HR for clinical event was 2.86 (95% CI 1.19 to 6.86, p = 0.019) within 1,000 days. Adjusted HRs were shown in Table S4 in the Data Supplement. Clinical event in the persistent TR group was not influenced by baseline factors.

Univariate analysis showed that AF, right ventricular end-diastolic area, TAD, and severe TR at baseline were associated with persistent TR. Multivariate analysis showed that AF and TAD were independent predictors of persistent TR after TAVI (Table 2). We subsequently drew receiver operator characteristic curve to determine the optimal cut-off value for TAD at baseline for persistent TR after TAVI. The optimal cut-off value of TAD at baseline was 37 mm with a sensitivity of 76% and specificity of 62%. The area under the curve was 0.72 (p <0.001; Figure 6). Additionally, there was significant association between persistent TR after TAVI and patients with the combination of AF and TAD ≥37 mm (OR 5.21, 95% CI 2.15 to 12.66, p <0.001).

Intraobserver and interobserver variabilities in right heart echocardiographic measurements were as follows: tethering height of tricuspid leaflet, 0.4 ± 1.1 mm and −0.6 ± 1.5 mm; TAD, −0.3 ± 2.2 and 0.5 ± 3.4 mm; and right ventricular fractional area change, 2.5 ± 8.6 and −2.5 ± 9.7%, respectively. Intraclass correlations (95% CI) for each of the measurements were as follows: tethering height of tricuspid leaflet, 0.92 (0.85 to 0.96) and 0.82 (0.67 to 0.90); TAD, 0.93 (0.87 to 0.96) and 0.84 (0.72 to 0.91); right ventricular fractional area change, 0.78 (0.63 to 0.88) and 0.79 (0.64 to 0.88).

Discussion

Our study demonstrated 3 important findings as follows: (1) 53% of patients with ≥moderate TR at baseline had persistent TR after TAVI. (2) Persistent TR after TAVI had a more than twofold increased risk of all-cause mortality and rehospitalization during long-term follow-up. (3) Predictors of persistent TR after TAVI were the presence of AF and tricuspid annular dilation (≥37 mm).

Schwartz et al reported worse outcomes of persistent TR after TAVI with a small study population (n = 41).[9] Our study in 100 patients was consistent with Schwartz's report. They also found that right ventricular dysfunction was associated with worse outcomes after TAVI. There are other studies referring to right ventricular dysfunction on worse clinical outcome after TAVI.[9,18,21] In our study, we demonstrated that persistent TR had a strong association with outcome after adjustment for right ventricular function parameters used in these previous studies. Worku et al also reported similar result which was consistent with our result.[22] However, the investigators used only univariate analysis. In contrast, we employed multivariate analysis adjusting for clinically important factors.[9,18,20] Additionally, they did not provide quantitative

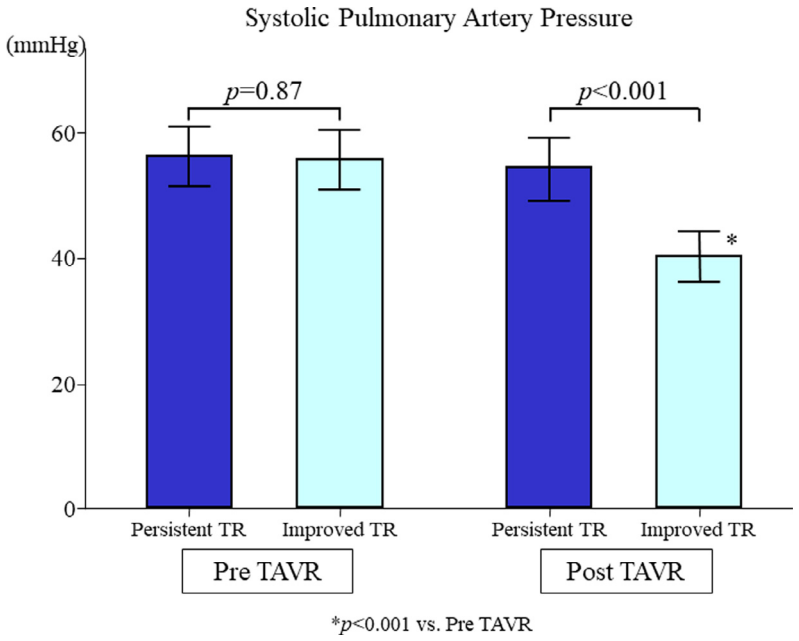

Figure 4. Change in SPAP after TAVI.
There was not significant difference in SPAP before TAVI between persistent TR and improved TR group (56 ± 17 vs 55 ± 16 mm Hg, p = 0.87). In contrast, there was a significant difference in SPAP after TAVI between both groups (54 ± 18 vs 40 ± 14 mm Hg, p <0.001). SPAP significantly decreased after TAVI in improved TR group whereas it didn't in persistent TR group.
TAVI = transcatheter aortic valve implantation; TR = tricuspid regurgitation; SPAP = systolic pulmonary artery pressure.

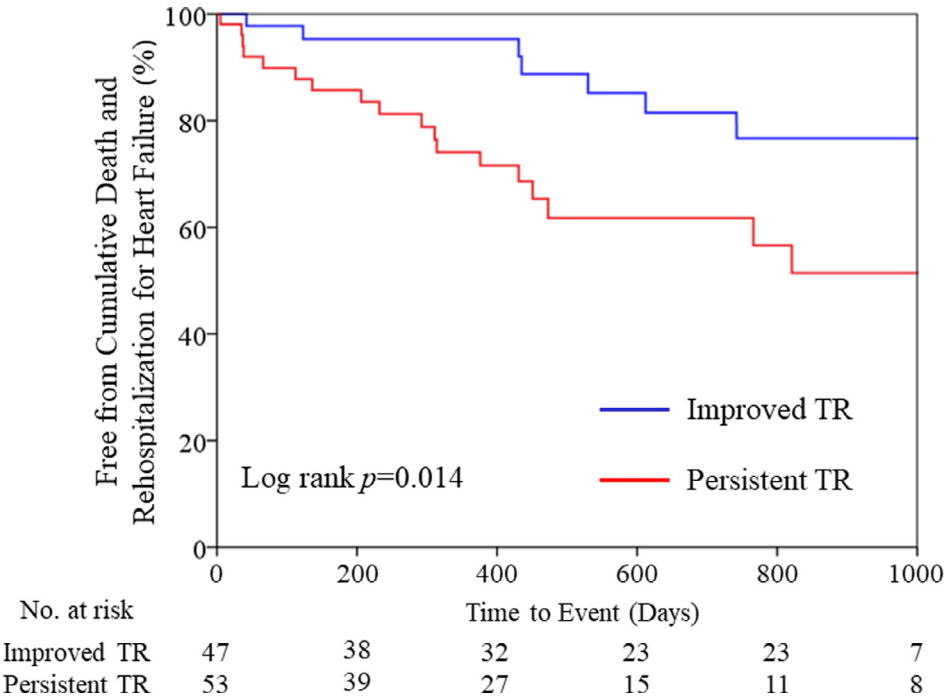

Figure 5. Kaplan-Meier estimates of all-cause mortality and rehospitalization for heart failure within 1,000 days according to presence or absence of persistent TR after TAVI.
During median follow-up of 439 [207 to 785] days, clinical event of all-cause mortality and rehospitalization for heart failure was higher in persistent TR compared with improved TR after TAVI.
TAVI = transcatheter aortic valve implantation; TR = tricuspid regurgitation.

analysis of TV annular size or right ventricular fractional area change.

In this study, we found 2 predictors of persistent TR—procedural AF and TAD. Schwartz et al reported that the factors associated with lack of improvement in TR after TAVI were preprocedural systolic pulmonary artery pressure, TAD, and AF.[9] However, it is not clear in the previous study how many patients had AF, and tricuspid annular

Table 2
Predictors of persistent TR after TAVR

Variables	Univariate analysis		Multivariate analysis	
	Odds ratio (95% CI)	p value	Odds ratio (95% CI)	p value
Age	1.02 (0.97−1.08)	0.35		
Atrial fibrillation	4.49 (1.92−10.48)	**0.001**	2.90 (1.14−7.36)	**0.025**
LV ejection fraction (per 1.0% increase)	1.02 (0.99−1.05)	0.17		
E/e' (per 1.0 decrease)	0.97 (0.92−1.02)	0.25		
e' (per 1.0 cm/s decrease)	1.08 (0.91−1.30)	0.38		
Systolic PA pressure (per 1 mm Hg increase)	1.00 (0.98−1.03)	0.87		
Right ventricular EDA (per 1.0 cm^2 increase)	1.06 (1.00−1.12)	**0.045**	1.01 (0.95−1.07)	0.80
Right atrial area (per 1.0 cm^2 increase)	1.06 (1.00−1.11)	0.051		
TV tethering height (per 1.0 mm increase)	0.94 (0.81−1.09)	0.40		
TAD (per 1.0 mm increase)	1.15 (1.07−1.24)	**<0.001**	1.10 (1.01−1.20)	**0.025**
Mitral regurgitation ≥moderate	0.64 (0.28−1.47)	0.64		
Mitral stenosis ≥moderate	1.80 (0.16−20.56)	0.64		
Severe tricuspid regurgitation at baseline	3.49 (1.05−11.60)	**0.041**	2.56 (0.71−9.28)	0.15

CI = confidence interval; EDA = end-diastolic area; LV = left ventricle; PA = pulmonary artery; STS-PROM = Society of Thoracic Surgeons Predicted Risk of Mortality; TAD = tricuspid annular dimension.

Bold p values indicate statistically significant (p <0.05).

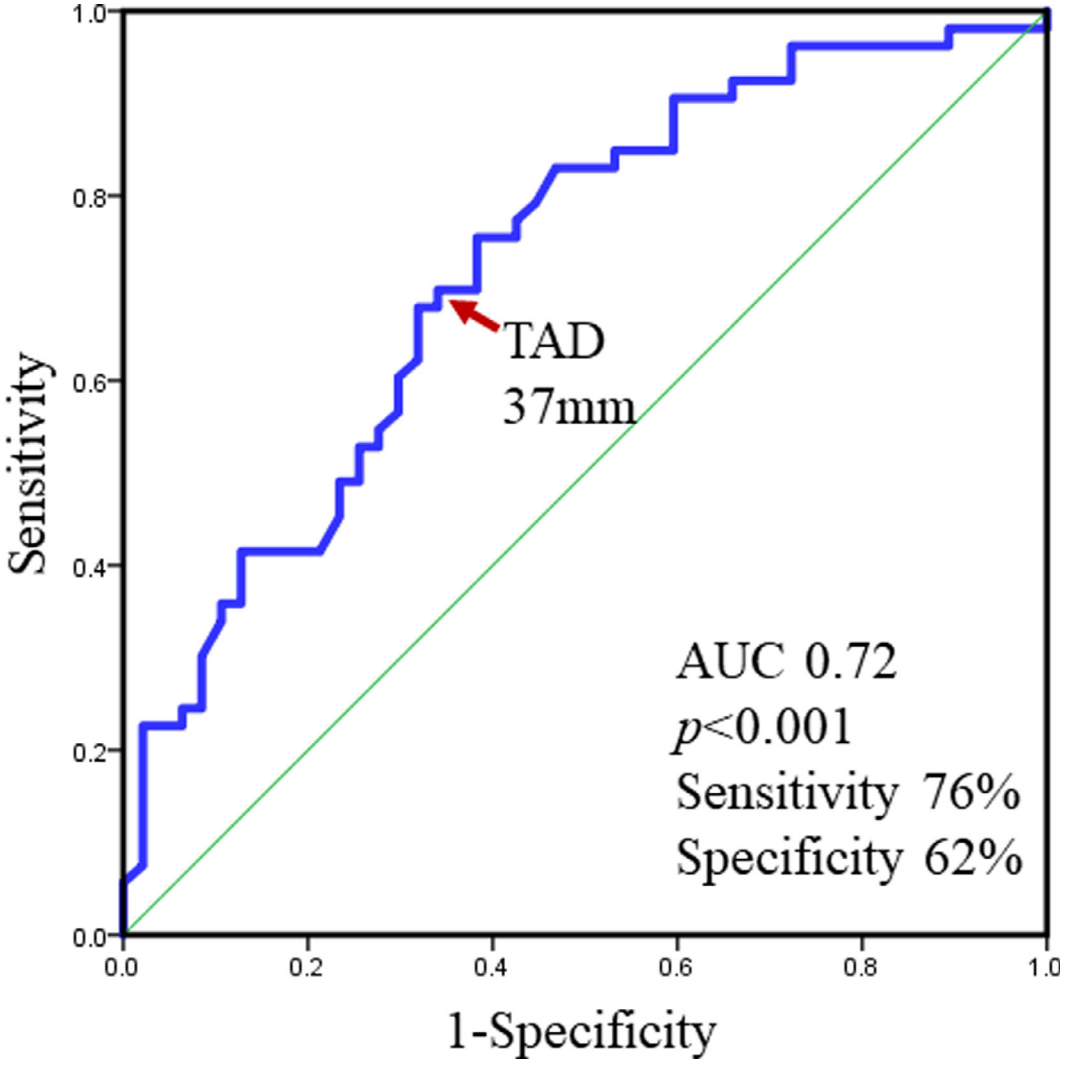

Figure 6. Receiver operating characteristics curve to determine the optimal cut-off value of TAD at baseline for persistent TR after TAVI.
The optimal cut-off value of TAD at baseline for predicting persistent TR after TAVI was 37 mm with a sensitivity of 76% and specificity of 62%. The area under the curve was 0.72 (p <0.001).
AUC = area under the curve; TAD = tricuspid annular dimension; TAVI = transcatheter aortic valve implantation; TR = tricuspid regurgitation.

dilation was not shown in absolute value of the annular size. We did not find preprocedural pulmonary hypertension to be associated with persistent TR. This may be explained by the fact that pulmonary hypertension of the same level existed in both groups before TAVI in our study. However, systolic pulmonary artery pressure significantly improved in patients with improved TR whereas it did not in those with persistent TR after TAVI. The fact that preprocedural pulmonary hypertension was not a predictor of persistent TR after procedure is consistent with results from cases involving left-sided heart surgery in previous studies.[23,24]

Current guidelines state that TV repair or TV replacement should be considered at the time of left-sided valve surgery if the severity of functional TR is severe or TAD is greater than 40 mm on transthoracic echocardiography.[10] A diastolic diameter >40 mm indicates an increased risk of persistent or progressive TR after isolated mitral valve surgery.[10] This was not the case of aortic valve surgery. In our study, we concluded the cut-off value of preprocedural TAD for persistent TR after TAVI was ≥37 mm.

In the present study, AF was present in 57 (57%) patients. It is noteworthy that AF was in 39 (74%) patients of persistent TR group after TAVI. AF itself begets right atrial dilation and tricuspid annular dilation, in association with AF duration. These dilations result in more TR and give way to a vicious cycle of AF and TR.[25] Therefore, it may be optimal to intervene in aortic stenosis and AF before TAD grows larger.

The present study had several limitations. First, this was a single-center study. Second, the right ventricular systolic function was relatively preserved in our patients. Therefore, it is not clear how the results of the present study could be applied to patients with right ventricular systolic dysfunction and dilation. Third, because of the retrospective nature of the present study, the echocardiograms were not obtained specifically to evaluate the anatomy and function of the TV annulus. A prospective study with these specific aims is needed to overcome this limitation. Fourth, sensitivity and specificity of the cut-off value of TAD were relatively weak, even though statistical analysis was significant.

In conclusion, the presence of AF and dilated TAD before TAVI predicted persistent TR which was associated with higher all-cause mortality and readmission for heart failure.

Disclosures

Dr. Yoshida is provided with a grant from Uehara Memorial Foundation, Tokyo, Japan. Dr. Makkar has received grant support from Edwards Lifesciences Corporation, Irvine, CA; and is a consultant for Abbot Vascular, Santa Clara, CA, Cordis, Santa Clara, CA, and Medtronic, and holds equity in Entourage Medical. The other authors have no conflict of interest.

Supplementary materials

Supplementary material associated with this article can be found in the online version at https://doi.org/10.1016/j.amjcard.2019.05.066.

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