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ORIGINAL RESEARCH: STRUCTURAL

Incidence, Clinical Implications, and Predictors of Paravalvular Leak Following Transcatheter Tricuspid Valve Replacement

The TRIPLACE Registry

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ABSTRACT

BACKGROUND The clinical efficacy of transcatheter tricuspid valve replacement (TTVR) in abolishing tricuspid regurgitation might be attenuated by the occurrence of paravalvular leak (PVL).

OBJECTIVES The aim of this study was to investigate the incidence, outcomes, and predictors of moderate or severe PVL post-TTVR.

METHODS All eligible patients undergoing TTVR in the multicenter TRIPLACE (Global Multicenter Registry on Transcatheter Tricuspid Valve Replacement) registry were stratified according to predischarge PVL severity. The primary endpoint was the occurrence of moderate or severe PVL post-TTVR. Secondary endpoints were 1-year mortality, 1-year heart failure hospitalization, and post-TTVR NYHA functional class III or IV. Outcomes were analyzed using logistic regression (PVL), Cox regression (mortality), and Gray's test (heart failure).

RESULTS Of 394 TTVR patients, 24 (6.1%) had moderate or severe, 88 (22.3%) mild, and 282 (71.6%) no or trace PVL post-TTVR. Patients with moderate or severe PVL had significantly higher TRI-SCOREs ($P < 0.001$), lower estimated glomerular filtration rates ($P = 0.001$), and larger right ventricular and right atrial dimensions ($P = 0.002$ for both). Patients with moderate or severe PVL post-TTVR had worse 1-month functional class (53% in NYHA functional class III or IV) than those with mild (16%) or no or trace (15%) PVL. The presence of moderate or severe PVL post-TTVR was associated with increased 1-year mortality (39.7%; adjusted HR: 2.6; 95% CI: 1.2-5.7) compared with those with mild (12.6%) or no or trace (10.5%) PVL. A larger right atrial volume ($P = 0.02$), device malposition ($P = 0.0002$), and type IV valve morphology ($P = 0.01$) were independently associated with moderate or severe PVL post-TTVR.

CONCLUSIONS Moderate or greater PVL occurred in 6.1% of patients post-TTVR and was associated with increased 1-year mortality and worse functional status. A larger right atrial volume, device malposition, and type IV valve morphology conferred a higher risk for developing moderate or severe PVL post-TTVR. These findings have important implications for future TTVR design and procedural optimization. (Global Multicenter Registry on Transcatheter Tricuspid Valve Replacement [TRIPLACE]; NCT06033274)
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ABBREVIATIONS AND ACRONYMS

HF = heart failure
PVL = paravalvular leak
RA = right atrial/atrium
TR = tricuspid regurgitation
TTVR = transcatheter tricuspid valve replacement
TV = tricuspid valve

Tricuspid regurgitation (TR) is the most prevalent valvular heart disease, particularly among elderly and comorbid patients, and is independently associated with increased morbidity and mortality.[1-3] Because of the large unmet clinical need for treating symptomatic patients with severe TR, transcatheter tricuspid valve replacement (TTVR) has emerged commercially as a viable solution to fully restore tricuspid valve (TV) function, achieving near complete resolution of TR in the majority of cases.[4-6] Although TTVR can achieve mild or less residual TR in >95% of patients,[5] there are some patients with paravalvular leak (PVL), a complication that may attenuate the clinical and hemodynamic benefits of valve replacement.

PVL has been extensively studied in the context of transcatheter aortic and mitral valve replacement, in which its presence has been linked to adverse outcomes, including heart failure (HF) symptoms, hemolysis, and increased mortality.[7-9] In the tricuspid position, the implications of PVL remain less clearly defined, given the unique anatomical challenges of the tricuspid annulus, frequent annular dilation, and lower pressure right-sided circulation.

The TRIPLACE (Global Multicenter Registry on Transcatheter Tricuspid Valve Replacement) registry (NCT06033274) is the largest multicenter, real-world data set of patients undergoing orthotopic TTVR with dedicated devices. The aim of the present analysis was to: 1) investigate the incidence and severity of PVL following TTVR; 2) explore the clinical impact of PVL on early postprocedural outcomes; and 3) identify procedural and anatomical predictors of PVL.

METHODS

REGISTRY DESIGN. TRIPLACE (NCT06033274) is an investigator-initiated, retrospective, single-arm, open-label, multicenter registry designed to evaluate the safety and efficacy of orthotopic TTVR systems in a real-world setting.[10] This registry was instituted independent of external financial support and is strategically designed to compile comprehensive data from a global array of centers proficient in TTVR. Data contributions to date were made by 21 centers across the United States, Europe, and Canada (Supplemental Appendix). Data collection included baseline clinical characteristics, preprocedural echocardiographic and computed tomographic data, as well as detailed procedural and postprocedural metrics. These were reported by the participating centers using a structured reporting form specifically designed for the registry. Data collection was approved by the local Institutional Review Boards, and the study was performed in accordance with the Declaration of Helsinki.

STUDY POPULATION. As per predefined inclusion criteria, the registry included all consecutive patients undergoing isolated orthotopic TTVR for severe TR using 8 dedicated TV devices.[10] A total of 193 patients (49%) were treated with the commercially approved EVOQUE system (Edwards Lifesciences), whereas the remaining 51% underwent TTVR using investigational devices currently seeking regulatory approval (Supplemental Table 1). The decision to perform TTVR was made by the multidisciplinary heart team at each center, with treatment protocols aligning with the standard-of-care practices at each site. In the present analysis focused on PVL post-TTVR, the following exclusion criteria were applied: intraprocedural mortality, surgical conversion during the index TTVR procedure, and absence of data on PVL at discharge.

STUDY ENDPOINTS. The primary endpoint was the post-TTVR incidence of moderate or severe PVL assessed at predischarge echocardiography. Secondary endpoints were 1-year all-cause and cardiac mortality, 1-year HF hospitalization, and changes in NYHA functional class according to PVL severity after TTVR.

Cardiac mortality was defined as any death directly attributable to a primary cardiac cause, including myocardial infarction, progressive or acute decompensated HF, fatal arrhythmias, sudden cardiac death, cardiogenic shock, or complications related to structural or valvular heart disease or its treatment. Deaths clearly attributable to noncardiac causes (eg, malignancy, infection, pulmonary disease, trauma, bleeding unrelated to the cardiac procedure) were not considered cardiac mortality.

ECHOCARDIOGRAPHIC ASSESSMENT. Echocardiographic evaluations were performed by experienced echocardiographers at high-volume structural heart centers, each with extensive expertise in TR and TTVR imaging following contemporary guidelines.[11,12] All patients underwent baseline transthoracic and transesophageal echocardiography, followed by serial transthoracic echocardiography post-TTVR, supplemented by transesophageal echocardiography when clinically indicated or when image quality was suboptimal. Definitions pertaining to the etiology and severity of TR, in addition to safety and efficacy endpoints, were established in accordance with the guidelines set forth by the Tricuspid Valve Academic Research Consortium.[13] The classification of TV anatomy adhered to a widely recognized system based on leaflet configuration: types I, II, IIIA, IIIB, IIIC, and IV.[14] PVL was defined as the presence of regurgitant flow occurring between the outer frame of the TTVR prosthesis and the native annulus or surrounding cardiac tissue, rather than through the valve's orifice. PVL was identified using color Doppler echocardiography and graded according to established multiparametric criteria, incorporating jet size, flow convergence, vena contracta, and hepatic vein flow pattern. In cases with multiple jets, the overall PVL grade represented an integrated assessment that accounted for the cumulative regurgitant burden. Severity was categorized as none or trace, mild, moderate, or severe according to standard echocardiographic guidelines. PVL assessment was performed at discharge and 1 month and 1 year post-TTVR.

CARDIAC COMPUTED TOMOGRAPHIC ASSESSMENT. Cardiac computed tomographic images were acquired with electrocardiographically synchronized acquisition to allow multiphase assessment of tricuspid annular geometry. A biphasic iodinated contrast injection (≈80-120 mL total) with bolus tracking in the right atrium (RA) ensured homogeneous opacification of right-sided chambers and venous access routes. Images were reconstructed at 0.5- to 0.75-mm thickness across multiple cardiac phases, and annular, leaflet, and venous access measurements were obtained using dedicated cardiac postprocessing software. Tricuspid annular dimensions were obtained in the best systolic and diastolic phases, tracing the annular area at the

leaflet insertion point in long- and short-axis multiplanar reconstruction images. The degree of TTVR device oversizing was calculated in both systolic and diastolic phases using the following formula: [(TTVR device diameter/perimeter-derived TV annular diameter) − 1] × 100. TV eccentricity index was calculated using the following formula: 1 − (short-axis diameter/long-axis diameter).

STATISTICAL ANALYSES. Categorical variables are expressed as numbers and corresponding proportions and were compared using the chi-square test with continuity correction or the Fisher exact test as appropriate. Continuous variables are expressed as mean ± SD or median (Q1-Q3) and were compared using 1-way analysis of variance (parametric test) or the Kruskal-Wallis test (nonparametric test) according to their distribution. Normality of continuous variables was assessed using the Shapiro-Wilk test.

One-year all-cause mortality was estimated using Kaplan-Meier curves, and differences per PVL severity were evaluated using the log-rank test. A multivariable Cox regression analysis was performed to explore the impact of PVL severity on 1-year mortality. Covariates included in the Cox proportional hazards regression model were selected according to established prognostic relevance in patients with severe TR and their clinical plausibility as determinants of outcomes after TTVR. Specifically, we included TRI-SCORE, RA volume, renal function, and atrial fibrillation, all of which have been consistently shown to independently predict mortality in advanced TR populations.[15-21] Common demographic variables such as age and sex were not included as separate covariates, because their prognostic effects are already integrated within TRI-SCORE, and adding overlapping variables could risk model overfitting. However, an additional Cox regression model was performed as a sensitivity analysis including the following variables: age, sex, left ventricular ejection fraction, pulmonary artery systolic pressure, and baseline TR. The proportional hazards assumption was formally tested using Schoenfeld residuals, and no significant violations were detected. Multicollinearity was assessed using variance inflation factors, confirming the absence of concerning collinearity among covariates. The incidence of HF hospitalization was estimated using the cumulative incidence function (Gray's test) accounting for death as a competing risk. To further investigate the hemodynamic impact of PVL post-TTVR, the following sensitivity analyses were performed: 1) landmark analysis on 1-year survival after a predefined time period of 30 days post-TTVR; 2) 30-day landmark analysis excluding patients with device malposition or necessitating intervention for valve dysfunction or valve-related complication; 3) 1-year survival in moderate vs no or mild PVL (ie, severe PVL excluded) after excluding patients with device malposition or necessitating intervention for valve dysfunction or valve-related complication.

A Sankey diagram was used to display changes in PVL severity between hospital discharge and 1-month follow-up following TTVR. Logistic regression analysis was performed to explore the association between baseline variables and the occurrence of moderate or severe PVL post-TTVR. Variables entered into the multivariable model were selected on the basis of clinical relevance and prior evidence, with univariable associations used only as an initial screening step. This approach ensured the inclusion of clinically meaningful covariates regardless of their univariable significance while minimizing the risk for overfitting. Because of the small number of events and potential issues with perfect separation, Firth's penalized logistic regression was used for the multivariable model to reduce bias in the parameter estimates. A 2-sided P value <0.05 was considered to indicate statistical significance. Statistical analyses were performed using R version 4.5.1 (R Foundation for Statistical Computing).

RESULTS

BASELINE CHARACTERISTICS. To date, 421 TTVR recipients have been enrolled in TRIPLACE from 2016 through 2025. After applying exclusion criteria relevant to this analysis (6 procedural deaths, 5 surgical conversions, 16 patients with missing post-TTVR PVL data), 394 patients were included (Supplemental Figure 1). Baseline clinical characteristics and laboratory values are displayed in **Table 1** and Supplemental Table 2. Overall, mean age was 76.6 ± 8.8 years, and 65.2% were women. The median European System for Cardiac Operative Risk Evaluation II mortality risk and TRI-SCORE were 4.2% and 5.0, respectively. Nearly one-half of patients (43.5%) had been hospitalized for HF within the 12 months preceding TTVR, and most of them (78.2%) presented in NYHA functional class III or IV.

A total of 24 patients (6.1%) had moderate or greater PVL post-TTVR, with mild PVL being observed in 88 (22.3%) and no or trace PVL in the remaining 282 patients (71.6%) (**Central Illustration**). Among patients with moderate or greater PVL, 17 (71%) had moderate PVL, and 7 (29%) had severe PVL. Compared with no or trace or mild PVL, patients with

TABLE 1 Baseline Characteristics per Paravalvular Leak Severity

	Overall (N = 394)	None/Trace (n = 282)	Mild (n = 88)	Moderate/Severe (n = 24)	*P* Value
Age, y	76.6 ± 8.8	76.1 ± 9.1	77.6 ± 8.0	77.5 ± 6.7	0.33
Women	257 (65.2)	194 (68.8)	51 (58)	12 (50)	0.05
EuroSCORE II, %	4.2 (2.7, 6.9)	4.2 (2.6, 6.8)	4.1 (3.0, 6.5)	4.9 (3.7, 8.6)	0.14
TRI-SCORE	5.0 (4.0, 6.0)	5.0 (4.0, 6.0)	5.0 (4.0, 6.0)	7.0 (6.0, 7.5)	<0.001
NYHA functional class III/IV	308 (78.2)	218 (77.3)	71 (80.7)	19 (79.2)	0.79
HF hospitalization (≤12 mo)	171 (43.5)	120 (42.6)	41 (47.1)	10 (41.7)	0.72
6MWD, m	231.0 ± 115.0	230.9 ± 113.2	240.2 ± 120.0	178.2 ± 149.9	0.61
KCCQ score	54.8 ± 20.0	55.7 ± 19.2	50.9 ± 24.3	53.9 ± 18.0	0.66
Device at baseline					0.25
None	261 (66.4)	193 (68.7)	52 (59.1)	16 (66.7)	
PM/CRT-P	94 (23.9)	63 (22.4)	24 (27.3)	7 (29.2)	
CRT-D/ICD	23 (5.9)	12 (4.3)	10 (11.4)	1 (4.2)	
Leadless PM	11 (2.8)	9 (3.2)	2 (2.3)	0 (0.0)	
Other	4 (1.0)	4 (1.4)	0 (0.0)	0 (0.0)	
CIED lead across the TV	109 (27.7)	70 (24.8)	31 (35.2)	8 (33.3)	0.133
CIED lead contributing to TR	75 (19.2)	48 (17.2)	24 (27.3)	3 (13.0)	0.09
Prior cardiac surgery	164 (41.6)	117 (41.5)	36 (40.9)	11 (45.8)	0.91
Prior TV surgery	19 (4.8)	14 (5.0)	3 (3.4)	2 (8.3)	0.60
Prior TAVR	24 (6.1)	18 (6.4)	4 (4.5)	2 (8.3)	0.66
Comorbidities					
Hypertension	274 (69.5)	194 (68.8)	62 (70.5)	18 (75.0)	0.80
Diabetes mellitus	91 (23.1)	63 (22.3)	20 (22.7)	8 (33.3)	0.47
Atrial fibrillation	342 (86.8)	218 (83.8)	83 (97.6)	21 (97.5)	0.004
CKD	188 (47.8)	128 (45.6)	48 (54.5)	12 (50.0)	0.33
Prior dialysis	14 (3.6)	8 (2.8)	3 (3.4)	3 (12.5)	0.07
COPD	56 (14.2)	41 (14.5)	11 (12.5)	4 (16.7)	0.82
Peripheral vascular disease	45 (11.5)	31 (11.0)	11 (12.5)	3 (12.5)	0.82
Peripheral edema	297 (75.4))	209 (74.1)	69 (78.4)	19 (79.2)	0.65
Ascites	76 (19.3)	50 (17.7)	18 (20.5)	8 (33.3)	0.17
Laboratory values					
Albumin, g/dL	3.8 ± 0.6	3.8 ± 0.6	3.8 ± 0.7	3.6 ± 0.6	0.62
Creatinine, mg/dL	1.2 (0.9-1.6)	1.1 (0.9-1.5)	1.3 (1.0-1.7)	1.7 (1.2-2.1)	<0.001
eGFR, mL/min/1.73 m^2	50.5 (37.0-68.0)	54.0 (39.0-69.0)	47.0 (36.0-61.2)	35.0 (27.5-47.5)	0.001
ALT, U/L	19.0 (14.0-26.0)	19.0 (14.0-26.0)	19.0 (14.0-25.0)	17.0 (13.5-22.0)	0.38
AST, U/L	27.0 (21.0-34.0)	27.0 (20.0-33.0)	28.0 (21.5-37.0)	27.0 (21.0-30.0)	0.51
Total bilirubin, mg/dL	0.9 (0.6-1.3)	0.8 (0.6-1.3)	0.9 (0.7-1.3)	0.8 (0.6-1.3)	0.27
INR	1.3 (1.1-1.6)	1.3 (1.1-1.6)	1.3 (1.1-1.8)	1.3 (1.2-1.4)	0.70
NT-proBNP, pg/mL	1,641 (949-3,117)	1,466 (829-2,762)	2,021 (1,128-3,509)	2,019 (1,215-4,918)	0.06
Hemoglobin, g/dL	12.1 ± 2.0	12.2 ± 1.9	11.9 ± 2.1	11.5 ± 2.1	0.13

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moderate or greater PVL had significantly higher TRI-SCOREs (7 vs 5 or 5; $P < 0.001$), lower estimated glomerular filtration rates (35 mL/min/1.73 m^2 vs 54 or 47 mL/min/1.73 m^2; $P = 0.001$). Other clinical comorbidities were similar among the study groups, except for atrial fibrillation, which was less common in patients with no or trace PVL ($P = 0.004$). Regarding baseline pharmacologic management, 91.1% of the cohort were on loop diuretic agents, while beta-blockers, aldosterone antagonists, and sodium-glucose cotransporter 2 inhibitors were part of the treatment regimen for 72.4%, 49.7%, and 30.3% of patients, respectively. Anticoagulation was common at 86% (59.9% on direct oral anticoagulant agents).

The baseline severity of TR was graded as severe in 30.7%, massive in 29.2%, and torrential in 40.1% of patients. The etiology of TR was classified as primary in 11.1% of cases, ventricular secondary in 32.7%, and atrial secondary in 42.5%. Although no differences were observed in baseline TR severity, patients with moderate or greater PVL were more likely to have atrial secondary TR (60%; $P = 0.03$). There was a nonsignificant trend for more PVL in the presence of cardiac implantable electronic device leads across the TV during TTVR: moderate or greater, 33.3%; mild, 35.2%; and no or trace PVL, 24.8% ($P = 0.13$). Patients with moderate or greater PVL were more likely to have a 4-leaflet TV, larger right-sided dimensions, and higher pulmonary artery systolic pressures.

TABLE 1 Continued

	Overall (N = 394)	None/Trace (n = 282)	Mild (n = 88)	Moderate/Severe (n = 24)	*P* Value
Medications					
Loop diuretics	359 (91.1)	258 (91.5)	81 (92.0)	20 (83.3)	0.38
Anticoagulation					0.79
None	55 (14.0)	43 (15.2)	10 (11.4)	2 (8.3)	
DOACs	236 (59.9)	167 (59.2)	55 (62.5)	14 (58.3)	
Warfarin	103 (26.1)	72 (25.2)	23 (26.1)	8 (33.3)	
Beta-blockers	284 (72.4)	203 (72.2)	68 (78.2)	13 (54.2)	0.07
ACEIs/ARBs/ARNIs	169 (43.1)	119 (42.2)	38 (44.2)	12 (50.0)	0.74
Aldosterone antagonists	193 (49.7)	129 (46.2)	52 (61.2)	12 (50.0)	0.06
SGLT2 inhibitors	118 (30.3)	82 (29.2)	28 (32.9)	8 (33.3)	0.76
Echocardiography					
TR severity					0.97
Severe	121 (30.7)	88 (31.2)	27 (30.7)	6 (25.0)	
Massive	115 (29.2)	82 (29.1)	26 (29.5)	7 (29.2)	
Torrential	158 (40.1)	112 (39.7)	35 (39.8)	11 (45.8)	
TR etiology					0.03
Primary	43 (11.1)	33 (11.9)	6 (6.9)	4 (17.4)	
Ventricular secondary	127 (32.7)	95 (34.2)	27 (31.0)	5 (21.7)	
Atrial secondary	165 (42.5)	118 (42.4)	33 (37.9)	14 (60.9)	
CIED	28 (7.2)	17 (6.1)	11 (12.6)	0 (0.0)	
Mixed	25 (6.4)	15 (5.4)	10 (11.5)	0 (0.0)	
TV morphology type					0.06
I	216 (58.5)	153 (57.7)	51 (62.2)	12 (54.5)	
II	10 (2.7)	7 (2.6)	3 (3.7)	0 (0.0)	
IIIa	22 (6.0)	17 (6.4)	5 (6.1)	0 (0.0)	
IIIb	89 (24.1)	70 (26.4)	15 (18.3)	4 (18.2)	
IIIc	18 (4.9)	10 (3.8)	6 (7.3)	2 (9.1)	
IV	14 (3.8)	8 (3.0)	2 (2.4)	4 (18.2)	
TV annular diameter, mm	45.0 (41.0-50.0)	44.0 (41.0-49.0)	47.0 (43.0-51.0)	48.0 (42.0-50.8)	0.01
RV basal diameter, mm	50.0 (44.5-56.0)	49.0 (44.0-55.0)	54.0 (46.0-58.0)	55.0 (50.0-63.0)	0.002
RA volume, mL	125.0 (86.0-161.0)	120.0 (79.0-153.0)	143.0 (110.0-182.0)	129 (121.5-197.5)	0.002
TAPSE, mm	17.0 (14.0-20.0)	17.0 (14.0-20.0)	17.0 (14.0-20.0)	18.0 (16.0-22.0)	0.28
PASP, mm Hg	37.0 (29.0-45.0)	37.0 (28.0-45.0)	35.0 (29.5-43.5)	43.0 (36.5-54.5)	0.03
LVEF, %	55.7 ± 8.7	56.3 ± 8.4	53.9 ± 9.6	55.8 ± 9.3	0.09
Computed tomography					
TV perimeter, diastole, mm	153.0 ± 16.9	151.1 ± 15.6	158.5 ± 20.2	156.4 ± 15.4	0.002
TV perimeter, systole, mm	145.7 ± 15.1	143.4 ± 14.0	153.2 ± 16.5	148.2 ± 15.0	<0.001
TV area, diastole, mm^2	1,725.2 ± 525.2	1,649.9 ± 521.4	1,943.0 ± 462.0	1,866.3 ± 557.8	<0.001
TV area, systole, mm^2	1,612.8 ± 898.8	1,559.6 ± 1,009.7	1,774.1 ± 459.2	1,691.7 ± 521.4	0.25
TV maximal diameter, mm	51.0 ± 6.2	50.2 ± 5.7	53.3 ± 6.8	52.4 ± 6.8	<0.001
TV minimal diameter, mm	44.1 ± 5.3	43.2 ± 5.1	46.4 ± 5.7	45.9 ± 4.6	<0.001
TV eccentricity index	0.13 ± 0.08	0.13 ± 0.09	0.13 ± 0.08	0.12 ± 0.07	0.57

Values are mean ± SD, n (%), or median (Q1-Q3).

6MWD = 6-minute walk test; ACEI = angiotensin-converting enzyme inhibitor; ALT = alanine transaminase; ARB = angiotensin II receptor blocker; ARNI = angiotensin receptor neprilysin inhibitor; AST = aspartate transaminase; CIED = cardiac implantable electronic device; CKD = chronic kidney disease; COPD = chronic obstructive pulmonary disease; CRT-D = cardiac resynchronization therapy defibrillator; CRT-P = cardiac resynchronization therapy pacemaker; DOAC = direct oral anticoagulant agent; eGFR = estimated glomerular filtration rate; EuroSCORE II = European System for Cardiac Operative Risk Evaluation II; HF = heart failure; ICD = implantable cardioverter-defibrillator; INR = international normalized ratio; KCCQ = Kansas City Cardiomyopathy Questionnaire; LVEF = left ventricular ejection fraction; NT-proBNP = N-terminal pro-B-type natriuretic peptide; PASP = pulmonary artery systolic pressure; PM = pacemaker; RA = right atrial; RV = right ventricular; SGLT2 = sodium-glucose cotransporter 2; TAPSE = tricuspid annular plane systolic excursion; TAVR = transcatheter aortic valve replacement; TR = tricuspid regurgitation; TV = tricuspid valve.

Preprocedural computed tomographic planning revealed larger TV annuli in patients with PVL post-TTVR, with median TV perimeters in diastole of 156.4 ± 15.4 mm vs 158.5 ± 20.2 mm vs 151.1 ± 15.6 mm (P = 0.002) for moderate or greater vs mild vs no or trace PVL, respectively. No differences were observed according to TV eccentricity. In line with larger TV annuli, patients demonstrating mild or greater PVL received larger prosthetic sizes for TTVR (Table 2). There was no significant difference in oversizing between PVL groups (P = 0.10). Valve malposition after TTVR was significantly associated with moderate or greater PVL (moderate or greater PVL, 20.8%; mild PVL, 1.1%; no or trace PVL, 1.4%; P < 0.001). Two patients (8.3%) with moderate or greater PVL required intervention for valve-related dysfunction, migration, thrombosis, or other complication, 2 (2.3%) with mild PVL, and 3 (1.1%, P = 0.04) with no or trace PVL.

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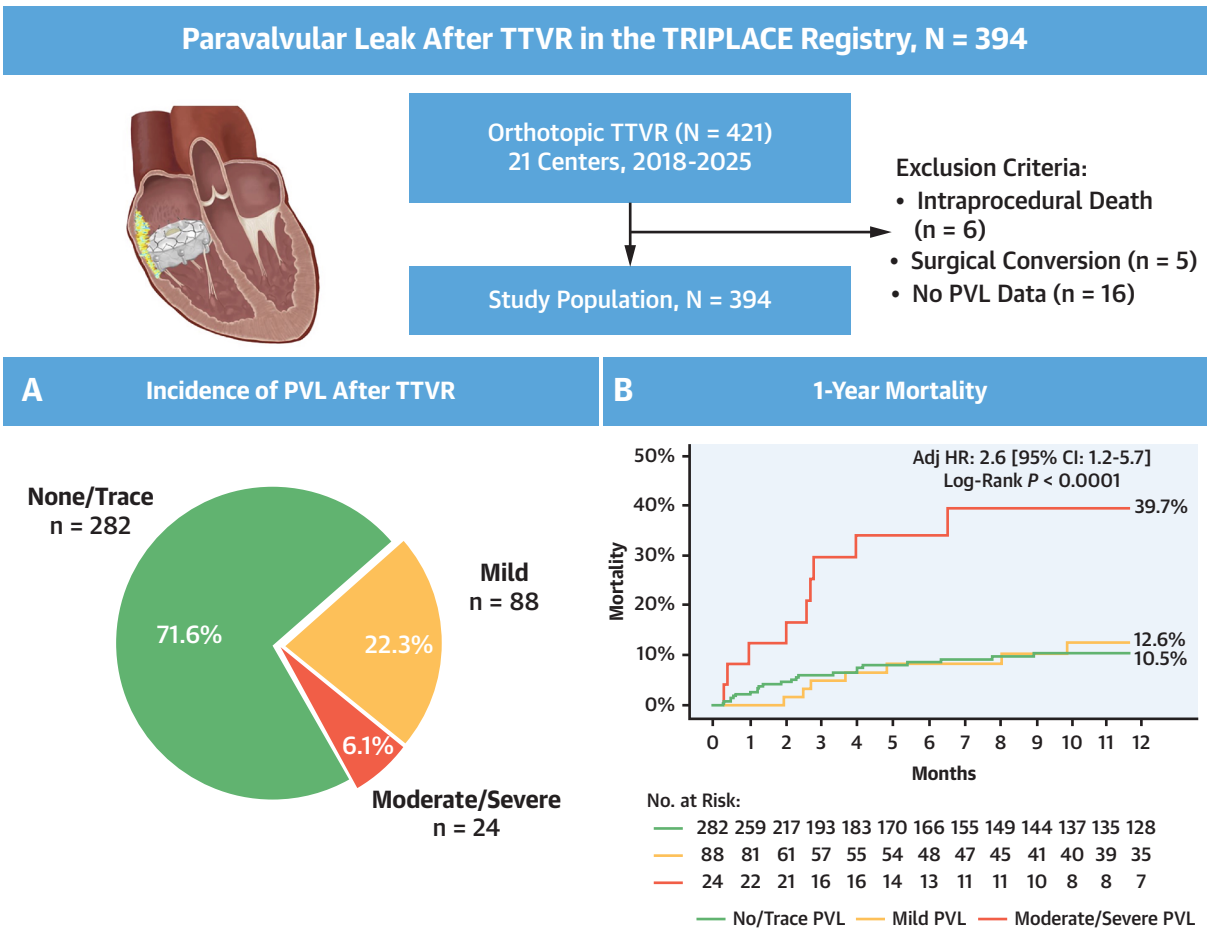
CENTRAL ILLUSTRATION PVL After TTVR



- Moderate or severe PVL occurred in 6.1% of patients after TTVR.
- Moderate/severe PVL was associated with increased 1-year mortality (39.7%, Adj HR: 2.6 [95% CI: 1.2-5.7]).
- Larger RA volume, type IV TV morphology, and valve malposition were predictors of ≥ moderate PVL.

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(A) Post-transcatheter tricuspid valve replacement (TTVR) incidence of paravalvular leak (PVL) graded as none or trace, mild, and moderate or severe. (B) One-year all-cause mortality after TTVR based on postprocedural PVL severity. Multivariable Cox regression analysis was performed for moderate or severe PVL vs no, trace, or mild PVL and adjusted for TRI-SCORE, atrial fibrillation, right atrial (RA) volume, and renal function. Adj HR = adjusted HR; TRIPLACE = Global Multicenter Registry on Transcatheter Tricuspid Valve Replacement; TV = tricuspid valve.

EVOLUTION OF PVL SEVERITY POST-TTVR. Paired discharge, 1-month, and 1-year echocardiograms were available for 159 patients. The evolution of PVL severity post-TTVR is illustrated by the Sankey diagram (Figure 1). At 1 year post-TTVR, a larger group of patients were diagnosed with moderate or greater PVL. A total of 7 patients showed reductions in PVL from moderate or greater to mild (n = 4) and none or trace (n = 3) at 1 month and 1 year. Of these patients, 4 were treated with intensification of diuretic therapy, and the other 3 showed favorable right ventricular reverse remodeling. Conversely, a group of those starting with mild or no or trace PVL (n = 7), or having mild or no or trace PVL at 1 month (n = 5) progressed to moderate or severe PVL at follow-up.

CLINICAL OUTCOMES. The presence of moderate or greater PVL was associated with increased 1-year all-cause mortality post-TTVR: moderate or greater,

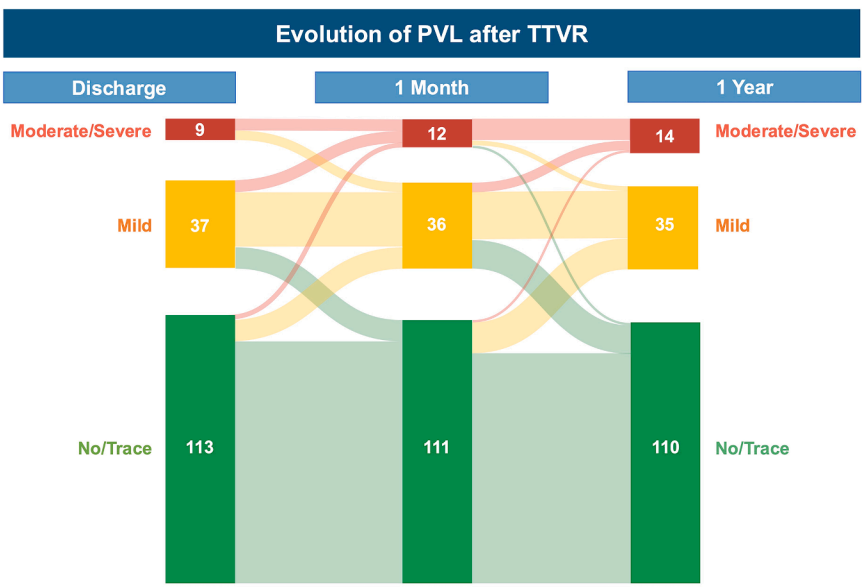
TABLE 2 Procedural Characteristics per Paravalvular Leak Severity

	Overall (N = 394)	None/Trace (n = 282)	Mild (n = 88)	Moderate/ Severe (n = 24)	*P* Value
Valve size					<0.001
≤45 mm	57 (14.5)	44 (15.6)	10 (11.4)	3 (12.5)	
45-55 mm	205 (52.0)	163 (57.8)	33 (37.5)	9 (37.5)	
≥55 mm	132 (33.5)	75 (26.6)	45 (51.1)	12 (50.0)	
TTVR device oversizing, diastole, %	8.9 ± 10.0	8.6 ± 9.9	8.6 ± 7.6	14.1 ± 15.1	0.10
TTVR device oversizing, systole, %	12.7 ± 10.2	12.5 ± 10.0	12.0 ± 8.9	16.5 ± 16.0	0.35
Device malposition	10 (2.5)	4 (1.4)	1 (1.1)	5 (20.8)	<0.001
Device migration	2 (0.5)	2 (0.7)	0 (0.0)	0 (0.0)	1
Device embolization	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	NA[a]
Valve-related dysfunction, migration, thrombosis, or other complication requiring intervention	7 (1.8)	3 (1.1)	2 (2.3)	2 (8.3)	0.04

Values are n (%) or mean ± SD. [a]Due to inaccuracy in zero event rate comparisons.
NA = not applicable; TTVR = transcatheter tricuspid valve replacement.

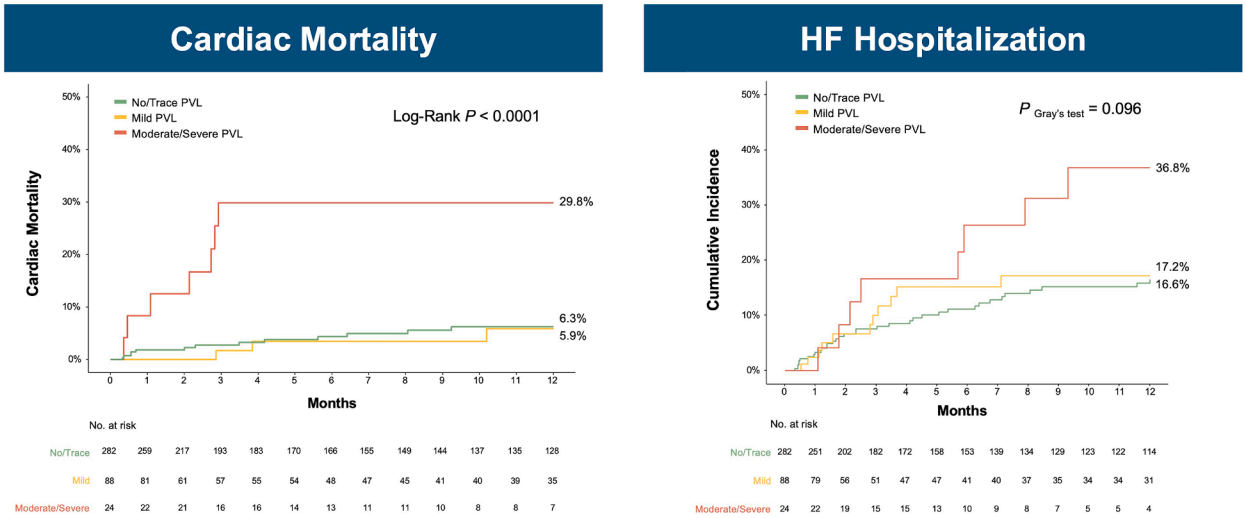
39.7%; mild, 12.6%; and none or trace, 10.5% (log-rank $P < 0.0001$) (**Central Illustration**). On multivariable Cox regression analysis, there was an independent association of moderate or greater PVL with 1-year all-cause mortality (adjusted HR: 2.6; 95% CI: 1.2-5.7; $P = 0.02$) (Supplemental Table 3). The landmark analysis censoring the first 30 days post-TTVR confirmed the overall results of increased 1-year all-cause mortality for patients with moderate or greater PVL (log-rank $P = 0.0007$) (Supplemental Figure 2). An additional landmark analysis excluding patients with device malposition or requiring intervention for valve dysfunction or valve-related complications confirmed the overall results, with increased 1-year all-cause mortality for patients with moderate or greater PVL (log-rank $P = 0.0031$) (Supplemental Figure 3). When analyzing the impact of moderate PVL, excluding patients with

FIGURE 1 Evolution of PVL After TTVR



Sankey diagram displaying paired changes in paravalvular leak (PVL) severity among post-transcatheter tricuspid valve replacement (TTVR), 1-month, and 1-year echocardiographic assessments (n = 159 patients with paired echocardiographic data).

FIGURE 2 Cardiac Mortality and HF Hospitalization at 1 Year After Transcatheter Tricuspid Valve Replacement



HF = heart failure; PVL = paravalvular leak.

severe PVL, device malposition, or requiring intervention for valve dysfunction or valve-related complication, there was an increased all-cause mortality compared with those with no or mild PVL (log-rank $P = 0.011$) (Supplemental Figure 4). The 2 patients in the moderate or greater PVL group requiring reintervention underwent valve-in-valve procedures with 29-mm SAPIEN 3 Ultra valves (Edwards Lifesciences) that were successfully deployed to stabilize the TTVR prosthesis (44-mm EVOQUE, 48-mm EVOQUE), with no impact on PVL severity, which remained in the moderate or greater grade.

At 1 year post-TTVR, cardiac mortality was higher in patients with moderate or greater PVL (29.8%) compared with those with mild (5.9%) and no or trace (6.3%) PVL ($P < 0.0001$) (**Figure 2**). The cumulative incidence of HF hospitalization, accounting for death as a competing risk, was numerically higher in patients with more severe PVL post-TTVR: moderate or greater, 36.8%; mild, 17.2%; none or trace, 16.6% ($P = 0.096$, Gray's test).

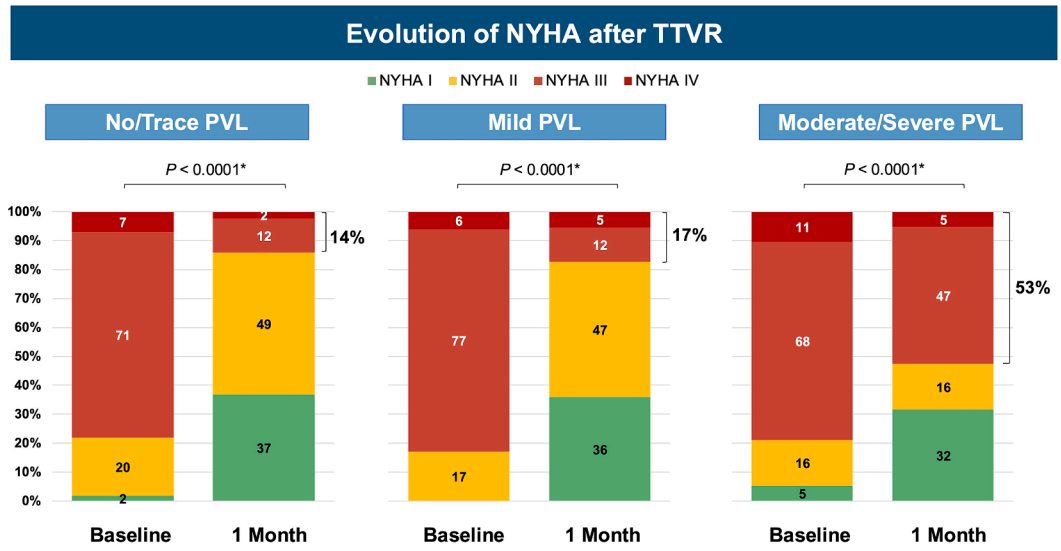
Changes in NYHA functional class between baseline and 1 month post-TTVR are illustrated in **Figure 3**. Although all patients undergoing TTVR experienced significant improvements in NYHA functional class ($P < 0.0001$ for all), less clinical improvement was observed in those with moderate or greater PVL. At 1 month post-TTVR, almost one-half of patients with moderate or greater PVL (53%) were in NYHA functional class III or IV. Conversely, only a minority of patients were in NYHA functional class III or IV in the mild (17%) and no or trace (14%) PVL groups.

PREDICTORS OF MODERATE OR GREATER PVL POST-TTVR. The association between baseline variables and moderate or greater PVL post-TTVR is presented in Supplemental Table 4. Variables associated with the outcome of interest and deemed to be clinically relevant were included in the multivariable analysis. The multivariable Firth bias-reduced logistic regression model revealed an independent association of baseline RA volume (adjusted OR: 1.47 per 50-mL increase; 95% CI: 1.09-1.99; $P = 0.017$), device malposition (adjusted OR: 17.67; 95% CI: 4.41-70.77; $P = 0.0002$), and type IV TV morphology (adjusted OR: 6.86; 95% CI: 1.81-25.95; $P = 0.012$) with moderate or greater PVL post-TTVR (**Table 3**, Supplemental Figure 5).

DISCUSSION

TRIPLACE, the largest TTVR cohort to date, offers critical insights into real-world outcomes associated with orthotopic TTVR. The present TRIPLACE analysis focused on the incidence, outcomes, and predictors of moderate or greater PVL post-TTVR. Key findings include the following: 1) moderate or severe PVL occurred in 6.1% of patients post-TTVR; 2) moderate or greater PVL was associated with

FIGURE 3 Evolution of NYHA Functional Class After Transcatheter Tricuspid Valve Replacement



Paired changes in NYHA functional class between post-TTVR and 1-month assessments stratified by PVL severity (patients with paired NYHA data: n = 243 with no or trace PVL, n = 77 with mild PVL, n = 19 with moderate or severe PVL). *P values for NYHA functional class III or IV vs NYHA functional class I or II. Abbreviations as in Figures 1 and 2.

increased 1-year mortality (39.7%) and worse functional status (53% in NYHA functional class III or IV); and 3) a larger RA volume, device malposition, and type IV TV morphology conferred a higher risk for developing moderate or greater PVL post-TTVR.

Moderate or severe PVL was observed in 6.1% of patients post-TTVR; an additional 22.3% had mild PVL, and 71.6% had no or trace PVL. This incidence is comparable with rates reported for PVL after transcatheter mitral valve replacement, for which moderate or greater PVL occurs in about 5% of cases and is associated with adverse clinical outcomes.[22] The tricuspid annulus, being large, elliptical, saddle shaped, and dynamic, might lead to incomplete apposition between the prosthesis and the native anatomy. However, grading PVL in the tricuspid position remains particularly challenging. PVL following TTVR is frequently characterized by low-pressure, eccentric jets that can be difficult to visualize or quantify with standard transthoracic or transesophageal echocardiography. Reverberation artifacts from the valve frame, limited acoustic windows, and the inherently low-pressure nature of right-sided flow further complicate assessment. As a result, current classification schemes, largely adapted from aortic and mitral contexts, may underestimate the prevalence or significance of PVL in the tricuspid space.

The clinical consequences of moderate or greater PVL in patients in the present analysis were profound. Patients with moderate or greater PVL exhibited a 1-year all-cause mortality rate of 39.7%, markedly higher than those with mild (12.6%) and no or trace (10.5%) PVL. Significant differences in baseline characteristics, including higher TRI-SCOREs, lower estimated glomerular filtration rates, and a greater prevalence of atrial fibrillation, might have contributed to the poor outcomes associated with moderate or greater PVL. However, despite multivariable adjustment including those variables, moderate or greater PVL was still independently associated with a 2.6-fold higher risk for mortality at

TABLE 3 Predictors of Moderate or Severe Paravalvular Leak After Orthotopic Transcatheter Tricuspid Valve Replacement

	Adjusted OR (95% CI)	*P* Value
RV basal diameter, per 5-mm increase	1.12 (0.87-1.43)	0.393
RA volume, per 50-mL increase	1.47 (1.09-1.99)	0.017
TV morphology (type IV)	6.86 (1.81-25.95)	0.012
Device malposition	17.67 (4.41-70.77)	0.0002

Abbreviations as in Tables 1 and 2.

1 year post-TTVR. This striking mortality difference underscores the prognostic influence of PVL post-TTVR. The observed higher incidence of 1-year cardiac mortality suggests that significant PVL might be a major driver, rather than a confounding factor, of adverse outcomes after TTVR. Interestingly, higher 1-year mortality in patients moderate or greater PVL was still observed after excluding those cases in which PVL might have been the consequence of a failed TTVR procedure (mortality within 30 days, device malposition, and reintervention because of valve dysfunction or other valve-related complication). Although residual PVL in left-sided valve procedures is well recognized as a predictor of adverse events, its impact in the tricuspid position is poorly understood.[7,22] Although the right-sided chambers of the heart have lower pressures than the left-sided circulation, the clinical impact of significant PVL might be explained by persistent systemic congestion, hepatic and renal dysfunction, and progressive right HF.

Beyond mortality, clinical improvement was significantly compromised in patients with moderate or greater PVL. Although the overall TRIPLACE cohort experienced substantial gains in symptom relief with better functional class, those with moderate or greater PVL were more likely to remain in NYHA functional class III or IV at short-term follow-up. This suggests that residual PVL may limit the hemodynamic improvements typically observed after TTVR, perpetuating RA volume overload and venous congestion. In contrast, residual TR following tricuspid transcatheter edge-to-edge repair is not uncommon, yet the clinical benefits of even a modest 1- or 2-grade TR reduction appear significant.[23-25] A plausible mechanism for these separate observations could be related to the annular sparing effects of tricuspid transcatheter edge-to-edge repair, whereas many orthotopic TTVR devices harbor some degree of tricuspid annular interaction, compromising annular function. The annular modifying properties of various orthotopic TTVR devices may ultimately prove pertinent, given that tricuspid annular function contributes up to 80% of right ventricular stroke volume.[26] Therefore, our findings reinforce the concept that near complete elimination of regurgitation, rather than partial improvement, should remain the goal of TTVR.

The present analysis identified a larger RA volume, device malposition, and a type IV TV morphology as independent predictors of moderate or greater PVL post-TTVR. Greater RA volume likely reflects chronic volume overload and annular remodeling, which could be associated with suboptimal prosthesis-annulus apposition. The chronicity of underlying atrial fibrillation may also be a driving factor for this. RA enlargement may serve as an integrative marker of atrial secondary TR and atrial fibrillation, both more common in patients with moderate or greater PVL compared with no or trace PVL. RA remodeling is also independently associated with adverse outcomes in medically treated patients with severe, symptomatic TR.[20,21] Tomaselli et al[20] found that reduced RA strain (<13%) and increased RA volume index (>48 mL/m^2) were associated with lower survival rates. In fact, excess mortality rose exponentially with RA volume index >51.3 mL/m^2. RA remodeling thus deserves closer attention not only for risk stratification but for possible device choice. The presence of RA enlargement may reflect a degree of device-anatomy mismatch, particularly with TTVR systems that rely on atrial flanges or skirts to achieve sealing at the atrial side of the annulus. In patients with markedly enlarged RAs, atrial walls are often dilated, compliant, and displaced, which can reduce mechanical apposition between the atrial valve flanges and surrounding tissue. This loss of contact may allow paravalvular jets to persist, especially near commissural or noncircular regions of the annulus and may limit the device's ability to fully eliminate regurgitant flow. As a result, devices that depend primarily on atrial anchoring or sealing may be less effective at PVL mitigation in anatomies with excessive atrial dilation, and future device designs may need to incorporate additional ventricular anchoring mechanisms or circumferential sealing features to overcome this limitation.

In addition to RA remodeling, device malposition emerged as the strongest predictor associated with PVL. Malposition can impair circumferential sealing, create asymmetrical anchoring, or leave portions of the annulus uncovered, thereby facilitating PVL. This finding underscores the critical importance of meticulous preprocedural imaging, device sizing, learning curve, and deployment techniques to ensure optimal alignment and annular coverage. Finally, type IV TV morphology, characterized by the presence of multiple smaller leaflets, was also independently associated with PVL. In this anatomical setting, prosthesis anchoring and sealing are more challenging: leaflet-based devices may fail to achieve uniform grasping across all leaflets, while valves relying on radial force may struggle to ensure complete circumferential sealing. Both mechanisms

increase the likelihood of paravalvular jets in patients with complex leaflet anatomy. Together, these findings highlight the interplay among atrial remodeling, device positioning, and native valve morphology in determining the risk for PVL after TTVR.

The present observations support the need for refinements in patient selection, implantation techniques, including intraprocedural imaging guidance, and device design to mitigate PVL risk at both the index procedure as well as during the device's and patient's lifetimes. Future orthotopic TTVR systems may benefit from enhanced conformability and sealing strategies tailored to patients with marked RA dilation or complex TV anatomy. Additionally, standardized imaging protocols incorporating RA volume and TV morphology in the preprocedural work-up could improve procedural planning and help anticipate challenging cases. As transcatheter TR therapies continue to evolve, the present analysis highlights for the first time that diagnosing and addressing residual PVL following orthotopic TTVR appears critical in optimizing clinical outcomes. Future research should focus on elucidating the mechanisms driving the increased mortality in patients with significant PVL post-TTVR. Furthermore, evaluating the impact of PVL closure on mortality and functional recovery is crucial to determine if targeted interventions could substantially improve patient outcomes.

STUDY LIMITATIONS. TRIPLACE is susceptible to selection bias from 21 high-volume orthotopic TTVR centers, which limits the generalizability of these results to centers with less experience in orthotopic TTVR or from different healthcare systems. Unmeasured confounding factors (ie, operator experience or device availability) may have affected procedural results and patient selection. There was limited 1-year echocardiographic follow-up, primarily reflecting the real-world nature of the registry, in which follow-up imaging is influenced by site practices, patient preferences, and logistical factors such as treatment at high-volume structural heart centers located far from patients' primary residences. There was no independent core laboratory analysis of procedural results and echocardiographic data or independent adjudication of clinical events. Grading PVL severity in orthotopic TTVR prostheses is challenging because of the anatomical variability of the tricuspid annulus (eg, noncircular TV, large and compliant RA and right ventricle), the presence of multiple or eccentric jets with low velocity, imaging limitations inherent to right-sided structures (eg, shadowing from the prosthesis frame), and the lack of validated grading criteria specific to tricuspid prostheses. Whether the use of multimodality imaging might improve the accuracy and reproducibility of PVL grading remains to be investigated. The relatively low incidence of clinical events within the study curtails the statistical power of the analyses. Nonetheless, given the current nascent stage of orthotopic TTVR, the present findings are crucial, as they represent the most extensive cohort evaluated to date, providing novel insights into the procedural outcomes and clinical implications.

CONCLUSIONS

In TRIPLACE, 6.1% of patients developed moderate or greater PVL following orthotopic TTVR. The presence of moderate or greater PVL was associated with increased 1-year mortality (39.7%) and worse functional status (53% in NYHA functional class III or IV). A larger RA volume, device malposition, and type IV valve morphology conferred a high risk for developing moderate or greater PVL post-TTVR. Ongoing innovations in orthotopic TTVR device design, patient selection, and procedural optimization will be essential to reduce the risk for postprocedural PVL to optimize clinical outcomes.

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Dr Puri is a consultant, speaker, and proctor for Medtronic and Abbott Laboratories; is a consultant for Centerline Biomedical, Philips, Products & Features, Shockwave Medical, VDyne, VahatiCor, Advanced Nanotherapies, NuevoSono, TherOx, GE Healthcare, Anteris, T45 Labs, Pi-Cardia, AngioWave, BioVentrix, Protembis, HRT, and Nyra Medical; and has equity interest in Centerline Biomedical, T45 Labs, VahatiCor and NuevoSono. Dr Coisne is a proctor for Abbott Cardiovascular; and has received speaker fees from Abbott Cardiovascular, Bristol Myers Squibb, Edwards Lifesciences, GE Healthcare, Merck Sharpe & Dohme, and Pfizer. Dr Stolz has received speaker honoraria from Edwards Lifesciences. Dr Ludwig has received research grants from the German Heart Foundation and 4C Medical; has received travel compensation from Shockwave Medical and Edwards Lifesciences; has received speaker honoraria from Abbott Laboratories; and has received advisory fees from New Valve Technology and Bayer. Dr Ho has served as a consultant for Medtronic, Abbott Laboratories, Edwards Lifesciences, Philips, GE, Biosense Webster, Tioga, Nyra, Meocor, Neochord, Half Moon, and Valgen Medtech. Dr Dumonteil has received consultancy and proctoring fees from Abbott Laboratories, Boston Scientific, Edwards Lifesciences, and Medtronic. Dr Byrne has received proctor fees and honoraria from Medtronic and Abbott Laboratories; and has received speaking honoraria from Abiomed/Johnson & Johnson. Dr Hausleiter has received research support and speaker honoraria from Edwards Lifesciences. Dr Maisano has received grant and/or research

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PERSPECTIVES

WHAT IS KNOWN? Limited data are available regarding the incidence, outcomes, and predictors of PVL following orthotopic TTVR.

WHAT IS NEW? In TRIPLACE, 6.1% of patients developed moderate or greater PVL after TTVR. The presence of moderate or greater PVL was associated with increased 1-year mortality (39.7%) and worse functional status (53% in NYHA functional class III or IV). A larger RA volume, device malposition, and type IV valve morphology conferred a high risk for developing moderate or greater PVL post-TTVR.

WHAT IS NEXT? Ongoing innovations in TTVR device design will be essential to reduce the risk of PVL and optimize clinical outcomes following orthotopic TTVR.

REFERENCES

1. Scotti A, Sturla M, Echarte-Morales J, et al. Burden of valvular heart diseases in a racially and ethnically diverse population: the Bronx-Valve Registry. *J Am Heart Assoc*. 2025;14(2):e035378. https://doi.org/10.1161/JAHA.124.035378

2. Topilsky Y, Maltais S, Medina Inojosa J, et al. Burden of tricuspid regurgitation in patients diagnosed in the community setting. *JACC Cardiovasc Imaging*. 2019;12(3):433–442. https://doi.org/10.1016/j.jcmg.2018.06.014

3. Chorin E, Rozenbaum Z, Topilsky Y, et al. Tricuspid regurgitation and long-term clinical outcomes. *Eur Heart J Cardiovasc Imaging*. 2020;21(2):157–165. https://doi.org/10.1093/ehjci/jez216

4. Scotti A, Sturla M, Granada JF, et al. Outcomes of isolated tricuspid valve replacement: a systematic review and meta-analysis of 5,316 patients from 35 studies. *EuroIntervention*. 2022;18(10):840–851. https://doi.org/10.4244/EIJ-D-22-00442

5. Hahn RT, Makkar R, Thourani VH, et al. Transcatheter valve replacement in severe tricuspid regurgitation. *N Engl J Med*. 2025;392(2):115–126. https://doi.org/10.1056/NEJMoa2401918

6. Stolz L, Cheung A, Boone R, et al. Transjugular transcatheter tricuspid valve replacement: early compassionate use outcomes. *JACC Cardiovasc Interv*. 2024;17(16):1936–1945. https://doi.org/10.1016/j.jcin.2024.06.014

7. Kodali S, Hahn R, Williams M, et al. Impact of paravalvular leak following transcatheter aortic valve replacement on one-year mortality: analysis of the combined PARTNER cohorts. *Eur Heart J*. 2013;34(suppl_1):2584. https://doi.org/10.1093/eurheartj/eht309.2584

8. Zito A, Buono A, Scotti A, et al. Incidence, predictors, and outcomes of paravalvular regurgitation after TAVR in Sievers type 1 bicuspid aortic valves. *JACC Cardiovasc Interv*. 2024;17(14):1652–1663. https://doi.org/10.1016/j.jcin.2024.05.002

9. Muller DWM, Sorajja P, Duncan A, et al. 2-Year outcomes of transcatheter mitral valve replacement in patients with severe symptomatic mitral regurgitation. *J Am Coll Cardiol*. 2021;78(19):1847–1859. https://doi.org/10.1016/j.jacc.2021.08.060

10. Scotti A, Puri R, Sturla M, et al. Incidence, predictors, and management of conduction disturbances after transcatheter tricuspid valve replacement: the TRIPLACE registry. *JACC: Cardiovasc Interv*. 2025;18(14):1789–1799. https://doi.org/10.1016/j.jcin.2025.05.029

11. Lang RM, Badano LP, Mor-Avi V, et al. Recommendations for cardiac chamber quantification by echocardiography in adults: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. *Eur Heart J Cardiovasc Imaging*. 2015;16(3):233–271. https://doi.org/10.1093/ehjci/jev014

12. Zoghbi WA, Adams D, Bonow RO, et al. Recommendations for noninvasive evaluation of

native valvular regurgitation: a report from the American Society of Echocardiography developed in collaboration with the Society for Cardiovascular Magnetic Resonance. *J Am Soc Echocardiogr*. 2017;30(4):303–371. https://doi.org/10.1016/j.echo.2017.01.007

13. Hahn RT, Lawlor MK, Davidson CJ, et al. Tricuspid Valve Academic Research Consortium definitions for tricuspid regurgitation and trial endpoints. *Eur Heart J*. 2023;44(43):4508–4532. https://doi.org/10.1093/eurheartj/ehad653

14. Hahn RT, Weckbach LT, Noack T, et al. Proposal for a standard echocardiographic tricuspid valve nomenclature. *JACC Cardiovasc Imaging*. 2021;14(7):1299–1305. https://doi.org/10.1016/j.jcmg.2021.01.012

15. Dreyfus J, Galloo X, Taramasso M, et al. TRI-SCORE and benefit of intervention in patients with severe tricuspid regurgitation. *Eur Heart J*. 2024;45(8):586–597. https://doi.org/10.1093/eurheartj/ehad585

16. Adamo M, Russo G, Pagnesi M, et al. Prediction of mortality and heart failure hospitalization after transcatheter tricuspid valve interventions. *JACC Cardiovasc Interv*. 2024;17(7):859–870. https://doi.org/10.1016/j.jcin.2024.02.013

17. Coisne A, L'Official G, Dreyfus J, et al. Echocardiographic outcomes after transcatheter edge-to-edge repair in patients with isolated tricuspid regurgitation. JACC Cardiovasc Imaging. doi:10.1016/j.jcmg.2025.09.010.

18. Kwak S, Lim J, Yang S, et al. Atrial functional tricuspid regurgitation. *JACC Cardiovasc Imaging*. 2023;16(5):575–587. https://doi.org/10.1016/j.jcmg.2022.11.014

19. Jorde UP, Benza R, McCarthy PM, et al. Impact of renal and liver function on clinical outcomes following tricuspid valve transcatheter edge-to-edge repair. *J Am Coll Cardiol*. 2024;84(25):2446–2456. https://doi.org/10.1016/j.jacc.2024.08.044

20. Tomaselli M, Radu DN, Badano LP, et al. Right atrial remodeling and outcome in patients with secondary tricuspid regurgitation. *J Am Soc Echocardiogr*. 2024;37(5):495–505. https://doi.org/10.1016/j.echo.2024.01.003

21. Galloo X, Fortuni F, Meucci MC, et al. Association of right atrial strain and long-term outcome in severe secondary tricuspid regurgitation. *Heart*. 2024;110(6):448–456. https://doi.org/10.1136/heartjnl-2023-323084

22. Ben Ali W, Ludwig S, Duncan A, et al. Characteristics and outcomes of patients screened for transcatheter mitral valve implantation: 1-year results from the CHOICE-MI registry. *Eur J Heart Fail*. 2022;24(5):887–898. https://doi.org/10.1002/ejhf.2492

23. Sorajja P, Whisenant B, Hamid N, et al. Transcatheter repair for patients with tricuspid regurgitation. *N Engl J Med*. 2023;388(20):1833–1842. https://doi.org/10.1056/NEJMoa2300525

24. Donal E, Dreyfus J, Leurent G, et al. Transcatheter edge-to-edge repair for severe isolated tricuspid regurgitation: the Tri.Fr randomized clinical trial. *JAMA*. 2025;333(2):124–132. https://doi.org/10.1001/jama.2024.21189

25. Scotti A, Coisne A, Taramasso M, et al. Sex-related characteristics and short-term outcomes of patients undergoing transcatheter tricuspid valve intervention for tricuspid regurgitation. *Eur Heart J*. 2022;44(10):822–832. https://doi.org/10.1093/eurheartj/ehac735

26. Carlsson M, Ugander M, Heiberg E, Arheden H. The quantitative relationship between longitudinal and radial function in left, right, and total heart pumping in humans. *Am J Physiol Heart Circ Physiol*. 2007;293(1):H636–H644. https://doi.org/10.1152/ajpheart.01376.2006

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APPENDIX For a list of TRIPLACE study investigators and supplemental figures and tables, please see the online version of this paper.