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FOCUS ON TRICUSPID INTERVENTIONS

ORIGINAL RESEARCH: STRUCTURAL

Frequency and Outcomes of Acute Hemodynamic Instability Following Transcatheter Tricuspid Valve Replacement

Insights From the CESAR-TR-Registry

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ABSTRACT

BACKGROUND Transcatheter tricuspid valve replacement (TTVR) eliminates tricuspid regurgitation, leading to acute hemodynamic changes. It is unclear whether this might lead to acute hemodynamic instability (AHI).

OBJECTIVES The aim of this study was to report the frequency, outcomes, and risk factors of AHI following TTVR.

METHODS Patients undergoing TTVR at 5 international centers were included. AHI after TTVR was defined as class ≥ C shock according to the Society for Cardiovascular Angiography and Interventions classification.

RESULTS The study included 200 patients with a mean age of 78 ± 11 years (64% women). The frequency of AHI was 8.5%, and in all patients the onset was within 24 hours after the procedure. Compared with patients without post-procedural AHI, intrahospital mortality was significantly higher in AHI patients (35.2% vs 0.1%; $P < 0.001$). Univariate regression identified low glomerular filtration rate, reduced left ventricular ejection fraction, decreased ratio of tricuspid annular plane systolic excursion to mean pulmonary artery pressure, and elevated pulmonary capillary wedge pressure as significant factors of AHI.

CONCLUSIONS AHI is a life-threatening complication in the early postprocedural period following TTVR. Elevated left-sided filling pressures, pulmonary hypertension, and impaired renal function are associated with an increased risk for AHI, suggesting that patient selection and preprocedural optimization are critical.

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Tricuspid regurgitation (TR) results in chronic volume overload of the right ventricle (RV), promoting adverse ventricular remodeling and progressive dysfunction, which, in turn, exacerbates the severity of TR.[1] Transcatheter tricuspid valve interventions have emerged as promising therapeutic strategies to address this vicious cycle, with transcatheter edge-to-edge repair (TEER) being the most commonly used and a generally safe technique.[2] Hemodynamic instability following TEER is uncommon, and the incidence is described to be approximately 2.8%.[3]

More recently, orthotopic transcatheter tricuspid valve replacement (TTVR) has been introduced as an alternative treatment option, offering the potential for more complete elimination of the regurgitation. Although residual TR is frequently observed following repair techniques, TTVR typically achieves near complete resolution of valvular insufficiency.[4] However, it has been hypothesized that in patients with advanced RV dysfunction, TR may serve as a compensatory mechanism or "pop-off" valve, mitigating the hemodynamic burden on the failing RV. Consequently, abrupt elimination of TR through TTVR may lead to acute volume unloading while imposing increased afterload, thereby precipitating heart failure.[3,5]

This phenomenon, often described as RV afterload mismatch, is thought to arise from the RV's inability to adequately adapt to increased intracavitary pressure following restoration of valve competence.[6] This complication may occur more frequently in TTVR patients, who typically present with more severe TR and RV dysfunction compared with those undergoing repair. It is well recognized that reliable assessment of the RV has been challenging, with various definitions used.

The objective of this study was to characterize the frequency, outcomes, and risk factors of acute hemodynamic instability (AHI) following TTVR.

METHODS

STUDY POPULATION. The CESAR-TR (CT Prediction for Transcatheter Tricuspid Interventions) registry is an investigator-initiated, retrospective, single-arm, open-label, multicenter registry designed to analyze the value of computed tomography (CT) undertaken for analyses of valvular heart disease prior to transcatheter tricuspid interventions (NCT06951126). As per predefined inclusion criteria, the registry included all consecutive patients undergoing isolated orthotopic TTVR for severe TR using dedicated tricuspid valve devices. This international retrospective registry included 200 patients from 5 centers who underwent full-cycle cardiac CT for analysis of valvular heart disease between 2020 and 2024. All patients were deemed to be high risk for conventional surgery by the local heart team.

The study was approved and overseen by the local ethics committee, and the requirement to obtain informed consent for retrospective study inclusion was waived.

ECHOCARDIOGRAPHIC AND COMPUTED TOMOGRAPHIC ANALYSIS. Echocardiographic assessments followed current American Society of Echocardiography and European Association of Cardiovascular Imaging guidelines. Specific attention was paid to RV systolic function parameters, including tricuspid annular plane systolic excursion (TAPSE) and mean pulmonary artery pressure (mPAP) to assess RV-pulmonary arterial coupling. Definitions regarding the etiology and severity of TR and the study endpoints were established in accordance with the Tricuspid Valve Academic Research Consortium.[1] The severity of TR was graded according to the 5-grade scheme proposed by Hahn and Zamorano.[7] Computed tomographic analysis was performed on electrocardiographically gated, contrast-enhanced CT scans. CT data sets were analyzed using heart.ai (LARALAB).

INVASIVE HEMODYNAMIC ASSESSMENT. Invasive right heart catheterization was a mandatory component of the preprocedural work-up for all patients to confirm anatomical eligibility and assess pulmonary vascular resistance. The specific timing of this

ABBREVIATIONS AND ACRONYMS

AHI = acute hemodynamic instability

CT = computed tomography

GFR = glomerular filtration rate

LVEF = left ventricular ejection fraction

mPAP = mean pulmonary artery pressure

PCWP = pulmonary capillary wedge pressure

RV = right ventricle/ventricular

SCAI = Society for Cardiovascular Angiography and Interventions

TAPSE = tricuspid annular plane systolic excursion

TEER = transcatheter edge-to-edge repair

TR = tricuspid regurgitation

TTVR = transcatheter tricuspid valve replacement

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The authors attest they are in compliance with human studies committees and animal welfare regulations of the authors' institutions and Food and Drug Administration guidelines, including patient consent where appropriate. For more information, visit the Author Center.

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assessment varied according to local center protocols and trial participation. Hemodynamic measurements were obtained either intraprocedurally (immediately prior to device implantation) or during preprocedural screening. In a representative subcohort of 97 patients with precise timing data available, the mean interval between invasive assessment and the index procedure was 36.5 ± 67.2 days. Patients were maintained on stable guideline-directed medical therapy between the hemodynamic assessment and the intervention.

DATA HANDLING AND IMPUTATION. Although right heart catheterization was performed as standard of care, specific hemodynamic values (eg, pulmonary capillary wedge pressure [PCWP], mPAP) were missing from the source documentation in approximately 20% of cases because of incomplete data entry. To minimize selection bias and allow a robust multivariable analysis of the full cohort (N = 200), a random forest-based imputation (using the missForest package in R) was performed to account for these missing baseline variables, as detailed in the "Statistical Analysis" section.

OUTCOME MEASURES. The primary outcome of AHI after TTVR was defined as hemodynamic instability classified as shock stage ≥ C according to the Society for Cardiovascular Angiography and Interventions (SCAI) classification postprocedure.[8]

SCAI stage C (classic shock). SCAI stage C was defined as a patient manifesting with hypoperfusion who required intervention (pharmacologic or mechanical) beyond volume resuscitation to restore perfusion. This included patients presenting with relative hypotension and those requiring vasopressors or inotropes to maintain blood pressure, typically accompanied by biochemical evidence of hypoperfusion (eg, lactate > 2 mmol/L) or hemodynamic compromise (cardiac index < 2.2 L/min/m^2).[8]

SCAI STAGES D (DETERIORATING) AND E (EXTREMIS). SCAI stage D was defined as failure of the initial support strategy to restore perfusion, evidenced by worsening hemodynamic status or the need for escalating vasopressor doses or additional mechanical support. Stage E represented refractory shock with actual or impending circulatory collapse, including patients undergoing cardiopulmonary resuscitation or requiring multiple simultaneous interventions to sustain life.[8]

Outcomes, including SCAI shock stage, procedural success, bleeding complications, and acute kidney injury, were adjudicated retrospectively by local investigators at each site on the basis of chart review and strictly according to Tricuspid Valve Academic Research Consortium consensus definitions.[1] Secondary outcomes included in-hospital mortality and 30-day mortality.

STATISTICAL ANALYSIS. Statistical analysis was performed using SPSS Statistics 27 (IBM) and R version 4.4.2 (R Foundation for Statistical Computing). Continuous and normally distributed variables are expressed as mean ± SD. Parameters that were not normally distributed are expressed as median (Q1-Q3). Continuous variables were compared using 1-way analysis of variance (parametric test) or the Kruskal-Wallis test (nonparametric test) according to their distribution. Categoric variables are reported as numbers and corresponding proportions and were compared using the chi-square test with continuity correction or the Fisher exact test as appropriate. Key baseline variables had missing data, most notably wedge pressure (22.7%), mPAP (19.1%), left ventricular ejection fraction (LVEF) (5.3%), and glomerular filtration rate (GFR) (3.6%). To account for missing data in key baseline variables, a random forest-based imputation (using the missForest package in R) was performed. Univariable logistic regression was used to evaluate the association between immediate postprocedural AHI and continuous variables. Kaplan-Meier analysis was performed for clinical outcomes using time to first event. Differences in time-to-event distributions were evaluated using the log-rank test. HRs were calculated using Cox regression. P values <0.05 were considered to indicate statistical significance.

RESULTS

BASELINE CHARACTERISTICS. The study included 200 patients with significant TR, with a mean age of 78 ± 11 years (64% women). Detailed baseline characteristics are presented in **Table 1**. TR severity was classified as torrential in 34% of patients, massive in 23%, severe in 42%, and moderate in 2%. Surgical risk was elevated, with a median predicted in-hospital mortality rate after isolated tricuspid valve surgery of 14% (Q1-Q3: 4%-22%) according to the TRI-SCORE. Exertional dyspnea of NYHA functional class ≥ III was present in 91% of patients. The median baseline N-terminal prohormone of brain natriuretic peptide concentration was 733 pg/mL

TABLE 1 Baseline Data

	Overall Cohort (N = 200)	Hemodynamically Stable (n = 183)	Hemodynamically Unstable (n = 17)	*P* Value
Age, y	78 ± 11	78 ± 11	78 ± 8	0.289
Female	127 (64)	119 (65)	8 (47)	0.283
Body mass index, kg/m^2	26 ± 5	26 ± 5	24 ± 9	0.334
TRI-SCORE	5 (3-6)	4 (3-6)	5 (4-8)	0.087
Predicted in-hospital mortality after isolated tricuspid valve surgery according to TRI-SCORE, %	14 (4-22)	8 (4-22)	14 (8-48)	0.086
Coronary artery disease	77 (38)	75	2 (12)	0.240
Diabetes mellitus	44 (22)	41	3 (18)	0.999
Atrial fibrillation	184 (95)	171	13 (76)	0.575
Lung disease	26 (13)	24	2 (12)	0.999
History of stroke	25 (13)	25	2 (12)	0.999
Pacemaker lead in the right ventricle	81 (41)	74	7 (41)	0.130
Glomerular filtration rate, mL/min	48 ± 20	50 ± 19	40 ± 15	0.044
Previous surgical or transcatheter intervention				
Mitral valve	52 (26)	45 (25)	7 (41)	0.152
Aortic valve	33 (17)	29 (16)	4 (24)	0.491
Tricuspid valve	14 (7)	14 (8)	0 (0)	0.614
NYHA functional class				0.579
I	2 (1)	2 (1)	0 (0)	
II	12 (8)	20 (11)	0 (0)	
III	130 (85)	144 (79)	13 (76)	
IV	9 (6)	13 (7)	1 (6)	
Tricuspid regurgitation severity at baseline				0.173
2+	3 (2)	3 (2)	0 (0)	
3	84 (42)	80 (44)	4 (24)	
4	45 (23)	38 (21)	7 (41)	
5	68 (34)	63 (34)	5 (29)	
Tricuspid regurgitation etiology				0.971
Primary	16 (8)	15 (8)	1 (6)	
Atrial	58 (29)	53 (29)	5 (29)	
Ventricular	97 (48)	89 (49)	8 (47)	
CIED related	29 (15)	26 (14)	3 (18)	
LVEF, %	56.2 ± 9.1	56.5 ± 9	53.4 ± 10.1	0.398
TAPSE, mm	16.5 ± 4.8	16.8 ± 4.7	14.9 ± 4.2	0.092
Echocardiographic sPAP, mm Hg	41 ± 13	41 ± 12	46 ± 20	0.475
Hemoglobin, g/dL	12.1 ± 1.8	12.1 ± 1.9	11.3 ± 1.6	0.104
NT-proBNP, pg/mL	733 (310-2,170)	662 (298-1,998)	1,920 (369-4,600)	0.098
Albumin, mg/dL	3,410 ± 1,380	3,950 ± 550	3,700 ± 500	0.193
Bilirubin, mg/dL	1.0 (0.7-1.4)	1.0 (0.7-1.4)	1.2 (0.7-1.8)	0.636
Glutamic oxaloacetic transaminase, U/L	27 (21-35)	26 (21-35)	33 (29-37)	0.103
Glutamate-pyruvate transaminase, U/L	17 (12-25)	17 (12-23)	18 (13-22)	0.722

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

CIED = cardiac implantable electronic device; LVEF = left ventricular ejection fraction; NT-proBNP = N-terminal prohormone of brain natriuretic peptide; sPAP = systolic pulmonary artery pressure; TAPSE = tricuspid annular plane systolic excursion.

(Q1-Q3: 310-2,170 pg/mL), and the median baseline bilirubin concentration was borderline at 1.0 mg/dL (Q1-Q3: 0.7-1.4 mg/dL).

Paroxysmal or persistent atrial fibrillation was diagnosed in 92% of the study cohort. Additionally, 38% presented with comorbid coronary artery disease, and 41% had pacemaker leads through the tricuspid valve. Previous surgical or interventional treatment for the mitral valve had been performed in 26%, for the aortic valve in 17%, and for the tricuspid valve in 7%.

COMPUTED TOMOGRAPHIC VALUES AND INVASIVE HEMODYNAMIC STATUS. Data regarding multislice CT and invasive hemodynamic status are presented in Table 2. Multislice CT revealed significantly

TABLE 2 Computed Tomographic and Hemodynamic Data Stratified by Wall Tension

	Overall Cohort	Hemodynamically Stable	Hemodynamically Unstable	*P* Value
Computed tomographic data				
LVEDV, mL	107 (87-130)	116 ± 50	130 ± 51	0.368
LVESV, mL	44 (34-60)	54 ± 40	70 ± 36	0.143
LVSV, mL	61 (48-72)	62 ± 22	60 ± 27	0.758
LVEF, %	55 ± 12	55 ± 12	47 ± 14	0.017
LA diastolic volume, mL	134 (108-175)	146 ± 51	150 ± 82	0.868
RVEDV, mL	240 ± 75	244 ± 78	244 ± 55	0.973
RVESV, mL	111 ± 50	113 ± 52	119 ± 38	0.588
RVSV, mL	129 ± 37	131 ± 39	124 ± 34	0.504
RVEF, %	55 ± 9	54 ± 9	51 ± 9	0.183
RA diastolic volume, mL	236 (194-284)	241 ± 85	233 ± 75	0.692
Hemodynamic data				
Mean pulmonary artery pressure, mm Hg	27 ± 7	24 ± 8	29 ± 5	0.038
Systolic pulmonary artery pressure, mm Hg	42 ± 11	36 ± 12	41 ± 9	0.112
Diastolic pulmonary artery pressure, mm Hg	17 ± 6	16 ± 6	22 ± 9	0.001
Wedge pressure, mm Hg	18 ± 7	16 ± 6	23 ± 4	0.001
Cardiac index, L/m^2	2.1 ± 0.8	3.6 ± 2.2	3.0 ± 1.5	0.291
Pulmonary vascular resistance, Wood units	2.9 (1.7-4.2)	2.4 ± 1.6	3.0 ± 2.0	0.383
TAPSE/mPAP ratio, mm/mm Hg	0.71 ± 0.28	0.74 ± 0.27	0.48 ± 0.18	<0.001

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

LA = left atrial; LVEDV = left ventricular end-diastolic volume; LVESV = left ventricular end-systolic volume; LVSV = left ventricular stroke volume; mPAP = mean pulmonary artery pressure; RA = right atrial; RVEDV = right ventricular end-diastolic volume; RVEF = right ventricular ejection fraction; RVESV = right ventricular end-systolic volume; RVSV = right ventricular stroke volume; other abbreviations as in Table 2.

enlarged right heart chambers, with a mean RV end-diastolic volume of 240 ± 75 mL and a median diastolic right atrial volume of 236 mL (Q1-Q3: 194-284 mL). RV stroke volume was significantly greater than left ventricular stroke volume (125 mL [Q1-Q3: 103-154 mL] vs 61 mL [Q1-Q3: 49-72 mL]; $P < 0.001$). Biventricular systolic function was overall preserved, with a mean LVEF of 55% ± 12% and a mean RV ejection fraction of 55% ± 9%.

Invasive hemodynamic assessment showed a mean systolic pulmonary artery pressure of 42 ± 11 mm Hg, diastolic pulmonary artery pressure of 17 ± 6 mm Hg, and mPAP of 27 ± 7 mm Hg. Additional parameters from right heart catheterization included a mean cardiac index of 2.1 ± 0.8 L/min/m^2, a mean PCWP of 18 ± 7 mm Hg, and a median pulmonary vascular resistance of 2.9 Wood units (Q1-Q3: 1.7-4.2 Wood units).

PROCEDURAL CHARACTERISTICS. Procedural outcomes and follow-up data are summarized in Table 3. Of the 200 patients who underwent TTVR, 194 (97%) received the EVOQUE valve (Edwards Lifesciences) and 6 (3%) received the LuX-Valve (Jenscare Scientific). Intraprocedural success was achieved in 196 patients (98%), with no significant difference between the 2 devices (100% vs 98%; $P = 0.999$). At discharge, TR was reduced to mild or less in 92% of patients. The median length of stay in the intensive care unit was 2 days (Q1-Q3: 1-3 days). All-cause in-hospital mortality occurred in 7 patients (3.5%).

FREQUENCY AND OUTCOMES OF AHI. The frequency of AHI was 8.5% (Table 1). In all affected patients, cardiac shock developed within 24 hours post-procedure. Intraprocedural success rates (94% vs 98%; $P = 0.301$) and procedure duration (104 ± 27 minutes vs 108 ± 44 minutes; $P = 0.441$) did not differ significantly between patients with and those without AHI (Table 3). However, patients with AHI had a significantly longer median intensive care unit stay compared with those without postprocedural instability (6 days [Q1-Q3: 2-13 days] vs 2 days [Q1-Q3: 1-3 days]; $P = 0.005$).

Patients with AHI had a numerically higher frequency of bleeding events (classified as Tricuspid Valve Academic Research Consortium type ≥3 bleeding: 18% vs 7%; $P = 0.142$) and a significantly higher rate of acute kidney injury ≥2 (29% vs 9%; $P = 0.001$). The occurrence of conduction system disturbances requiring permanent pacemaker implantation was not different between the 2 groups (18% vs 8%; $P = 0.164$). In-hospital mortality was also significantly higher in AHI patients (35.2% vs 0.1%; $P < 0.001$). At 30 days, total mortality for the cohort was 3.5% (n = 7). Notably, no new mortality events

TABLE 3 Outcome After Transcatheter Tricuspid Valve Replacement

	Overall Cohort	Hemodynamically Stable	Hemodynamically Unstable	*P* Value
Type of device (n = 160)				0.466
EVOQUE	194 (97)	178 (97)	16 (94)	
LuX-Valve	6 (3)	5 (3)	1 (6)	
In-hospital death	7 (3.5)	1 (1)	6 (35)	<0.001
Procedure time, min	107 ± 43	108 ± 44	104 ± 27	0.705
Intraprocedural success	196 (98)	180 (98)	16 (94)	0.301
Days in intensive care unit	2 (1-3)	2 (1-3)	6 (2-13)	0.005
TVARC bleeding type ≥ 3	16 (8)	13 (7)	3 (18)	0.142
Acute kidney injury ≥2	22 (11)	17 (9)	5 (29)	0.001
Cerebrovascular event at 30 d	2 (1)	2 (1)	0 (0)	0.999
Conduction disturbances requiring permanent pacemaker implantation	17 (9)	14 (8)	3 (18)	0.164
TR at discharge (n = 191)				0.137
0	122 (61)	112 (61)	10 (59)	
1	62 (31)	58 (32)	4 (24)	
2	2 (1)	1 (1)	1 (6)	
3	5 (3)	5 (3)	0 (0)	

Values are n (%), mean ± SD, or median (Q1-Q3).
TR = tricuspid regurgitation; TVARC = Tricuspid Valve Academic Research Consortium.

occurred between hospital discharge and 30 days, indicating that the excess mortality risk was confined to the acute in-hospital phase.

Baseline comparisons revealed that AHI patients had numerically higher predicted in-hospital mortality according to the TRI-SCORE, though this difference was not statistically significant (14% [Q1-Q3: 8%-48%] vs 8% [Q1-Q3: 4%-22%]; $P = 0.086$). AHI patients had significantly lower GFRs (mean 40 ± 15 mL/min vs 50 ± 19 mL/min; $P = 0.044$) and lower CT-derived LVEFs (mean 47% ± 14% vs 55% ± 12%; $P = 0.017$) (**Central Illustration**). In addition, baseline mPAP (29 ± 5 mm Hg vs 24 ± 8 mm Hg; $P = 0.038$) and PCWP (23 ± 4 mm Hg vs 16 ± 6 mm Hg; $P = 0.001$) were significantly elevated in patients who developed AHI. Assessment of RV-pulmonary arterial coupling, defined as the ratio of TAPSE to mPAP, revealed significantly impaired coupling in patients who developed AHI compared with patients in stable condition (0.48 ± 0.18 mm/mm Hg vs 0.74 ± 0.27 mm/mm Hg; $P < 0.001$).

AHI PRESENTATION AND TREATMENT. Among patients with AHI, 11 (65%) presented with cardiac shock classified as stage C according to the SCAI classification, while 1 patient (6%) was classified as stage D and 5 patients (29%) as stage E (**Table 4**). Noradrenaline was the most commonly used catecholamine, administered in 76% of cases, followed by vasopressin in 12% and adrenaline or dobutamine in 6%. Noninvasive ventilation was required in 4 patients (24%), and reintubation was necessary in 3 patients (18%). Additionally, extracorporeal membrane oxygenation was used in 1 patient.

PREDICTORS FOR AHI OCCURRENCE. Regression analyses identified GFR, CT-derived LVEF, mPAP, TAPSE, TAPSE/mPAP ratio, and PCWP as significant predictors of AHI following TTVR (**Table 5**). A sensitivity analysis performing complete case analysis (excluding imputed data) confirmed that the primary predictors (PCWP, mPAP, TAPSE, TAPSE/mPAP ratio, and GFR) remained statistically significant with consistent effect sizes.

LANDMARK ANALYSIS. Survival outcomes were further analyzed by comparing patients with and those without AHI. A landmark analysis starting 30 days postprocedure was performed, with a median follow-up duration of 258 days (Q1-Q3: 79-364 days). Follow-up data beyond 30 days were available for 10 patients (59%) in the AHI cohort and 172 patients (94%) in the non-AHI cohort. Notably, no additional deaths occurred within 1 year postprocedure among AHI patients. As a result, in this landmark analysis, the HR for 1-year mortality was not significantly higher in AHI patients (0 deaths) compared with non-AHI patients (14 deaths; HR: 0.46; 95% CI: 0.00-1,318; $P = 0.556$).

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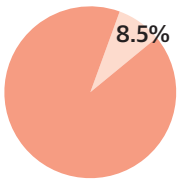
CENTRAL ILLUSTRATION Hemodynamic Instability Following TTVR

Acute Hemodynamic Instability (AHI) Following Transcatheter Tricuspid Valve Replacement: The CESAR-TR Registry, N = 200

- Consecutive TTVR patients from 5 centers
- AHI defined as SCAI-Shock class ≥C after TTVR



Hemodynamic instability:
- Overall frequency 8.5%
- 100% onset within 24 hours
- In-hospital mortality 35%

Risk Factors and Their Association With AHI

Variable	OR (95% CI)	*P* Value
Age, per y	1.00 (0.96-1.05)	0.868
RVEF, per %	0.96 (0.91-1.02)	0.185
GFR, per mL	0.97 (0.94-0.99)	0.047
PCWP, per mm Hg	1.17 (1.06-1.28)	0.001
LVEF, per %	0.96 (0.91-0.99)	0.021
TAPSE/mPAP ratio, per mm/mm Hg	0.01 (0.001-0.128)	<0.001

- AHI occurred in 8.5% of TTVR patients, with all events manifesting within 24 hours postprocedure.
- The development of AHI was associated with a 35% in-hospital mortality compared to <1% among stable patients.
- Significant risk factors for AHI included elevated pulmonary capillary wedge pressure, reduced LVEF, decreased TAPSE/mPAP ratio, and impaired renal function.

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AHI = acute hemodynamic instability; CESAR-TR = CT Prediction for Transcatheter Tricuspid Interventions; GFR = glomerular filtration rate; LVEF = left ventricular ejection fraction; mPAP = mean pulmonary artery pressure; PCWP = pulmonary capillary wedge pressure; RVEF = right ventricular ejection fraction; SCAI = Society for Cardiovascular Angiography and Interventions; TAPSE = tricuspid annular plane systolic excursion; TTVR = transcatheter tricuspid valve replacement.

TABLE 4 Clinical Presentation and Treatment of Hemodynamic Instability

SCAI shock class	
A or B	183 (92)
C	11 (6)
D	1 (1)
E	5 (3)
Treatment of AHI patients	
Reintubation	3 (18)
NIV	4 (24)
ECMO	1 (6)
Use of catecholamine in AHI patients	
Noradrenaline	13 (76)
Adrenaline	1 (6)
Dobutamine	1 (6)
Vasopressin	2 (12)

Values are n (%).

AHI = acute hemodynamic instability; ECMO = extracorporeal membrane oxygenation; NIV = noninvasive ventilation; SCAI = Society for Cardiovascular Angiography and Interventions.

DISCUSSION

This TTVR cohort offers critical insights into real-world outcomes associated with the TTVR procedure. The present analysis WAS focused on the frequency, outcomes, and risk factors of AHI following TTVR. The primary findings are as follows: 1) AHI occurred in 8.5% of the patients following TTVR; 2) all instances of AHI were observed in the first 24 hours post-TTVR; 3) if AHI occurred, mortality dramatically increased to 35%; and 4) GFR, CT-derived LVEF, TAPSE/mPAP ratio, and PCWP were significantly associated with the occurrence of AHI after TTVR.

FREQUENCY AND MORTALITY. Compared with tricuspid TEER, which has shown safety and efficacy in symptom alleviation, albeit with variable clinical and echocardiographic outcomes, TTVR shows a markedly different risk profile concerning

periprocedural risks.[9,10] This risk specifically includes conduction disturbances, bleeding, and cardiac shock following TTVR.[4]

In our cohort, AHI, defined as cardiac shock class C or worse according to the SCAI, occurred in 8.5% of patients following TTVR. This occurrence is substantially higher than previously reported for TEER, for which AHI has been observed in up to 2.3% of patients.[6] Furthermore, in-hospital mortality among patients who developed AHI was 35%, comparable with short-term mortality rates seen in general cardiogenic shock populations, which range from 30% to 40%.[8]

Furthermore, all-cause in-hospital mortality in our study was 3.5%. Although this is substantially higher than reported for T-TEER and is comparable with the mortality rate observed in the TRISCEND II trial (also 3.5%),[4] the severity of baseline TR is more severe, and TR reduction is more complete. It is also noteworthy that the overall in-hospital mortality rate of 3.5% remains markedly lower than the estimated mortality for surgical treatment of TR, as predicted by the TRI-SCORE at 14% (Table 1).

HEMODYNAMIC MECHANISM OF AHI. It has been shown that both TEER and TTVR procedures lead to a sudden increase in left ventricular filling due to improved RV forward flow. This increase in left ventricular end-diastolic volume has been reported to be approximately 10% and, in patients with more severe TR, up to 30%.[9] Additionally, pulmonary artery pressure has been observed to remain stable shortly after TTVR and TEER in patients with preserved left ventricular and RV function.[11-13] In advanced heart failure patients, it was assumed that the increase in left ventricular end-diastolic volume without an increase in left ventricular end-diastolic pressure in TEER is mitigated by residual TR functioning as a pressure outlet.[11] However, in TTVR this outlet might be closed, explaining the higher occurrence of AHI compared with TEER.

In our study, all cases of AHI occurred within the first 24 hours following the procedure, suggesting a strong association between the acute hemodynamic changes after TTVR and the onset of instability. The pathophysiology of AHI is complex and likely varies among individual patients. Because of the retrospective nature of the analysis, we can only speculate that the inability to adapt to the sudden reduction in regurgitant volume, combined with a simultaneous increase in RV forward flow, may be a key mechanism underlying AHI. Given that the mean ratio was significantly lower in the AHI group, this finding suggests that a key mechanism of AHI may be RV afterload mismatch. Noradrenaline was most often used in these patients, which seems reasonable, as it is often the catecholamine of first choice in cardiogenic shock patients.[8] Notably, in 4 patients (24%) noninvasive ventilation was necessary. Treatment of AHI with noninvasive ventilation indicates that at least in some patients, left heart failure and, consequently, pulmonary edema might have been a leading clinical problem. This is interesting because noninvasive ventilation increases RV afterload, which was thought to be a major driver of AHI after transcatheter tricuspid valve intervention. Additionally, regression analyses revealed that beyond baseline TAPSE/mPAP ratio, as well as CT-derived LVEF, PCWP, and kidney function, were significantly associated with the occurrence of AHI after TTVR. These findings suggest that TTVR imposes complex hemodynamic changes not just on the right heart but also interventricular dependence, left ventricular filling pressures, as well as systemic fluid balance, all of which may play critical roles in the development of AHI.

TABLE 5 Univariable Logistic Regression Predictors of Acute Hemodynamic Instability

	OR (95 CI)	*P* Value
Age, per year	1.00 (0.96-1.05)	0.868
Male	1.61 (0.59-4.4)	0.348
GFR, per mL	0.969 (0.94.0.99)	0.047
NT-proBNP, per pg/mL	1.00 (1.00-1.00)	0.284
TAPSE, per mm	0.87 (0.78-0.97)	<0.001
Previous valve intervention	1.3 (0.38-4.15)	0.710
Procedure time, per minute	0.99 (0.97-1.01)	0.439
Mean pulmonary artery pressure, per mm Hg	1.09 (1.01-1.15)	0.022
Mean wedge pressure, per mm Hg	1.17 (1.06-1.28)	0.001
LVEDV, per mL	1.00 (0.97-1.01)	0.352
LVESV, per mL	1.00 (0.99-1.01)	0.194
LVEF, per percent	0.955 (0.91-0.99)	0.021
RVEDV, per mL	1.00 (0.99-1.00)	0.979
RVESV, per mL	1.00 (0.99-1.01)	0.669
RVEF, per percent	0.96 (0.91-1.02)	0.185
TAPSE/mPAP ratio, per mm/mm Hg	0.01 (0.001-0.128)	<0.001

GFR = glomerular filtration rate; other abbreviations as in Tables 1 to 3.

PROPOSED MECHANISM OF AHI AFTER TTVR. Although the pathophysiology of AHI after TTVR is complex, and our interpretation is limited by the retrospective nature of this analysis, our results suggest that elevated PCWP plays a key role in its development. The sudden increase in RV forward flow may lead to

volume overload of the left ventricle, causing a further rise in PCWP. This adds to the often chronically elevated afterload on the RV. In patients undergoing TEER, this increased afterload can "pop off" through residual TR, acting as a pressure relief. However, in TTVR patients, elimination of this outlet may contribute to acute left and right heart failure. Additionally, impaired kidney function limits the ability to reduce intravascular volume, promoting persistent cardiac volume overload. These complex and abrupt hemodynamic changes, combined with an inability to adapt, drive the development of AHI and the clinical overlap of right and left heart failure.

MID-TERM SURVIVAL. It is reasonable to speculate that patients who experience AHI are generally more clinically or hemodynamically more tenuous and, therefore, more susceptible to adverse events than those who do not. To explore this, we performed a landmark analysis starting 30 days after the procedure. Our results show that once patients survive this critical early period, mid-term survival is comparable between those who experience AHI and those who do not. However, because of the limited number of remaining patients in the AHI cohort, these findings should be interpreted with caution.

Notably, the risk factors previously identified for AHI—such as reduced LVEF, pulmonary hypertension, and kidney dysfunction—are also established predictors of mortality following TEER. Therefore, it is expected that AHI patients may have worse mid- and long-term outcomes compared with non-AHI patients. Nonetheless, our data suggest that mortality does not remain markedly elevated once the acute AHI period has been overcome.

CLINICAL IMPLICATIONS. Our study provides valuable real-world insights into the risks of TTVR. As heart teams face the decision among surgery, TTVR, TEER, and conservative management, the risk for AHI must be weighed carefully. Although TTVR offers a more complete resolution of TR, the elimination of the "pop-off" mechanism carries a distinct hemodynamic cost.

The identification of decreased TAPSE/mPAP ratio, elevated PCWP, reduced LVEF, and renal dysfunction as predictors suggests that patients with combined precapillary and postcapillary pulmonary hypertension or biventricular compromise are at highest risk. For such patients, a preprocedural strategy focused on "ventricular unloading," such as aggressive diuretic dosing to normalize filling pressures before the procedure, might potentially lower the risk for AHI. However, whether this approach effectively decreases AHI frequency remains to be determined.

STUDY LIMITATIONS. This study has limitations inherent to retrospective analyses. Because of the design, it was not possible to reliably differentiate among right, left, and global heart failure phenotypes in every case. For this reason, we used the inclusive term *hemodynamic instability*. Furthermore, there was no independent core laboratory adjudication of clinical events, though definitions were standardized.

Crucially, the relatively small number of AHI events (n = 17) limits the statistical power of the regression analyses. Therefore, the identified predictors should be viewed as significant associations rather than components of a validated predictive model. Also, routine prehabilitation was not used at the centers. This limits the ability to discern whether this strategy could be beneficial is preselected cohorts.

Additionally, we lacked systematic invasive hemodynamic data immediately postprocedure in AHI patients, which limits our ability to granularly characterize the shock state (eg, differentiating pure RV failure from mixed shock). This reflects current clinical practice, in which invasive monitoring is not universal. Future studies with dedicated hemodynamic protocols are needed to further elucidate the mechanisms of AHI.

CONCLUSIONS

AHI is a clinically significant and life-threatening complication following TTVR, occurring in 8.5% of patients. Elevated wedge pressure, decreased TAPSE/mPAP ratio, reduced LVEF, and impaired GFR are key factors associated with its development. Recognition of these risk factors is essential for patient selection and may guide preprocedural optimization strategies.

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PERSPECTIVES

WHAT IS KNOWN? TTVR is increasingly used to treat patients with severe TR. By eliminating TR, TTVR induces acute changes in hemodynamic status.

WHAT IS NEW? Following TTVR, AHI occurs in 8.5% of cases and represents a life-threatening complication. Elevated baseline PCWP, RV–pulmonary arterial uncoupling, reduced LVEF, and impaired renal function are associated with the development of AHI.

WHAT IS NEXT? Future studies should evaluate whether preprocedural strategies such as aggressive pharmacologic ventricular unloading to reduce filling pressures can mitigate the risk for AHI in patients at risk.

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