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TRI-SCORE and Right Ventricular Free-Wall Longitudinal Strain for Mortality Risk Stratification After Transcatheter Tricuspid Valve Intervention

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Abstract

Risk stratification after transcatheter tricuspid valve intervention (TTVI) remains challenging, and right ventricular free-wall longitudinal strain (RVFWLS) may complement established clinical assessment. This prospective single-centre cohort study evaluated the prognostic performance of the TRI-SCORE and the incremental value of pre-interventional RVFWLS for all-cause mortality after TTVI. Analyses included 56 patients at 12 months and 54 at 24 months. Discrimination was assessed using receiver operating characteristic analysis, paired DeLong tests, Kaplan–Meier analysis and Cox regression. Eleven patients died within 12 months and sixteen within 24 months. RVFWLS magnitude was lower among 24-month non-survivors than survivors (18.0% [IQR 15.0–23.2] vs. 23.5% [19.0–26.0]; $p = 0.010$). TRI-SCORE AUCs were 0.778 and 0.746 for 12- and 24-month mortality, respectively. Adding RVFWLS increased the AUCs to 0.806 (ΔAUC 0.028; $p = 0.322$) and 0.771 (ΔAUC 0.025; $p = 0.387$), respectively. High TRI-SCORE remained independently associated with mortality at 12 months (HR 6.39, 95% CI 1.29–31.79) and 24 months (HR 5.10, 95% CI 1.55–16.81), whereas RVFWLS was not independently associated. TRI-SCORE provided robust mortality stratification after TTVI. RVFWLS identified greater right ventricular vulnerability among non-survivors, but did not significantly improve discrimination in this cohort.

Keywords: tricuspid regurgitation; transcatheter tricuspid valve intervention; TRI-SCORE; right ventricular free-wall longitudinal strain; echocardiography; mortality

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1. Introduction

Severe tricuspid regurgitation (TR) is associated with progressive right-sided heart failure, recurrent hospitalisation, end-organ dysfunction and excess mortality [1–3].

Transcatheter tricuspid valve intervention (TTVI) has expanded treatment options for patients who are elderly, multimorbid and considered at high surgical risk. Contemporary transcatheter edge-to-edge repair and valve implantation studies have demonstrated substantial reductions in TR severity and clinically meaningful improvements in symptoms and quality of life [4–8]. Most recently, the randomised TRIC-I-HF trial showed that transcatheter tricuspid valve repair plus medical therapy reduced the combined risk of death or heart-failure hospitalisation compared with medical therapy alone [9]. Nevertheless, mortality among patients undergoing TTVI remains considerable, reflecting late referral, advanced right ventricular (RV) disease and the systemic consequences of chronic venous congestion [2,10].

Patient selection therefore extends beyond anatomical feasibility. A clinically useful risk model should identify advanced disease, quantify competing risk and help distinguish patients likely to derive durable benefit from those in whom intervention may be futile. The TRI-SCORE was originally developed to predict in-hospital mortality after isolated tricuspid valve surgery and integrates medical history, clinical status and baseline echocardiographic findings [11]. Subsequent work has supported its value across severe TR and transcatheter cohorts [12–14]. However, any composite score necessarily simplifies RV pathophysiology and may not fully capture myocardial reserve.

Conventional RV indices such as tricuspid annular plane systolic excursion (TAPSE) and fractional area change are influenced by loading conditions, geometry and regional contraction. Two-dimensional speckle-tracking-derived right ventricular free-wall longitudinal strain (RVFWLS) quantifies myocardial deformation and may detect subclinical dysfunction before conventional measures become abnormal [15]. In significant TR, impaired RVFWLS has been associated with adverse outcomes [16]. Studies of TTVI populations further suggest that baseline RV function, contraction pattern and postprocedural remodelling influence prognosis [17–22]. The marked preload and afterload changes produced by TR reduction make an integrated clinical and imaging assessment particularly attractive.

However, evidence on whether pre-interventional RVFWLS adds measurable discrimination beyond a dedicated TR risk score remains limited. We therefore evaluated the association of the pre-interventional TRI-SCORE and RVFWLS with 12- and 24-month all-cause mortality after TTVI. We hypothesised that the TRI-SCORE would identify a high-risk subgroup and that incorporation of RVFWLS would improve mortality discrimination beyond the TRI-SCORE alone.

2. Materials and Methods

2.1. Study Design and Population

This prospective single-centre cohort study included 68 patients from an ongoing TTVI registry at the Heart Centre Brandenburg, Brandenburg Medical School Theodor Fontane. Patients were prospectively enrolled between 13 November 2022 and 7 April 2024. Follow-up data were collected through 20 May 2026. Adults with symptomatic severe TR selected for TTVI by an interdisciplinary Heart Team were eligible. Severe TR was defined as grade ≥ 3 using the expanded five-grade classification [23].

Eligibility for intervention was based on clinical status, imaging, anatomical feasibility and surgical risk. Exclusion criteria in the underlying registry were severe comorbid conditions precluding safe participation, untreated clinically relevant left-sided valve disease and refusal or inability to provide written informed consent. Cohort selection is summarised in Figure 1.

The registry has supported related risk-score, biomarker and haemodynamic analyses [13,24–28]. The present study evaluates the distinct relationship of baseline RVFWLS to the TRI-SCORE and mortality through 24 months. Reporting follows the Strengthening

the Reporting of Observational Studies in Epidemiology (STROBE) recommendations [29].

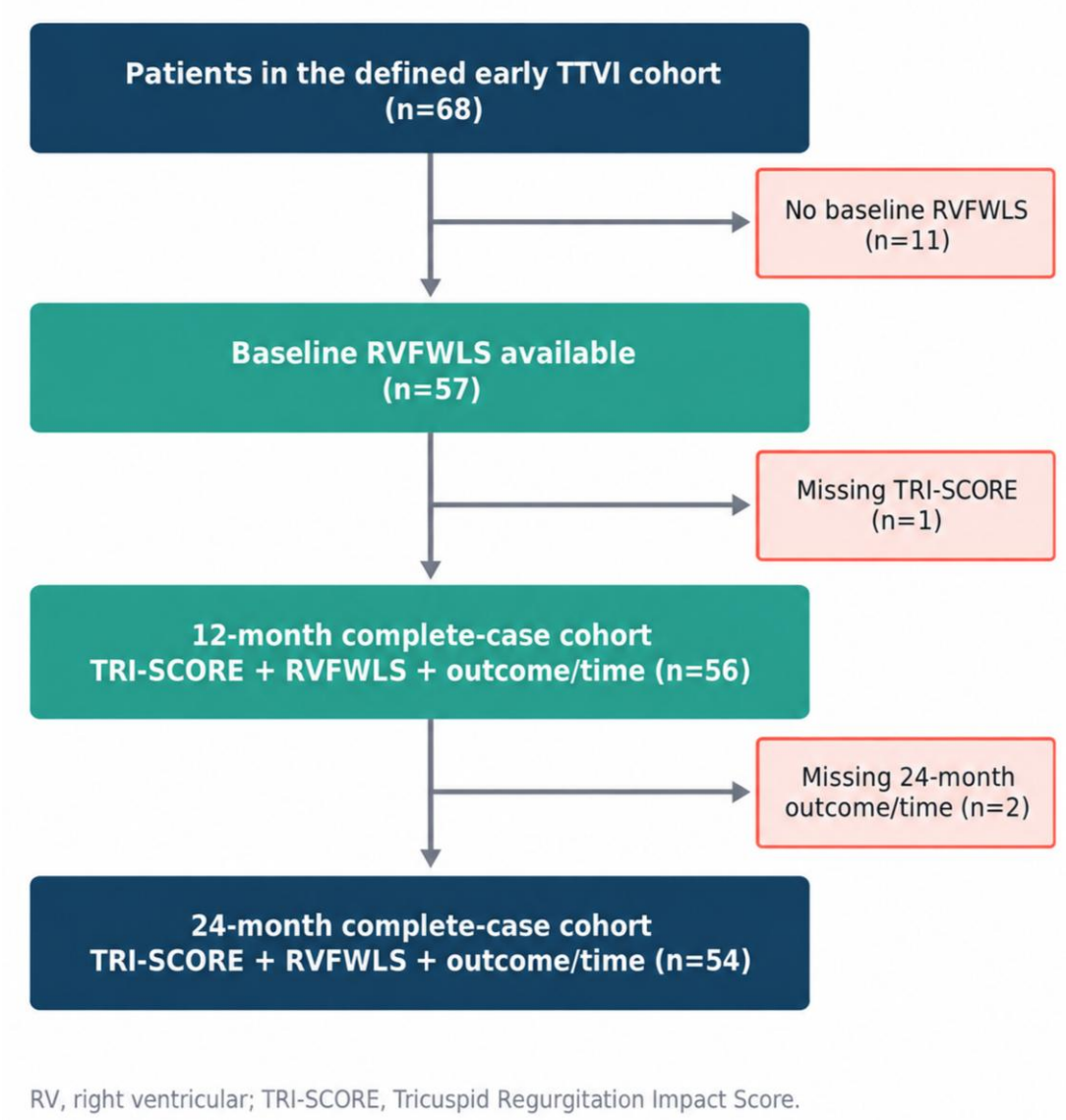


Figure 1. Study flow chart. Patients in the defined early TTVI cohort, availability of baseline RVFWLS and TRI-SCORE, and complete-case cohorts for 12- and 24-month mortality. RVFWLS, right ventricular free-wall longitudinal strain; TRI-SCORE, Tricuspid Regurgitation Impact Score; TTVI, transcatheter tricuspid valve intervention.

2.2. Ethics

This study was approved by the institutional ethics committee of Brandenburg Medical School Theodor Fontane (E-01-20190503). All participants provided written informed consent. This study was conducted in accordance with the Declaration of Helsinki.

2.3. Clinical and Echocardiographic Assessment

Demographic characteristics, comorbidities, laboratory values, risk scores and echocardiographic measurements were collected before TTVI. The baseline 6 min walk distance was recorded. TR severity was graded before and after TTVI using the five-grade scheme from mild to torrential [23]. TricValve and EVOQUE™ were excluded from the TR reduction analysis due to their distinct mechanisms. Left ventricular ejection fraction, TAPSE, RV fractional area change, RV basal diameter, right atrial area and three-

dimensional RV volumes were recorded where available in accordance with contemporary chamber-quantification principles [30,31]. Echocardiography at discharge was used to document residual TR. Baseline RVFWLS was measured before intervention from a dedicated RV-focused apical four-chamber view in all patients, including the patient subsequently treated with EVOQUE™. No post-interventional RVFWLS measurements were used in the present analyses. Images were acquired using a Vivid E95 ultrasound system (GE Healthcare; system software version 202:81.0), generally within a few days before TTVI, and analysed offline using EchoPAC (GE Healthcare). The reported RVFWLS represented the mean strain value across the basal, midventricular and apical free-wall segments. In patients with atrial fibrillation, EchoPAC used multiple cardiac cycles to automatically derive a representative averaged cycle and select the technically most suitable loop for analysis. A valid electrocardiographic signal and a frame rate of at least 50 frames per second were required by the software. RVFWLS measurements were performed and independently reviewed by two investigators blinded to subsequent clinical outcomes.

RVFWLS is stored in the database as an absolute magnitude, such that a lower positive value denotes worse longitudinal deformation. No separate formal intraobserver or interobserver reproducibility analysis was performed.

2.4. TRI-SCORE

The TRI-SCORE was calculated according to the original definition [11]. It comprises eight weighted variables: age ≥ 70 years, New York Heart Association (NYHA) functional class III or IV, left ventricular ejection fraction < 60%, and moderate or severe RV dysfunction each contribute one point, signs of right-sided heart failure, a daily furosemide dose ≥125 mg, estimated glomerular filtration rate < 30 mL/min/1.73 m^2, and elevated total bilirubin each contribute two points. The total score ranges from 0 to 12 points. Creatinine-based estimated glomerular filtration rate was calculated using the Berlin Initiative Study 1 (BIS-1, 2012) equation. For survival analyses, the low- and intermediate-risk categories were combined (≤5 points) and compared with the high-risk category (≥6 points). The continuous score was retained for receiver operating characteristic (ROC) modelling.

2.5. Transcatheter Intervention and Invasive Haemodynamics

Device selection was determined by anatomy, RV function, clinical characteristics and Heart Team consensus. Procedures comprised transcatheter edge-to-edge repair (T-TEER) with PASCAL™ (Edwards Lifesciences, Irvine, CA, USA) or TriClip™ (Abbott Vascular, Santa Clara, CA, USA), heterotopic caval valve implantation with TricValve® (P+F Products + Features GmbH, Vienna, Austria) and orthotopic valve implantation with EVOQUE™ (Edwards Lifesciences, Irvine, CA, USA). Echocardiographic procedural success at discharge was evaluated only after T-TEER and was defined as a reduction in TR of at least two grades with residual TR ≤ 2. TricValve and EVOQUE™ were excluded from the TR reduction analysis because caval valve implantation does not reduce native TR and orthotopic valve replacement is expected to eliminate relevant residual TR entirely. Right-heart catheterisation was performed before intervention as part of the structured screening pathway. Pulmonary vascular resistance, right atrial pressure, RV end-diastolic pressure and mean pulmonary artery pressure were available for descriptive analyses.

2.6. Endpoints and Follow-Up

All-cause mortality was assessed at 12 and 24 months after TTVI, with the 24-month outcome serving as the primary endpoint. Vital status and survival time were ascertained through review of institutional medical records, scheduled follow-up examinations at our heart centre and, when required, contact with primary care physicians or patients' relatives by telephone. Patients with incomplete ascertainment were classified as lost to

follow-up and excluded from the analysis at the respective time point. Follow-up time was censored at 12 or 24 months, respectively.

2.7. Statistical Analysis

Continuous variables are presented as median and interquartile range (IQR), and categorical variables as count and percentage. Baseline characteristics were described for the 12-month complete-case cohort and compared between patients alive and dead at 24 months using the Mann–Whitney U test for continuous variables and Fisher's exact test for categorical variables. Analyses were based on available data. Therefore, denominators varied across variables. Discharge TR grade was evaluated only in patients undergoing T-TEER. Baseline comparisons were exploratory and no adjustment for multiple testing was applied. The relationship between baseline RVFWLS magnitude and continuous TRI-SCORE was assessed using Spearman's rank correlation coefficient.

Fixed-horizon discrimination for all-cause mortality was assessed using receiver operating characteristic curves. Areas under the receiver operating characteristic curve (AUCs) and 95% confidence intervals (CIs) were estimated using DeLong's method. At each time horizon, an unpenalized logistic regression model including continuous TRI-SCORE and RVFWLS was used to generate predictions for the combined model. Paired DeLong tests compared the TRI-SCORE with RVFWLS alone and with the combined model. Due to the limited number of events, multivariable survival models were restricted to two covariates. To contextualize statistical sensitivity, approximate detectable AUC differences for 80% power at a two-sided $\alpha = 0.05$ were calculated as (1.96 + 0.84) times the observed standard error of the paired AUC difference, assuming unchanged variance.

Kaplan–Meier curves compared high- versus low/intermediate-risk TRI-SCORE categories using the log-rank test. Cox proportional hazards regression estimated hazard ratios (HRs) with 95% confidence intervals for the high-risk category, first in univariable models and subsequently after inclusion of RVFWLS. Exact intervention dates were available for all patients, whereas exact death dates were available only for a subset. Survival time was recorded as completed months after intervention, with fractional months rounded down. Deaths occurring before completion of the first month were recorded as 0 months and assigned 0.5 months for Kaplan–Meier and Cox analyses. Ties were handled using the Efron method. All tests were two-sided, and $p < 0.05$ was considered statistically significant. An exploratory sensitivity analysis restricted to T-TEER recipients repeated the fixed-horizon ROC analyses, paired DeLong comparisons and two-covariate Cox models using the same analytical approach. Statistical analyses were performed using IBM SPSS Statistics, version 29.0 (IBM Corp., Armonk, NY, USA), and Python 3 (Python Software Foundation, Wilmington, DE, USA).

3. Results

3.1. Study Population

The 12-month complete-case cohort included 56 patients, of whom 54 had complete 24-month follow-up data (Figure 1). Median age was 82.0 years (IQR 77.0–85.0), and 27 patients (48.2%) were men. T-TEER was performed in 45 patients (80.4%), comprising 35 PASCAL and 10 TriClip procedures. Ten patients underwent TricValve implantation (17.9%) and one underwent EVOQUE™ implantation (1.8%). A total of 7 of the 10 TricValve recipients (70.0%) died within 24 months. TricValve use was more frequent among non-survivors than survivors (43.8% vs. 7.9%; $p = 0.004$; Table 1). Among patients undergoing T-TEER, echocardiographic procedural success at discharge was achieved in 34 of 45 patients (75.6%). Further baseline characteristics are summarised in Table 1.

Table 1. Baseline characteristics according to 24-month survival status.

Characteristic	Overall (*n* = 56)	Alive at 24 Months (*n* = 38)	Dead by 24 Months (*n* = 16)	*p* Value
Demographics				
Age, years	82.0 [77.0–85.0]	81.5 [77.0–84.0]	84.5 [79.5–85.0]	0.437
Male sex	27 (48.2)	19 (50.0)	7 (43.8)	0.770
Body mass index, kg/m²	24.9 [24.0–27.9]	24.8 [24.1–28.2]	25.6 [23.8–26.8]	0.985
Procedure				
PASCAL	35 (62.5)	27 (71.1)	7 (43.8)	0.072
TriClip	10 (17.9)	8 (21.1)	2 (12.5)	0.705
TricValve	10 (17.9)	3 (7.9)	7 (43.8)	0.004
EVOQUE™	1 (1.8)	0 (0.0)	0 (0.0)	1.000
Risk scores				
MELD-XI	14.0 [11.0–17.0]	13.0 [11.0–16.0]	16.0 [11.0–18.5]	0.143
EuroSCORE II, %	4.3 [2.6–6.5]	3.6 [2.3–5.9]	5.6 [4.2–7.8]	0.097
TRI-SCORE, points	5.0 [4.0–6.0]	4.0 [3.0–5.0]	6.0 [5.8–7.0]	0.004
TRI-SCORE high-risk category (≥6)	22 (39.3)	9 (23.7)	12 (75.0)	<0.001
Comorbidities and renal function				
eGFR, mL/min/1.73 m²	43.0 [32.0–49.2]	45.0 [33.0–50.8]	36.0 [28.8–43.8]	0.113
Type 2 diabetes mellitus	22 (39.3)	14 (36.8)	7 (43.8)	0.762
Coronary artery disease	31 (55.4)	22 (57.9)	8 (50.0)	0.765
Atrial fibrillation	53 (94.6)	36 (94.7)	15 (93.8)	1.000
COPD	9 (16.1)	6 (15.8)	3 (18.8)	1.000
Functional status				
6 min walk distance, m	212.5 [79.2–340.0]	300.0 [100.0–363.0]	94.0 [60.5–222.5]	0.075
Echocardiography				
LVEF, %	52.0 [44.0–58.0]	52.0 [45.0–58.0]	45.0 [39.8–57.8]	0.258
TAPSE, mm	16.0 [14.0–18.5]	16.0 [14.0–18.0]	16.0 [13.8–18.2]	0.861
TR grade at baseline	4.0 [4.0–5.0]	4.0 [4.0–5.0]	4.5 [4.0–5.0]	0.248
TR grade at discharge, T-TEER	2.0 [1.2–2.0]	2.0 [1.1–2.0]	1.5 [1.5–2.0]	0.583
Pre-interventional RVFWLS magnitude, %	23.0 [18.0–25.0]	23.5 [19.0–26.0]	18.0 [15.0–23.2]	0.010
RV fractional area change, %	39.0 [33.5–43.1]	39.1 [34.9–44.2]	34.7 [27.5–40.1]	0.085
RV basal diameter, mm	61.0 [51.0–67.2]	61.0 [51.2–67.8]	59.0 [47.0–64.0]	0.617
Right atrial area, cm²	36.8 [27.7–41.3]	36.8 [28.4–40.2]	34.4 [26.8–41.8]	0.887
RV end-diastolic volume, mL	162.5 [116.8–198.2]	168.0 [125.5–198.8]	151.0 [112.0–192.0]	0.617
RV stroke volume index, mL/m²	46.6 [32.3–60.7]	45.6 [32.5–59.5]	52.1 [29.9–62.0]	0.923
Invasive haemodynamics				
Pulmonary vascular resistance, WU	2.0 [1.6–2.4]	1.9 [1.3–2.4]	2.1 [1.9–2.5]	0.237
Right atrial pressure, mmHg	11.0 [9.0–16.0]	10.0 [7.8–16.0]	13.5 [11.8–16.0]	0.049

RV end-diastolic pressure, mmHg	10.5 [8.0–13.0]	9.0 [8.0–13.0]	13.0 [9.5–14.0]	0.195
Mean pulmonary artery pressure, mmHg	26.5 [23.0–32.0]	26.0 [23.0–35.0]	28.0 [24.5–29.5]	0.841

Values are presented as median [IQR] or *n* (%). The overall column describes the 12-month complete-case cohort (*n* = 56); survival-group comparisons include the 54 patients with complete 24-month outcome and follow-up data. Analyses used available data, so denominators may vary by variable. P values compare survivors and non-survivors at 24 months. COPD, chronic obstructive pulmonary disease; eGFR, estimated glomerular filtration rate; IQR, interquartile range; LVEF, left ventricular ejection fraction; MELD-XI, Model for End-Stage Liver Disease excluding INR; RV, right ventricular; RVEDP, right ventricular end-diastolic pressure; RVFWLS, right ventricular free-wall longitudinal strain; TAPSE, tricuspid annular plane systolic excursion; TR, tricuspid regurgitation; T-TEER, transcatheter tricuspid edge-to-edge repair; WU, Wood unit.

3.2. Mortality and TRI-SCORE Risk Categories

A total of 11 of 56 patients (19.6%) died within 12 months and 16 of 54 (29.6%) died within 24 months. The 12-month mortality was 5.9% (2/34) in the low/intermediate-risk group and 40.9% (9/22) in the high-risk group. At 24 months, mortality was 12.1% (4/33) and 57.1% (12/21), respectively. Kaplan–Meier analysis demonstrated significantly poorer survival in the high-risk group (log-rank $p < 0.001$; Figure 2).

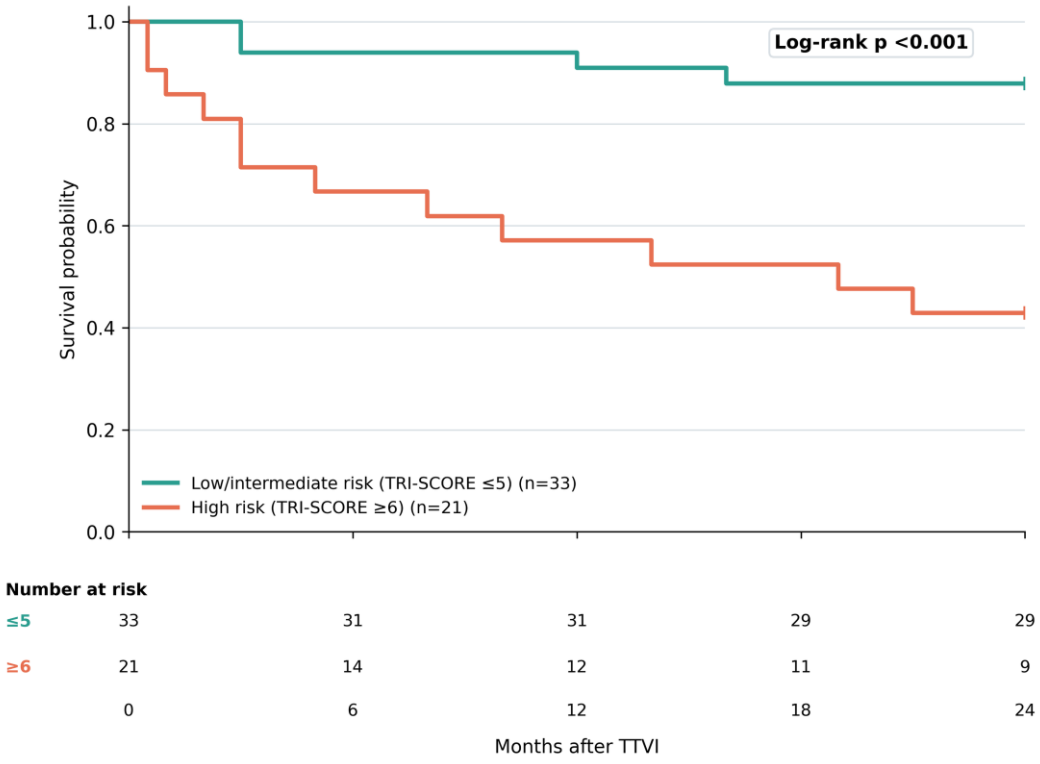


Figure 2. Kaplan–Meier survival according to TRI-SCORE risk category. The 24-month survival after TTVI in patients with low/intermediate (≤5) versus high (≥6) TRI-SCORE. Tick marks denote censoring; numbers at risk are displayed below the plot. TRI-SCORE, Tricuspid Regurgitation Impact Score; TTVI, transcatheter tricuspid valve intervention.

3.3. RVFWLS and Mortality

Patients who died by 12 months had lower baseline RVFWLS magnitude than survivors (17.0% [IQR 14.5–22.5] vs. 23.0% [19.0–26.0]; $p = 0.030$). The difference was also present at 24 months (18.0% [15.0–23.2] vs. 23.5% [19.0–26.0]; $p = 0.010$). Among conventional imaging variables, neither TAPSE nor RV basal diameter differed according to 24-month

survival status; RV fractional area change was numerically lower among non-survivors. These unadjusted comparisons should be interpreted as descriptive because of the small cohort and multiple correlated RV measures. Baseline RVFWLS magnitude was inversely correlated with continuous TRI-SCORE (Spearman's $\rho = -0.358$; $p = 0.007$; $n = 56$). TAPSE was available in 55 patients, of whom 32 (58.2%) had a TAPSE < 17 mm.

3.4. Discrimination at 12 and 24 Months

For 12-month mortality, the TRI-SCORE achieved an AUC of 0.778 (95% CI 0.651–0.904), compared with 0.713 (0.519–0.907) for RVFWLS. The combined TRI-SCORE/RVFWLS model achieved an AUC of 0.806 (0.676–0.936), an absolute increase of 0.028 over the TRI-SCORE alone. This difference was not statistically significant (paired DeLong $p = 0.322$).

For 24-month mortality, the corresponding AUCs were 0.746 (95% CI 0.592–0.900) for the TRI-SCORE, 0.725 (0.574–0.875) for RVFWLS and 0.771 (0.621–0.921) for the combined model. The absolute AUC increase was 0.025 and was not statistically significant (paired DeLong $p = 0.387$) (Table 2; Figure 3). In the exploratory T-TEER-only sensitivity analysis, 4/45 patients died within 12 months and 9/44 within 24 months. Adding RVFWLS increased the AUC from 0.841 to 0.860 at 12 months (ΔAUC 0.018; $p = 0.700$) and from 0.724 to 0.763 at 24 months (ΔAUC 0.040; $p = 0.432$). These apparent discrimination estimates were derived and evaluated in the same subgroup. Detailed results are provided in Supplementary Table S1.

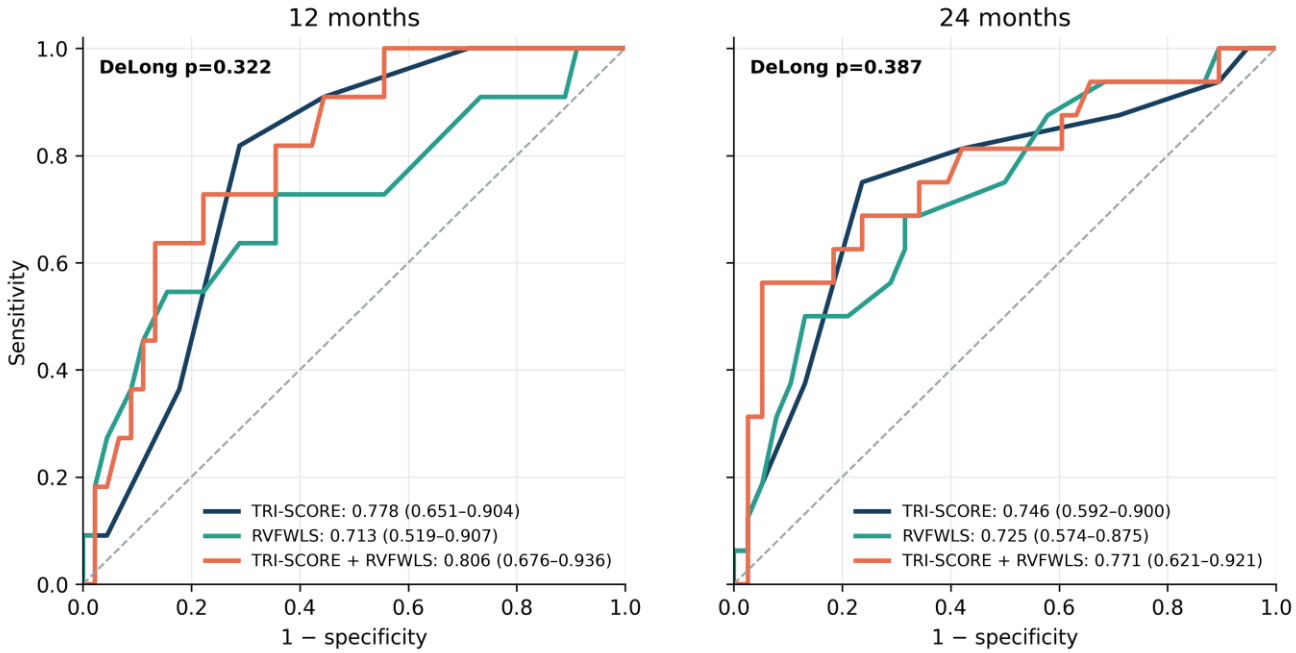


Figure 3. Receiver operating characteristic curves for 12- and 24-month mortality. Discrimination of the TRI-SCORE, RVFWLS and the combined TRI-SCORE/RVFWLS logistic model. AUC values are shown with 95% confidence intervals. Paired DeLong p values compare the combined model with TRI-SCORE alone. AUC, area under the receiver operating characteristic curve; RVFWLS, right ventricular free-wall longitudinal strain; TRI-SCORE, Tricuspid Regurgitation Impact Score. The diagonal dashed line represents the line of no discrimination (AUC = 0.50).

Table 2. Discrimination for all-cause mortality.

Horizon	Model	Events/n	AUC (95% CI)	ΔAUC	DeLong *p*
12 months	TRI-SCORE	11/56	0.778 (0.651–0.904)	0.000	—
12 months	RVFWLS	11/56	0.713 (0.519–0.907)	−0.065	0.465
12 months	TRI-SCORE + RVFWLS	11/56	0.806 (0.676–0.936)	0.028	0.322
24 months	TRI-SCORE	16/54	0.746 (0.592–0.900)	0.000	—
24 months	RVFWLS	16/54	0.725 (0.574–0.875)	−0.021	0.809
24 months	TRI-SCORE + RVFWLS	16/54	0.771 (0.621–0.921)	0.025	0.387

Values are AUCs with 95% CIs. Events/n indicates deaths/total patients analysed. ΔAUC is the absolute difference from the TRI-SCORE alone at the corresponding horizon; DeLong p values compare each model with the TRI-SCORE alone. The combined model included continuous TRI-SCORE and RVFWLS. AUC, area under the receiver operating characteristic curve; CI, confidence interval; RVFWLS, right ventricular free-wall longitudinal strain; TRI-SCORE, Tricuspid Regurgitation Impact Score.

3.5. Cox Regression Associations

In Cox regression, high TRI-SCORE category was associated with 12-month mortality (HR 8.49, 95% CI 1.83–39.33; $p = 0.006$) and 24-month mortality (HR 6.46, 95% CI 2.08–20.11; $p = 0.001$). The association remained after inclusion of RVFWLS at 12 months (HR 6.39, 95% CI 1.29–31.79; $p = 0.023$) and 24 months (HR 5.10, 95% CI 1.55–16.81; $p = 0.007$). RVFWLS magnitude was not independently associated with mortality after adjustment for TRI-SCORE category at 12 months (HR per 1 percentage point 0.94, 95% CI 0.86–1.03; $p = 0.168$) or 24 months (HR 0.95, 95% CI 0.88–1.02; $p = 0.130$) (Table 3; Figure 4). In the exploratory T-TEER-only analysis, RVFWLS was associated with 24-month mortality after adjustment for TRI-SCORE category (HR per 1 percentage point higher magnitude 0.843, 95% CI 0.714–0.995; $p = 0.043$), whereas the corresponding association at 12 months was not statistically significant (HR 0.832, 95% CI 0.644–1.075; $p = 0.160$; Supplementary Table S1).

Table 3. Cox regression associations with all-cause mortality.

Horizon	Exposure	Model	HR (95% CI)	*p* Value
12 months	TRI-SCORE high vs. low/intermediate	Univariable	8.49 (1.83–39.33)	0.006
12 months	TRI-SCORE high vs. low/intermediate	Adjusted for RVFWLS	6.39 (1.29–31.79)	0.023
12 months	RVFWLS per 1 percentage point higher magnitude	Adjusted for TRI-SCORE category	0.94 (0.86–1.03)	0.168
24 months	TRI-SCORE high vs. low/intermediate	Univariable	6.46 (2.08–20.11)	0.001
24 months	TRI-SCORE high vs. low/intermediate	Adjusted for RVFWLS	5.10 (1.55–16.81)	0.007
24 months	RVFWLS per 1 percentage point higher magnitude	Adjusted for TRI-SCORE category	0.95 (0.88–1.02)	0.130

High risk was defined as TRI-SCORE ≥ 6 and compared with low/intermediate risk (≤5). RVFWLS is expressed as absolute magnitude; its HR represents a 1 percentage point higher value. Adjusted models included both TRI-SCORE category and RVFWLS. For deaths recorded as 0 whole months, 0.5 months was assigned. CI, confidence interval; HR, hazard ratio; RVFWLS, right ventricular free-wall longitudinal strain; TRI-SCORE, Tricuspid Regurgitation Impact Score.

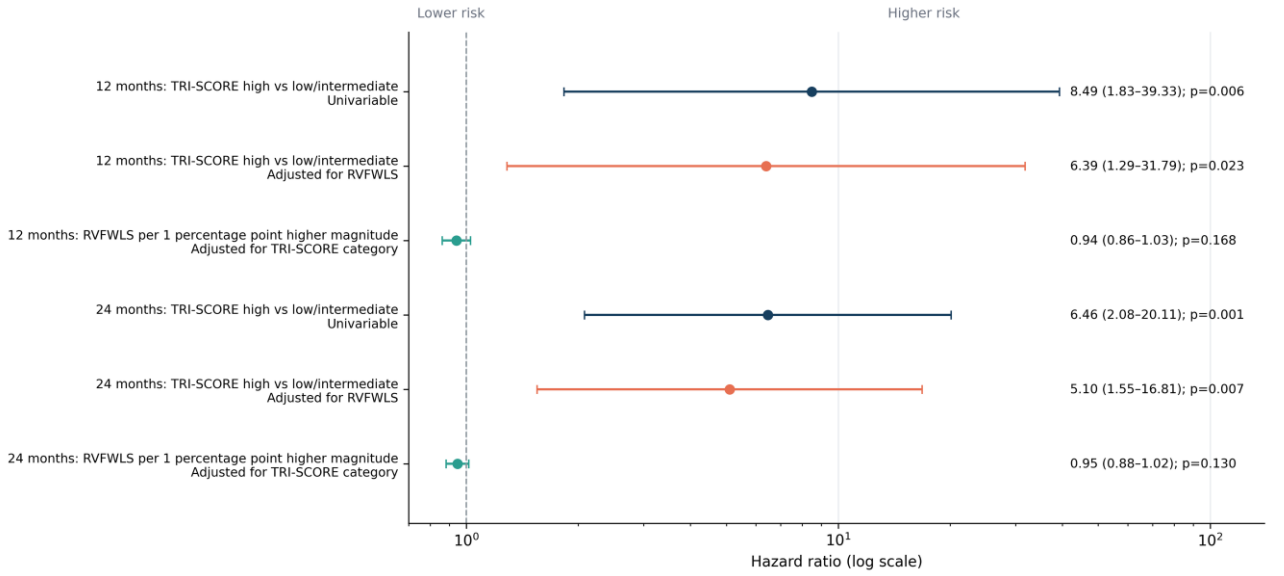


Figure 4. Cox regression associations with all-cause mortality. Hazard ratios are shown for high versus low/intermediate TRI-SCORE category and for each 1 percentage point higher RVFWLS magnitude. Adjusted models include both variables. Dark blue indicates univariable TRI-SCORE estimates, orange indicates TRI-SCORE estimates adjusted for RVFWLS, and teal indicates RVFWLS estimates adjusted for TRI-SCORE. Dots represent hazard ratios, solid horizontal lines indicate 95% confidence intervals, and the vertical dashed line represents the reference value (HR = 1). For deaths recorded as 0 whole months, 0.5 months was assigned. HR, hazard ratio; RVFWLS, right ventricular free-wall longitudinal strain; TRI-SCORE, Tricuspid Regurgitation Impact Score.

4. Discussion

4.1. Principal Findings

In this prospective cohort of patients undergoing TTVI, the TRI-SCORE stratified patients according to 12- and 24-month mortality. Baseline RVFWLS magnitude was lower among non-survivors, reflecting clinically relevant RV vulnerability. Incorporation of RVFWLS produced small, directionally consistent AUC increases at both time points. These gains were not statistically significant, and RVFWLS was not independently associated with mortality in the adjusted models for the overall cohort. An exploratory association was observed at 24 months in the T-TEER subgroup, but this finding requires confirmation. The data therefore support RVFWLS as complementary phenotyping information, but they do not establish incremental predictive utility beyond the TRI-SCORE.

4.2. TRI-SCORE in the Transcatheter Setting

The TRI-SCORE was derived in surgical patients, yet its components reflect the broader biology of advanced TR, including symptoms, high diuretic requirement, renal and hepatic dysfunction, impaired left ventricular function and RV dysfunction [11]. Dreyfus and colleagues subsequently linked the score to expected benefit from intervention in severe TR [12]. Our findings extend this concept to a contemporary TTVI subset with follow-up through 24 months. Mortality rose from 12.1% in the low/intermediate group to 57.1% in the high-risk group by 24 months, illustrating the dominant influence of systemic disease stage even after a transcatheter procedure. Recent T-TEER biomarker data likewise underscore the prognostic relevance of systemic stress, inflammation and multiorgan dysfunction beyond conventional cardiac markers [27].

The strong risk gradient is clinically useful, but should not be interpreted as an automatic futility threshold. Risk scores support Heart Team judgement. Individualized

treatment decisions should therefore remain guided by anatomical suitability, comprehensive clinical assessment and patient goals. Because the high-risk group was small and event counts limited, the HR estimates require external validation. The association between TricValve use and mortality may reflect confounding by indication, as device selection depended on anatomy, RV function and clinical characteristics. It should not be interpreted as a causal device effect. Restricting the analysis to T-TEER recipients likewise showed no statistically significant incremental discrimination from adding RVFWLS, although the small number of events limits this finding. The adjusted association between RVFWLS and 24-month mortality in the T-TEER subgroup suggests a possible prognostic signal, despite the absence of a significant improvement in AUC. Given the exploratory analysis and only nine deaths, this finding requires confirmation.

4.3. RVFWLS May Add Complementary Information

The RV is challenging to quantify because of its crescentic geometry, load dependence and complex longitudinal, radial and anteroposterior contraction [30,31]. TAPSE primarily reflects basal longitudinal motion and may remain deceptively preserved in severe TR because of volume loading. RVFWLS interrogates myocardial deformation across the RV free wall and may reveal impaired contractile reserve earlier than conventional measures [15,16]. Prihadi and colleagues demonstrated prognostic value of RVFWLS in significant functional TR [16], while transcatheter studies have shown that baseline RV function and contraction patterns modify outcome and reverse remodelling [17–22].

In a related retrospective study of 79 patients undergoing isolated tricuspid valve surgery, Ancona et al. found that adding RVFWLS to the TRI-SCORE improved discrimination for 30-day peri-operative mortality (AUC 0.874 vs. 0.787; $p = 0.05$) [32]. This provides important proof-of-concept for combining a clinical risk score with deformation imaging. However, the surgical cohort, peri-operative endpoint and short time horizon differ substantially from our transcatheter population and 24-month outcome. The absence of a statistically significant incremental gain in our study may therefore reflect differences in patient selection and intervention type, as well as limited statistical power.

Our finding of lower RVFWLS magnitude among non-survivors further supports its prognostic relevance. Although the TRI-SCORE includes an indicator of RV dysfunction, this is based on conventional echocardiographic assessment, including TAPSE, which primarily reflects annular excursion and may not fully characterise RV free-wall function. The inverse correlation between RVFWLS magnitude and TRI-SCORE indicates partial overlap between the two measures. Inclusion of RV dysfunction within the score may therefore constrain the additional prognostic information provided by RVFWLS. RVFWLS provides a more detailed assessment of myocardial deformation and may therefore offer complementary information. The modest, non-significant incremental improvement observed in our cohort should be interpreted in the context of the small sample size and limited number of events, rather than as evidence of absent additional value. As RVFWLS is influenced by loading conditions, it should be regarded as an integrated measure of myocardial performance under the prevailing haemodynamic conditions.

4.4. Interpretation of the Combined Model

The combined model increased the AUC from 0.778 to 0.806 at 12 months and from 0.746 to 0.771 at 24 months. The consistency of direction is hypothesis generating, but the small absolute changes and non-significant DeLong tests preclude claims of superior performance. Using the observed standard errors of the paired AUC differences and assuming unchanged variance, a normal approximation yielded detectable ΔAUC values of approximately 0.080 at 12 months and 0.083 at 24 months for 80% power at a two-sided $\alpha = 0.05$. These illustrative estimates indicate limited sensitivity to small incremental gains;

non-significance therefore does not exclude a modest benefit. The combined model is also vulnerable to optimism from in-sample fitting. Internal validation by bootstrap or cross-validation would be unstable at this sample size and was not used to imply generalizability.

Future studies should test a prespecified combined model in larger multicentre cohorts and evaluate whether strain changes management or improves overall clinical benefit. Serial strain assessment after TTVI may be more informative than a single baseline value by distinguishing favourable reverse remodelling from afterload mismatch or progressive RV failure, as suggested by contemporary remodelling studies [20–22]. More broadly, this highlights the potential value of dynamic follow-up parameters rather than isolated baseline measurements. Serial biomarker trajectories may provide complementary information by reflecting changes in congestion and end-organ stress after TTVI [28].

4.5. Clinical Implications

The TRI-SCORE provides a practical framework for mortality risk assessment after TTVI using routinely available clinical, laboratory and conventional echocardiographic information. RVFWLS offers a quantitative assessment of RV myocardial deformation, but requires suitable image acquisition and dedicated strain analysis. Although RVFWLS did not significantly improve discrimination beyond the TRI-SCORE in this cohort, its lower magnitude among non-survivors and its exploratory association with 24-month mortality in the T-TEER subgroup warrant further investigation. These findings do not establish a role for RVFWLS-guided management, but support further evaluation of its prognostic contribution in larger cohorts with independent validation.

4.6. Limitations

This study has several limitations. This single-centre observational study included a small complete-case cohort with a limited number of events. Consequently, estimates can be imprecise and multivariable adjustment was intentionally restricted. Survival times were available at whole-month resolution, which reduced temporal precision and introduced additional ties in the survival analyses. Device heterogeneity and confounding by indication limit interpretation of the pooled estimates. The exploratory T-TEER-only analysis addressed this concern in part, but small device-specific groups and limited events precluded reliable comparisons between individual devices. TR reduction and echocardiographic procedural success were therefore evaluated only in the T-TEER subgroup. No formal intraobserver or interobserver reproducibility analysis was performed. Although all RVFWLS measurements were independently reviewed by a second experienced echocardiographer, this does not quantify measurement agreement. Measurement variability may have attenuated the observed associations and limited the ability to detect incremental prognostic value. Accordingly, the findings cannot distinguish limited incremental value from the influence of measurement variability. Vendor dependence may further limit generalisability.

5. Conclusions

The TRI-SCORE provided robust mortality risk stratification through 24 months after TTVI. In the overall cohort, lower baseline RVFWLS magnitude characterised non-survivors, but the incremental AUC gains and adjusted associations with mortality were not statistically significant. These findings support integrated clinical and echocardiographic phenotyping and justify larger multicentre validation using standardised RVFWLS methods.

Supplementary Materials: The following supporting information can be downloaded at: https://www.mdpi.com/article/10.3390/jcdd13110566/s1, Table S1: Exploratory sensitivity analysis restricted to T-TEER recipients.

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Abbreviations

AUC	area under the receiver operating characteristic curve
BIS-1	Berlin Initiative Study 1
CI	confidence interval
COPD	chronic obstructive pulmonary disease
eGFR	estimated glomerular filtration rate
FAC	fractional area change
HR	hazard ratio
INR	international normalized ratio
IQR	interquartile range
LVEF	left ventricular ejection fraction
MELD-XI	Model for End-Stage Liver Disease excluding INR
mPAP	mean pulmonary artery pressure
NYHA	New York Heart Association
PVR	pulmonary vascular resistance
ROC	receiver operating characteristic
RV	right ventricular
RVEDP	right ventricular end-diastolic pressure
RVFWLS	right ventricular free-wall longitudinal strain
STROBE	Strengthening the Reporting of Observational Studies in Epidemiology
TAPSE	tricuspid annular plane systolic excursion
TR	tricuspid regurgitation
T-TEER	transcatheter tricuspid edge-to-edge repair
TTVI	transcatheter tricuspid valve intervention
WU	Wood unit

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