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Postoperative Right Ventriculo-Pulmonary Artery Coupling Assessed by TAPSE/PASP as a Predictor of Long-Term Outcomes After TAVI

ABSTRACT

Background: Right ventriculo-pulmonary artery (RV–PA) coupling, commonly assessed by the ratio of tricuspid annular plane systolic excursion to pulmonary artery systolic pressure (TAPSE/PASP), has emerged as an important prognostic marker in various cardiovascular diseases. However, its predictive value after transcatheter aortic valve implantation (TAVI) remains insufficiently defined. This study aimed to evaluate whether postoperative TAPSE/PASP is independently associated with long-term all-cause mortality in patients undergoing transfemoral TAVI.

Methods: We retrospectively analyzed 786 consecutive patients who underwent transfemoral TAVI between June 2020 and March 2025. Postoperative TAPSE/PASP was measured within the first week after the procedure. Receiver-operating characteristic (ROC) curve analysis determined the optimal TAPSE/PASP cut-off for predicting long-term mortality. Survival analyses were performed using the Kaplan–Meier method and Cox proportional hazards regression.

Results: During a median follow-up of 509 days (interquartile range: 283-847), 61 patients (9.0%) died. Receiver-operating characteristic (ROC) analysis identified 0.52 mm/mmHg as the optimal postoperative TAPSE/PASP cut-off (AUC = 0.626, $P < .001$, 95% CI: 0.57-0.68). Patients with TAPSE/PASP <0.52 (n = 278) had worse clinical, echocardiographic, and laboratory profiles than those with TAPSE/PASP ≥0.52 (n = 508). Kaplan–Meier analysis demonstrated significantly reduced survival in the lower TAPSE/PASP group (12.6% vs. 5.1% mortality, log-rank $P < .001$). In multiple Cox regression, age (HR = 1.044, $P = .044$, 95% CI: 1.001-1.089), chronic obstructive pulmonary disease (COPD) (HR = 2.261, $P = .012$, 95% CI: 1.192-4.290), and postoperative TAPSE/PASP (HR = 0.856 per 0.1 mm/mm Hg increase, $P = .033$, 95% CI: 0.743-0.988) remained independent predictors of long-term mortality.

Conclusions: Lower postoperative TAPSE/PASP (<0.52 mm/mmHg) is independently associated with increased long-term mortality after TAVI, supporting its use for early postoperative risk stratification.

Keywords: Right ventricular function, right ventriculo, prognosis, pulmonary artery coupling, transcatheter aortic valve implantation

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INTRODUCTION

Severe aortic stenosis (AS) is a common and progressive valvular disease among the elderly, often remaining undiagnosed until advanced stages.[1,2] Despite the substantial clinical burden, risk stratification remains challenging. Transcatheter aortic valve implantation (TAVI) has become the standard treatment across all surgical risk categories; however, long-term outcomes after the procedure remain heterogeneous, with reported 5-year survival rates of 40-60%.[3] This variability indicates that conventional risk models may not fully capture patient heterogeneity, underscoring the need for novel prognostic markers that reflect cardiac and hemodynamic adaptation beyond left-sided parameters.

Right ventricular (RV) function has gained increasing recognition as an important determinant of outcomes in left-sided valvular diseases. The RV–pulmonary circulation unit is functionally interconnected with left heart hemodynamics through

ventricular interdependence and shared loading conditions.[4] In severe AS, chronic left ventricular pressure overload frequently leads to elevated left atrial pressures, secondary pulmonary hypertension, and progressive RV dysfunction. All these conditions have been associated with adverse clinical outcomes.

The ratio of tricuspid annular plane systolic excursion to pulmonary artery systolic pressure (TAPSE/PASP) has been proposed as a simple and reproducible echocardiographic index of RV–pulmonary artery (PA) coupling.[5] By incorporating both RV contractile function (TAPSE) and afterload (PASP), this ratio provides a more comprehensive assessment of RV performance within its hemodynamic environment compared to isolated measurements. Lower TAPSE/PASP values have been associated with unfavorable prognosis in several cardiovascular conditions, including pulmonary hypertension, heart failure, and valvular heart disease.[6-10]

In patients undergoing TAVI, TAPSE/PASP has recently been explored as a potential prognostic marker. While some studies have demonstrated an association between lower TAPSE/PASP and increased mortality,[11] others have reported inconsistent findings, likely influenced by differences in study design, patient characteristics, and timing of measurement.[12] Notably, most previous studies have predominantly focused on preoperative TAPSE/PASP, aiming to characterize baseline RV–PA uncoupling and its impact on procedural or short-term outcomes. However, preprocedural measurements may not reflect the dynamic changes that occur after relief of valvular obstruction. Given that TAVI itself can substantially alter RV function and pulmonary pressures, postoperative assessments may provide incremental prognostic insights beyond preoperative values. Nevertheless, the prognostic value of postoperative TAPSE/PASP, particularly beyond the early recovery phase, remains insufficiently defined.

Given that TAVI markedly modifies RV loading conditions and pulmonary pressures, postoperative RV–PA coupling may carry distinct prognostic information, integrating both the degree of RV recovery and the residual hemodynamic burden after intervention. The change in coupling (ΔTAPSE/PASP) may further differentiate patients with reversible dysfunction from those with persistent uncoupling. Accordingly, postoperative assessment of RV–PA coupling may represent an early window into right ventricular adaptive capacity following TAVI, allowing identification of patients with persistent hemodynamic vulnerability despite technically successful valve implantation.

The aim of this study was to evaluate the prognostic significance of postoperative right ventriculo–pulmonary artery (RV–PA) coupling, assessed by the TAPSE/PASP ratio, in patients undergoing TAVI. We further sought to examine the perioperative change in TAPSE/PASP (ΔTAPSE/PASP) to determine whether improvement or persistence of RV–PA uncoupling after valve implantation carries prognostic implications for long-term mortality. We hypothesized that postoperative RV–PA coupling reflects the ability of the right ventricle to recover after relief of left-sided pressure overload and therefore provides incremental prognostic information beyond preoperative measurements.

HIGHLIGHTS

- Postoperative tricuspid annular plane systolic excursion to pulmonary artery systolic pressure (TAPSE/PASP) ratio <0.52 mm/mmHg independently predicts long-term mortality after transcatheter aortic valve implantation (TAVI).
- Impaired right ventriculo-pulmonary artery (RV–PA) coupling reflects persistent hemodynamic stress and right ventricular dysfunction despite valve intervention.
- Tricuspid annular plane systolic excursion to pulmonary artery systolic pressure (TAPSE/PASP) offers a simple, non-invasive echocardiographic parameter for postoperative risk stratification in TAVI patients.
- Routine assessment of RV–PA coupling may help identify high-risk individuals who could benefit from closer follow-up and tailored therapies.

METHODS

Study Design and Population

This was a single-center, retrospective observational study including consecutive patients who underwent transfemoral TAVI at our institution between June 1, 2020, and March 1, 2025. Of the total cohort, patients who experienced in-hospital death (n = 73) or had incomplete echocardiographic or clinical data (n = 35) were excluded prior to the outcome analyses. The final study cohort consisted of 786 patients with available postoperative TAPSE/PASP measurements. The study was approved by the institutional ethics committee, and the need for written informed consent was waived owing to the retrospective design. All procedures were performed in accordance with the Declaration of Helsinki.

Data Collection

Baseline demographic characteristics, comorbidities, and laboratory values at admission were obtained from electronic medical records. Pre-procedural coronary calcium burden was quantified by the Agatston score derived from computed tomography scans. Clinical endpoints and follow-up data were collected through hospital records and the national mortality registry.

Echocardiographic Assessment

Transthoracic echocardiographyTTE was performed by experienced sonographers using commercially available ultrasound systems in accordance with American Society of Echocardiography/European Association of Cardiovascular Imaging (ASE/EACVI) recommendations.[13,14] Postoperative echocardiographic examinations were routinely performed between postoperative days 3 and 7 (median 4 [IQR 3-6]), depending on patient stability and discharge timing.

- Tricuspid annular plane systolic excursion (TAPSE) was measured in the apical 4-chamber view with M-mode, aligning the cursor with the lateral tricuspid annulus.

- Pulmonary artery systolic pressure (PASP) was estimated using the peak tricuspid regurgitation velocity and the simplified Bernoulli equation, with right atrial pressure estimated from inferior vena cava size and respiratory variability.
- The TAPSE/PASP ratio was calculated as TAPSE (in mm) divided by PASP (in mm Hg), expressed in mm/mmHg.

Intra- and inter-observer reproducibility were assessed in a randomly selected subset of 30 patients, yielding intraclass correlation coefficients of 0.92 and 0.89, respectively, for TAPSE measurements.

Outcome Measures

The primary outcome was long-term all-cause mortality. The follow-up period was calculated from the date of the procedure to the date of death or last contact, with a median follow-up of 509 (IQR: 283-847) days.

Statistical Analysis

Continuous variables are expressed as mean ± standard deviation or median (interquartile range), depending on data distribution, and categorical variables are presented as counts and percentages. Normality of continuous variables was assessed using the Shapiro–Wilk test. Group comparisons were performed using the Student's *t*-test for normally distributed variables or the Mann–Whitney *U*-test for non-normally distributed variables, and the chi-square test for categorical variables. Within-group pre- and postoperative comparisons of echocardiographic parameters, including TAPSE/PASP, were performed using paired statistical tests (paired Student's *t*-test or Wilcoxon signed-rank test, as appropriate). Between-group differences in periprocedural change (ΔTAPSE/PASP) were assessed using independent group comparisons.

Receiver-operating characteristic (ROC) curve analysis was performed to determine the optimal cut-off value of postoperative TAPSE/PASP for predicting long-term mortality. Based on this analysis, patients were stratified into 2 groups according to the identified cut-off. Survival curves were constructed using the Kaplan–Meier method and compared with the log-rank test.

Univariate Cox proportional hazards regression was conducted to identify variables potentially associated with long-term mortality, including baseline characteristics, comorbidities, postoperative echocardiographic parameters, laboratory findings, and Agatston score. Variables with $P < .10$ in univariate analysis and those deemed clinically relevant were entered into the multiple Cox regression model. Backward stepwise elimination was applied to retain independent predictors of mortality. Hazard ratios (HR) and 95% confidence intervals (CIs) were reported. A 2-tailed *P*-value < .05 was considered statistically significant. All statistical analyses were performed using SPSS software (IBM Corp., Armonk, NY, USA).

RESULTS

A total of 786 patients undergoing transfemoral TAVI were included in the analysis after exclusion criteria were applied. The median follow-up duration was 509 days (IQR: 283-847), during which 61 patients (9.0%) died from all causes.

Receiver operating characteristic (ROC) curve analysis was first performed to determine the prognostic threshold for postoperative TAPSE/PASP. The analysis identified 0.52 mm/mm Hg as the optimal cut-off for predicting long-term mortality, with AUC = 0.626, $P < .001$, 95% CI: 0.57-0.68, sensitivity of 62.1%, and specificity of 60.4%. This cut-off demonstrated modest but statistically significant discriminatory ability and was therefore used to stratify patients for outcome analyses (Figure 1).

Baseline characteristics differed substantially between groups defined by this cut-off. As summarized in Table 1,

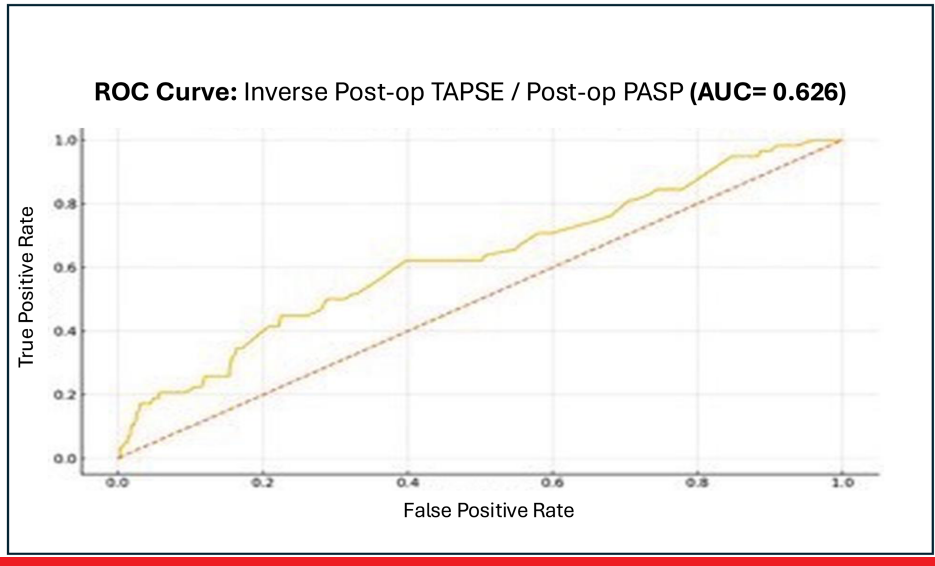

Figure 1. Receiver-operating characteristic (ROC) curve for postoperative tricuspid annular plane systolic excursion to pulmonary artery systolic pressure (TAPSE/PASP). Receiver-operating characteristic curve illustrating the predictive value of postoperative TAPSE/PASP for long-term mortality after transfemoral transcatheter aortic valve implantation (TAVI). The optimal cut-off value of 0.52 mm/mmHg yielded AUC = 0.626, *P* < .001, 95% CI: 0.57-0.68, with sensitivity of 62.1% and specificity of 60.4%.

Table 1. Baseline Clinical, Laboratory, Echocardiographic Characteristics, and Outcomes of Patients Stratified by Post-operative TAPSE/PASP Ratio (<0.52 vs. ≥0.52)

Variables	<0.52 (n = 278)	≥ 0.52 (n = 508)	*P*
Baseline clinical characteristics			
Age (years)	78.40 ± 6.64	77.64 ± 6.30	.116
Sex (1 = Female, 2 = Male)			.008
1	109 (39%)	251 (49%)	
2	169 (61%)	257 (51%)	
HT (0/1)			.943
0	49 (18%)	92 (18%)	
1	229 (82%)	416 (82%)	
CAD (0/1)			.311
0	125 (45%)	249 (49%)	
1	153 (55%)	259 (51%)	
DM (0/1)			.772
0	183 (66%)	341 (67%)	
1	95 (34%)	167 (33%)	
CHF (0/1)			< .001
0	192 (69%)	436 (86%)	
1	86 (31%)	72 (14%)	
COPD (0/1)			1.000
0	238 (86%)	435 (86%)	
1	40 (14%)	73 (14%)	
CVA (0/1)			.047
0	248 (89%)	475 (94%)	
1	30 (11%)	33 (6%)	
AF (0/1)			< .001
0	151 (54%)	405 (80%)	
1	127 (46%)	103 (20%)	
CKD (0/1)			.126
0	219 (79%)	424 (83%)	
1	59 (21%)	84 (17%)	
Agatston Score	2775.00 (1952.00-4123.00)	3076.50 (2124.25-4315.00)	.062
Laboratory findings			
Hgb (g/dL)	11.48 ± 1.91	11.97 ± 1.90	.001
Plt (10^3/μL)	236.44 ± 75.08	231.14 ± 78.99	.288
Wbc (10^3/μL)	7.79 ± 2.90	7.98 ± 5.49	.968
Cre (mg/dL)	1.18 ± 0.74	1.11 ± 0.76	.012
AST (U/L)	27.86 ± 34.59	24.79 ± 37.01	.060
ALT (U/L)	20.31 ± 22.08	18.20 ± 34.91	.209
Albumin (g/dL)	3.88 ± 0.41	3.93 ± 0.42	.017
CRP (mg/L)	15.76 ± 28.58	13.14 ± 25.03	.029
BNP (pg/mL)	1085.00 (476.00-2928.00)	432.00 (182.00-1080.00)	< .001
Post-op platelet (10^3/μL)	194.25 ± 76.55	192.54±75.74	.737
Post-op Cre (mg/dL)	1.16 ± 0.76	1.07 ± 0.70	.132
Post-op AKF (1 = Yes)	24 (8.7%)	24 (4.8%)	.043
Post-op BNP (pg/mL)	481.00 (183.00-1462.00)	227.50 (74.25-580.00)	< .001
Echocardiographic parameters (preoperative)			
LVEF (%)	53.45 ± 13.09	58.27 ± 10.44	< .001
LVEDD (cm)	4.87 ± 0.65	4.72 ± 0.59	< .001
LVESD (cm)	3.29 ± 0.83	3.00 ± 0.71	< .001
PASP (mm Hg)	50.12 ± 15.74	33.90 ± 11.68	< .001
Ao Max Grad (mm Hg)	70.75 ± 22.12	73.94 ± 20.00	.030
Ao Mean Grad (mm Hg)	43.44 ± 14.36	45.91 ± 13.03	.019
AVA (cm^2)	0.64 ± 0.21	0.69 ± 0.18	< .001
Ao Vmax (m/s)	4.14 ± 0.67	4.26 ± 0.61	.031

(*Continued*)

Table 1. Baseline Clinical, Laboratory, Echocardiographic Characteristics, and Outcomes of Patients Stratified by Post-operative TAPSE/PASP Ratio (<0.52 vs. ≥0.52) (Continued)

Variables	<0.52 (n = 278)	≥ 0.52 (n = 508)	*P*
LVOT diameter (cm)	1.93 ± 0.19	1.96 ± 0.20	.066
Ao VTI (cm)	96.20 ± 22.37	97.43 ± 21.10	.393
Lvot VTI (cm)	20.41 ± 6.10	22.91 ± 7.53	< .001
TAPSE (mm)	1.81 ± 0.35	2.07 ± 0.32	< .001
MR (Grade 0-4)			< .001
0	22 (8%)	35 (7%)	
1	81 (29%)	280 (55%)	
2	121 (44%)	165 (32%)	
3	52 (19%)	26 (5%)	
4	2 (1%)	1 (0%)	
AR (Grade 0-4)			.016
0	59 (21%)	135 (27%)	
1	123 (44%)	253 (50%)	
2	77 (28%)	101 (20%)	
3	17 (6%)	16 (3%)	
4	2 (1%)	2 (0%)	
TR (Grade 0-4)			< .001
0	4 (1%)	67 (13%)	
1	72 (26%)	326 (64%)	
2	126 (45%)	99 (19%)	
3	65 (23%)	13 (3%)	
4	11 (4%)	2 (0%)	
LFLGAS (1 = yes)	53 (19%)	46 (9%)	< .001
Echocardiographic parameters (post-operative)			
Post-op LVEF (%)	54.44 ± 12.33	59.43 ± 9.08	< .001
Post-op TAPSE (mm)	1.77 ± 0.29	2.04 ± 0.31	< .001
Post-op PASP (mm Hg)	47.88 ± 12.02	28.51 ± 5.74	< .001
Post-op Ao Max Grad (mm Hg)	16.42 ± 7.31	17.51 ± 7.03	.016
Post-op Ao Mean Grad (mm Hg)	8.64 ± 4.69	9.19 ± 4.37	.032
Post-op Ao Vmax (m/s)	1.54 ± 0.52	1.60 ± 0.54	.228
Post-op MR (Grade 0-4)			< .001
0	27 (10%)	71 (14%)	
1	109 (39%)	342 (67%)	
2	111 (40%)	73 (14%)	
3	30 (11%)	11 (2%)	
4	1 (0%)	0 (0%)	
Post-op AR (Grade 0-4)			.002
0	121 (44%)	284 (56%)	
1	126 (45%)	183 (36%)	
2	28 (10%)	28 (6%)	
3	3 (1%)	3 (1%)	
4	0 (0%)	0 (0%)	
Post-op TR (Grade 0-4)			< .001
0	1 (0%)	110 (22%)	
1	86 (31%)	335 (66%)	
2	133 (48%)	44 (9%)	
3	49 (18%)	7 (1%)	
4	9 (3%)	1 (0%)	
Operative outcomes and clinical endpoints			
Long-term mortality (1 = yes)	35 (12.6%)	26 (5.1%)	< .001
Follow-up duration (days)	506 (274-847.75)	511 (289-843.25)	.814
Moderate/severe PVL (1 = yes)	32 (12%)	36 (7%)	.059

AF, Atrial fibrillation; AKF, acute kidney failure; ALT, alanine aminotransferase; AR, aortic regurgitation; AST, aspartate aminotransferase; AVA, aortic valve area; BNP, B-type natriuretic peptide; CAD, coronary artery disease; CHF, congestive heart failure; CKD, chronic kidney disease; COPD, chronic obstructive pulmonary sisease; CRP, C-reactive protein; Cre, creatinine; CVA, cerebrovascular accident; DM, diabetes mellitus; Hgb, hemoglobin; HT, hypertension; LFLGAS, low-flow; low-gradient aortic stenosis; LVEDD, left ventricular end-diastolic diameter; LVEF, left ventricular ejection fraction; LVESD, left ventricular end-systolic diameter; LVOT, left ventricular outflow tract; MR, mitral regurgitation; PASP, pulmonary artery systolic pressure; Plt, platelet; Post-op, postoperative; PVL, paravalvular leak; TAPSE, tricuspid annular plane systolic excursion; TR, tricuspid regurgitation; VTI, velocity time integral; Wbc, white blood cell.

patients with postoperative TAPSE/PASP <0.52 (n = 278) were older (78.4 ± 6.6 vs. 77.6 ± 6.3 years, *P* = .116), although this difference was not statistically significant. Clinically, patients with postoperative TAPSE/PASP <0.52 exhibited a higher prevalence of congestive heart failure (31% vs. 14%, *P* < .001), atrial fibrillation (46% vs. 20%, *P* < .001), and cerebrovascular disease (11% vs. 6%, *P* = .047). They also showed evidence of more advanced systemic congestion and neurohormonal activation, with significantly higher CRP (15.7 ± 28.6 vs. 13.1 ± 25.0 mg/L, *P* = .029) and BNP levels (1085 vs. 432 pg/mL, *P* < .001). Although serum albumin was slightly lower in this group (3.88 ± 0.41 vs. 3.93 ± 0.42 g/dL, *P* = .017), this difference is likely of limited clinical relevance. Echocardiographic assessment showed that these patients had worse preoperative cardiac function, characterized by lower LVEF (53.4 ± 13.1% vs. 58.3 ± 10.4%, *P* < .001) and higher PASP (50.1 ± 15.7 vs. 33.9 ± 11.7 mm Hg, *P* < .001). Postoperatively, impaired RV–PA coupling persisted, with significantly elevated pulmonary pressures (47.9 ± 12.0 vs. 28.5 ± 5.7 mm Hg, *P* < .001) and reduced TAPSE (17.7 ± 2.9 vs. 20.4 ± 3.1 mm, *P* < .001).

During follow-up, patients with TAPSE/PASP <0.52 experienced markedly higher mortality compared with those with preserved RV–PA coupling (≥0.52) (12.6% vs. 5.1%, *P* < .001). The Kaplan–Meier survival curve (Figure 2) demonstrated an early and persistent divergence in survival trajectories, indicating that impaired postoperative RV–PA coupling was associated with sustained adverse outcomes. The log-rank test confirmed that this difference was statistically significant (*P* < .001).

In the univariate Cox regression analysis (Table 2), several factors were significantly associated with increased long-term mortality, including age, chronic obstructive pulmonary disease (COPD), atrial fibrillation, renal dysfunction (creatinine), hypoalbuminemia, elevated C-reactive protein, reduced postoperative left ventricular ejection fraction, and lower postoperative TAPSE/PASP. Notably, postoperative TAPSE/PASP was strongly associated with outcome, with HR = 0.808, *P* = .002, 95% CI: 0.708-0.922 per 0.1 mm/mmHg increase.

In the multiple Cox regression analysis (Table 3), after adjustment for potential confounders, only age (HR = 1.044, *P* = .044, 95% CI: 1.001-1.089), COPD (HR = 2.261, *P* = .012, 95% CI: 1.192-4.290), and postoperative TAPSE/PASP (HR = 0.856 per 0.1 mm/mm Hg increase, *P* = .033, 95% CI: 0.743-0.988) remained independent predictors of long-term mortality. These associations and their relative strengths are illustrated in the forest plot (Figure 3), which highlights the prognostic significance of postoperative RV–PA coupling.

Paired pre- and postoperative echocardiographic data were available in a subset of patients (n = 723). When stratified according to postoperative TAPSE/PASP category, the ratio increased from 0.36 ± 0.09 to 0.37 ± 0.10 in the <0.52 group and from 0.61 ± 0.12 to 0.72 ± 0.13 in the ≥0.52 group (*P* < .001 for both). The mean change (ΔTAPSE/PASP) was +0.01 ± 0.11 and +0.11 ± 0.12, respectively (*P* < .001 between groups). These findings, summarized in Table 4, demonstrate that RV–PA coupling improved significantly only in patients with preserved postoperative TAPSE/PASP, supporting the concept of persistent uncoupling in those with lower postoperative ratios.

Overall, the identified threshold of 0.52 mm/mm Hg effectively discriminated patients at higher risk of adverse outcomes. Those with impaired postoperative RV–PA coupling not only presented with a worse clinical and echocardiographic profile but also experienced significantly higher long-term mortality. Importantly, postoperative TAPSE/PASP preserved its prognostic value even after adjusting for

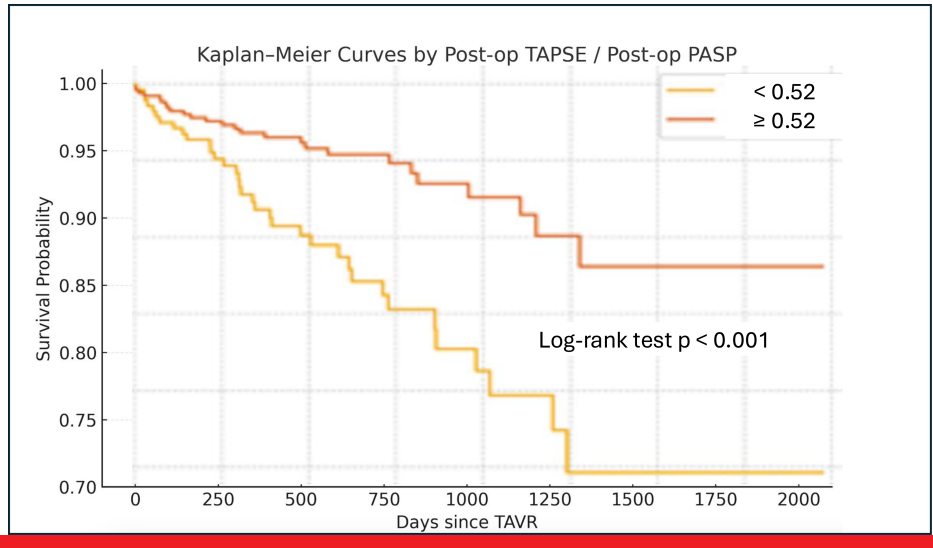

Figure 2. Kaplan–Meier survival curves stratified by postoperative tricuspid annular plane systolic excursion to pulmonary artery systolic pressure (TAPSE/PASP). Kaplan–Meier survival analysis comparing patients with postoperative TAPSE/PASP <0.52 versus ≥0.52 mm/mmHg. Patients with impaired right ventriculo-pulmonary artery (RV–PA) coupling (<0.52) exhibited significantly reduced survival during follow-up. The log-rank test confirmed a statistically significant difference between groups (*P* < .001).

Table 2. Univariate Cox Regression Analysis for the Prediction of Long-term Mortality

Variables	Hazard Ratio	95% Confidence Interval	*P*
Baseline clinical characteristics			
Age (per 1-year increase)	1.044	1.002-1.087	.037
Sex (male)	0.936	0.566-1.548	.797
Hypertension	0.861	0.466-1.589	.632
Coronary artery disease	1.573	0.932-2.653	.089
Diabetes mellitus	1.260	0.751-2.117	.380
Chronic obstructive pulmonary disease	2.024	1.114-3.680	.020
History of cerebrovascular accident	1.629	0.739-3.588	.225
Atrial fibrillation	2.034	1.229-3.366	.005
Laboratory parameters at admission			
Hemoglobin (per 1 g/dL increase)	0.891	0.781-1.016	.087
Creatinine (per 1 mg/dL increase)	1.274	1.023-1.588	.030
Aspartate aminotransferase (per 1 IU/L increase)	1.003	1.000-1.006	.047
Alanine aminotransferase (per 1 IU/L increase)	1.002	0.997-1.006	.385
Albumin (per 1 g/dL increase)	0.601	0.401-0.902	.014
C-reactive protein (per 1 mg/L increase)	1.008	1.003-1.014	.001
Post-operative echocardiographic parameters			
Left ventricular ejection fraction (per 1% increase)	0.969	0.950-0.988	.002
TAPSE/PASP ratio (per 0.01 mm/mm Hg increase)	0.808	0.708-0.922	.002
Maximal aortic gradient (per 1 mm Hg increase)	0.954	0.913-0.997	.036
Moderate-to-severe paravalvular leak	1.658	0.815-3.374	.163
Pre-operative computed tomography			
Agatston Score	0.745	0.469-1.182	.211

other risk factors, confirming its role as an independent and clinically relevant marker for risk stratification in the TAVI population.

DISCUSSION

In this retrospective analysis of patients undergoing transfemoral TAVI, we observed that a lower postoperative TAPSE/PASP ratio (<0.52 mm/mm Hg) revealed an independent association with increased risk of long-term mortality. Notably, this parameter remained an independent predictor after adjustment for established clinical and echocardiographic risk factors. These findings suggest that RV–PA coupling, as assessed by TAPSE/PASP, retains prognostic relevance beyond procedural success and conventional determinants of outcome. Importantly, our additional analysis of ΔTAPSE/PASP demonstrated that RV–PA coupling improved significantly only in patients with preserved postoperative ratios, suggesting the persistence of RV–PA uncoupling among those with lower postoperative TAPSE/PASP values. This observation highlights the concept that the extent of RV functional recovery after TAVI, rather than

Table 3. Multivariate Cox Regression Analysis for the Prediction of Long-Term Mortality

Variables	Hazard Ratio	95% Confidence Interval	*P*
Age (per 1-year increase)	1.044	1.001-1.089	.044
Sex (male)	1.001	0.564-1.776	.997
Chronic obstructive pulmonary disease	2.261	1.192-4.290	.012
Atrial fibrillation	1.311	0.752-2.287	.339
Hemoglobin (per 1 g/dL increase)	0.947	0.817-1.098	.471
Creatinine (per 1 mg/dL increase)	1.218	0.920-1.614	.167
Aspartate aminotransferase (per 1 IU/L increase)	1.001	0.997-1.006	.459
Albumin (per 1 g/dL increase)	0.884	0.447-1.747	.723
C-reactive protein (per 1 mg/L increase)	1.003	0.995-1.010	.473
Post-op left ventricular ejection fraction (per 1% increase)	0.983	0.961-1.007	.162
Post-op TAPSE/PASP ratio (per 0.01 mm/mm Hg increase)	0.856	0.743-0.988	**.033**
Post-op maximal aortic gradient (per 1 mm Hg increase)	0.962	0.917-1.008	.108

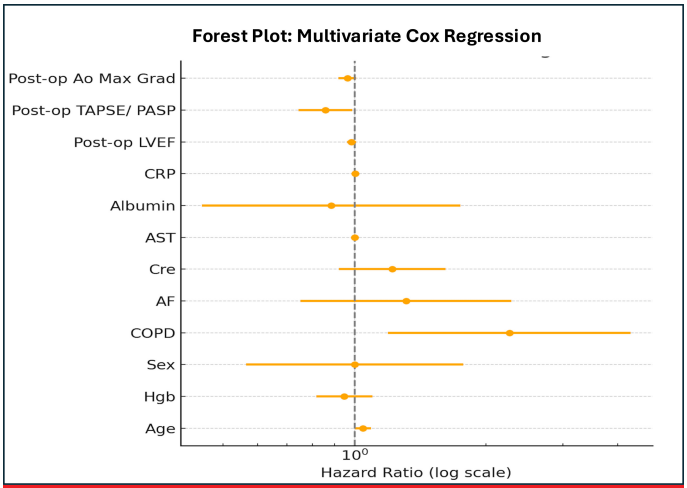

Figure 3. Forest plot of multiple Cox regression analysis. Forest plot depicting the independent predictors of long-term mortality after transcatheter aortic valve implantation (TAVI) identified in the multiple Cox regression model. Age, chronic obstructive pulmonary disease (COPD), and postoperative tricuspid annular plane systolic excursion to pulmonary artery systolic pressure (TAPSE/PASP) were retained as significant predictors, with postoperative TAPSE/PASP demonstrating an independent protective effect (per 0.1 mm/mm Hg increase; HR 0.856, 95% CI: 0.743-0.988, *P* = .033).

the preprocedural status alone, may be a critical determinant of long-term prognosis.

Our findings are consistent with and extend previous evidence regarding the prognostic significance of RV–PA coupling in patients undergoing TAVI. Sultan et al[11] (2019) demonstrated that a lower baseline TAPSE/PASP ratio was independently associated with increased mortality following the procedure. In a subsequent study, Adamo and colleagues (2022) confirmed that impaired RV–PA coupling identified patients at higher risk, irrespective of traditional surgical risk scores.[15] However, the majority of earlier studies primarily focused on preoperative assessments, which characterize chronic RV–PA uncoupling but not the dynamic hemodynamic recovery that follows TAVI.[11,15,16] More recently, Mendes et al[12] (2024) reported that postoperative TAPSE/PASP measurements offered superior prognostic discrimination compared with preprocedural values, highlighting the importance of RV functional adaptation and pulmonary pressure reduction after valve intervention. Our findings reinforce and extend these observations, demonstrating that early postoperative TAPSE/PASP not only reflects residual hemodynamic burden but also encapsulates the degree of RV recovery, thereby serving as an integrative marker of both structural and functional remodeling.

Table 4. Pre- and Postoperative TAPSE/PASP Ratio and its Change (Δ) According to Postoperative Coupling Group

Variables	<0.52 (n = 278)	≥ 0.52 (n = 508)	*P*
Pre-op TAPSE/PASP, cm/mm Hg	0.36 ± 0.09	0.61 ± 0.12	< .001
Post-op TAPSE/PASP, cm/mm Hg	0.37 ± 0.10	0.72 ± 0.13	< .001
Δ TAPSE/PASP, cm/ mm Hg	+ 0.01 ± 0.11	+ 0.11 ± 0.12	< .001

PASP, pulmonary artery systolic pressure; TAPSE, tricuspid annular plane systolic excursion.

Lillo et al[17] (2022) investigated RV systolic function and RV–PA coupling in patients with severe AS, focusing on early changes after TAVI. They reported that while early postoperative RV systolic function did not show significant improvement, a reduction in pulmonary artery systolic pressure led to an increase in TAPSE/PASP, indicating partial restoration of RV–PA coupling. In our cohort, the prognostic association of postoperative TAPSE/PASP beyond the early phase suggests that long-term outcomes are influenced by the persistence or reversal of uncoupling, supporting the hypothesis that early hemodynamic unloading does not uniformly translate into sustained myocardial recovery. Future prospective studies incorporating serial echocardiographic follow-up could clarify whether normalization of TAPSE/PASP over time confers durable prognostic benefit.

Furthermore, recent meta-analytic data support the broader prognostic role of TAPSE/PASP across cardiovascular conditions. In a systematic review and meta-analysis including nearly 9,000 patients with heart failure, Anastasiou et al[18] (2023) reported that lower TAPSE/PASP values were strongly associated with increased all-cause mortality, with patients below the commonly reported threshold of 0.36 mm/mmHg experiencing a nearly threefold higher risk of death. Although this meta-analysis focused primarily on heart failure cohorts, the consistent prognostic signal across different patient populations reinforces the clinical relevance of RV–PA coupling assessment. Taken together, these findings suggest that TAPSE/PASP integrates the hemodynamic and functional dimensions of RV performance, providing incremental prognostic value not only in heart failure but also in the post-TAVI setting examined in our study.

The mechanisms underlying this association are likely multifactorial. Severe aortic stenosis is characterized by chronic left ventricular pressure overload leading to secondary pulmonary hypertension, increased RV afterload, and progressive structural remodeling with fibrosis and impaired contractile reserve.[19] Although TAVI reduces left-sided pressures, the pulmonary vasculature may exhibit fixed remodeling, limiting afterload reduction for the RV. Consequently, persistent RV–PA uncoupling after TAVI may reflect advanced myocardial and vascular remodeling rather than technical procedural failure. The observed associations between lower TAPSE/PASP, higher BNP levels, and a greater prevalence of atrial fibrillation in our cohort support the concept of ongoing myocardial stress and impaired reverse remodeling in these patients.

The threshold identified in our analysis (0.52 mm/mm Hg) lies toward the upper range of those previously reported, which have varied from 0.32 to 0.55 mm/mmHg.[12,15] Differences in patient selection, procedural techniques, and timing of assessments may account for this variability. Nevertheless,

the consistent direction of findings across studies supports the clinical relevance of impaired RV–PA coupling as a marker of increased risk.

The ΔTAPSE/PASP analysis further underscores that only patients demonstrating postoperative RV–PA recoupling achieved significant hemodynamic improvement, highlighting the prognostic importance of early RV adaptation after TAVI. From a clinical perspective, TAPSE/PASP is an easily obtainable, reproducible parameter that can be incorporated into routine postoperative echocardiographic evaluations without additional resource requirements. Identification of patients with impaired postoperative RV–PA coupling may guide closer follow-up, tailored heart failure management, and targeted therapies aimed at reducing pulmonary pressures or improving RV contractile performance. However, given the moderate discriminatory ability observed in our cohort, TAPSE/PASP should be used as part of a multiparametric risk assessment strategy—in conjunction with clinical variables, biomarkers, and advanced imaging indices—rather than as a standalone prognostic tool. Future studies integrating TAPSE/PASP into comprehensive risk models or machine-learning frameworks could refine patient stratification and optimize post-TAVI care.

Limitations

Several limitations should be acknowledged. First, this was a single-center, retrospective study, which may limit generalizability and introduce selection bias. Second, echocardiographic evaluations were performed between postoperative days 3 and 7 (median 4 [IQR 3-6]); although this reflects real-world practice, the lack of serial imaging beyond this period precluded assessment of long-term temporal trends in RV–PA coupling. Third, advanced imaging modalities such as right ventricular strain analysis and quantitative assessment of tricuspid regurgitation were not systematically available, limiting comprehensive evaluation of RV function. Fourth, the primary endpoint was all-cause mortality; differentiation between cardiovascular and non-cardiovascular deaths was not feasible because of incomplete adjudication data. Fifth, the study period overlapped with the COVID-19 pandemic, which may have influenced patient selection and follow-up patterns. Finally, although multivariable adjustments were performed, the potential for residual confounding due to unmeasured variables—such as medication optimization or heart failure management—cannot be excluded. Despite these limitations, the study provides novel, hypothesis-generating evidence that warrants prospective multicenter validation.

CONCLUSION

In summary, this study demonstrates that a lower postoperative TAPSE/PASP ratio (<0.52 mm/mm Hg) is independently associated with increased long-term mortality, reflecting persistent right ventriculo–pulmonary artery (RV–PA) uncoupling despite technically successful TAVI. These findings emphasize the clinical value of postoperative RV–PA coupling as an integrative marker of residual pulmonary load and right ventricular recovery, suggesting that its routine assessment may enhance early post-TAVI risk reclassification and guide closer clinical follow-up or targeted therapy in high-risk patients. Prospective multicenter studies incorporating serial echocardiographic and advanced imaging assessments are needed to validate these observations, determine optimal timing and thresholds for TAPSE/PASP evaluation, and clarify whether interventions aimed at improving RV–PA coupling can translate into better long-term outcomes after TAVI.

Ethics Committee Approval: This study was approved by the Ethics Committee of Koşuyolu High Specialization Training and Research Hospital (Approval No: 2025/12/1198, Date: 22/07/2025).

Informed Consent: Informed consent was waived due to the retrospective nature of the study.

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