

Tricuspid Regurgitation Severity and 1-Year Mortality in Echocardiography-Selected Adult Intensive Care Unit Patients: A Medical Information Mart for Intensive Care-IV-Echocardiogram Cohort Study

ABSTRACT

Objective: To evaluate the association between tricuspid regurgitation (TR) severity measured near intensive care unit (ICU) admission and 365-day all-cause mortality in echocardiography-selected adult ICU patients.

Methods: This secondary analysis linked Medical Information Mart for Intensive Care-IV (MIMIC-IV) version 3.1 with MIMIC-IV-echocardiogram. Adults with transthoracic echocardiography from 24 hours before to 72 hours after ICU admission and documented TR severity were included. None/trivial and mild TR were combined as the low-grade reference group; moderate and severe TR were compared with this group. Multiple Cox regression analyses evaluated 28-, 90-, and 365-day mortality. Sodium-adjusted and biomarker-availability analyses evaluated additional laboratory information.

Results: The cohort included 16 991 patients: 12 417 (73.1%) had low-grade, 3467 (20.4%) moderate, and 1107 (6.5%) severe TR. Crude mortality increased across TR severity at 28 days (15.1%, 24.5%, and 32.7%), 90 days (20.3%, 32.5%, and 42.7%), and 365 days (27.2%, 42.9%, and 53.4%). Compared with low-grade TR, 365-day multiple-adjusted estimates were hazard ratio (HR) = 1.32, $P < .001$, 95% CI: 1.24-1.41 for moderate TR and HR = 1.67, $P < .001$, 95% CI: 1.52-1.83 for severe TR; 28- and 90-day estimates were directionally consistent, and sodium-adjusted estimates were similar.

Conclusion: Moderate and severe TR on early echocardiography were associated with higher 1-year mortality in echocardiography-selected adult ICU patients. This observational association remained consistent after sodium adjustment but should not be interpreted as a treatment effect.

Keywords: One-year mortality, echocardiography, intensive care unit, right ventricular function, tricuspid regurgitation

INTRODUCTION

Tricuspid regurgitation (TR) is common in contemporary echocardiography practice, and moderate or greater disease is more frequent in hospitalized and older patients than in the general community.¹ Longitudinal echocardiography cohorts have linked higher TR severity with excess mortality, which supports attention to this finding beyond incidental valve reporting.^{2,3}

Most prognostic evidence for TR has come from ambulatory, valve-disease, heart failure, or procedure-based cohorts.^{4,5} In these settings, TR usually reflects accumulated right-sided remodeling, pulmonary vascular load, atrial disease, or left-sided cardiac disease.

Critical illness may change the meaning of TR because right-sided loading conditions can vary over hours. Positive-pressure ventilation, pulmonary vascular load, volume shifts, vasoactive drugs, and acute right ventricular (RV) dysfunction can increase right-sided afterload or annular dilation.^{6,7} Acute RV failure statements

ORIGINAL INVESTIGATION

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also emphasize the interaction between venous congestion, organ dysfunction, and right-sided reserve.⁸

Intensive care unit (ICU)—specific evidence remains limited. In sepsis, RV dysfunction has been associated with short- and long-term mortality, while impaired RV—pulmonary artery coupling has been associated with hospital and 1-year mortality in cardiac ICU cohorts.^{9,10} Most studies have emphasized acute physiology or selected critical-care populations.

Linked critical-care and echocardiography data allow routine TR grading to be studied at scale with explicit cohort flow and mortality follow-up.^{11–14} This study evaluated whether moderate and severe TR, compared with a harmonized low-grade reference group, were associated with 365-day mortality in echocardiography-selected adult ICU patients and examined measurement availability and exploratory subgroup findings.

METHODS

Study Design and Data Source

This secondary analysis used a retrospective cohort derived from Medical Information Mart for Intensive Care IV (MIMIC-IV) version 3.1 linked to the MIMIC-IV-echocardiogram (ECHO) module. Medical Information Mart for Intensive Care-IV contains deidentified electronic health record data from critically ill patients treated at Beth Israel Deaconess Medical Center, and the linked echocardiography module provides structured and report-derived echocardiographic information for eligible patients.^{11–13} Access to the data was restricted to credentialed users through PhysioNet under a Data Use Agreement.¹⁴

The study estimated prognostic associations rather than causal effects. Intensive care unit admission was the time origin, and the exposure was interpreted as an early echocardiography-selected ICU phenotype. Reporting followed Strengthening the Reporting of Observational Studies in Epidemiology and REporting of studies Conducted using Observational Routinely-collected health Data recommendations.^{15,16}

Ethical Considerations

This study used deidentified data from MIMIC-IV version 3.1 and the linked MIMIC-IV-ECHO module. The source MIMIC-IV data-sharing initiative was approved by the institutional review boards of the Massachusetts Institute of Technology

and Beth Israel Deaconess Medical Center, with waiver of informed consent for secondary use of deidentified data. Database access was limited to credentialed users who completed the required training and accepted the PhysioNet Data Use Agreement. Additional departmental academic board approval was obtained for this secondary analysis.

Study Population

Adults aged 18 years or older were eligible if they had a transthoracic ECHO with documented TR assessment from 24 hours before to 72 hours after ICU admission. Only the first ICU stay per patient was retained; when multiple studies were available, the study closest to ICU admission was selected. Patients without a usable routine-report TR label were excluded.

Follow-up began at ICU admission. Because the qualifying ECHO could occur up to 72 hours later, the estimates apply to patients selected for early echocardiography and do not represent all ICU admissions at time zero.

Exposure Definition

The selected routine echocardiography report supplied the TR label. Physiologic and trivial labels were mapped to none/trivial; mild [1+] to mild; mild-mod [1-2+] and mod [2+] to moderate; and mod-severe [3+] and severe [4+] to severe. No image re-reading was performed. Vena contracta width, reader identity, and reproducible massive/torrential categories were not present as structured cohort variables. Because none/trivial and mild grades may be difficult to distinguish under rapidly changing ICU loading conditions, these categories were combined into a low-grade reference group.¹⁷

Categorical models compared moderate and severe TR with low-grade TR. Ordinal 4-grade TR was used only for exploratory subgroup analyses.

Outcomes

The primary outcome was 365-day all-cause mortality after ICU admission. Secondary outcomes were 90-day and 28-day all-cause mortality. Mortality was derived from the date-of-death field in the MIMIC-IV v3.1 patients table, which integrates hospital and state death records and provides follow-up through 1 year after the last hospital discharge. Full-cohort event counts and complete-case model event counts are reported separately.

Kaplan–Meier curves described unadjusted 365-day and 28-day survival for low-grade, moderate, and severe TR; the log-rank test compared curves. Mortality status was treated as ascertainable through 365 days for the cohort; therefore, without censoring before day 365, 1-Kaplan–Meier survival at 365 days equals the crude 365-day mortality proportion. The cohort flow, survival curves, adjusted models, and Kaplan–Meier-derived 1-year mortality are presented in Figure 1, Figure 2, Figure 3, and Figure 4, respectively.

Covariates

Clinical covariates were drawn from the early ICU period. The fully adjusted model included age, sex, Sequential Organ Failure Assessment (SOFA) score, Charlson Comorbidity

HIGHLIGHTS

- In an echocardiography-selected adult intensive care unit cohort, moderate and severe tricuspid regurgitation (TR) was associated with higher 365-day mortality.
- Combining none/trivial and mild TR into a low-grade reference group did not attenuate the association.
- Sodium-adjusted and selectively measured troponin analyses showed a consistent direction of association.
- Biomarker and right-ventricular measurements were reported according to actual availability and were not treated as complete-cohort covariates.

Patient selection and TR exposure groups

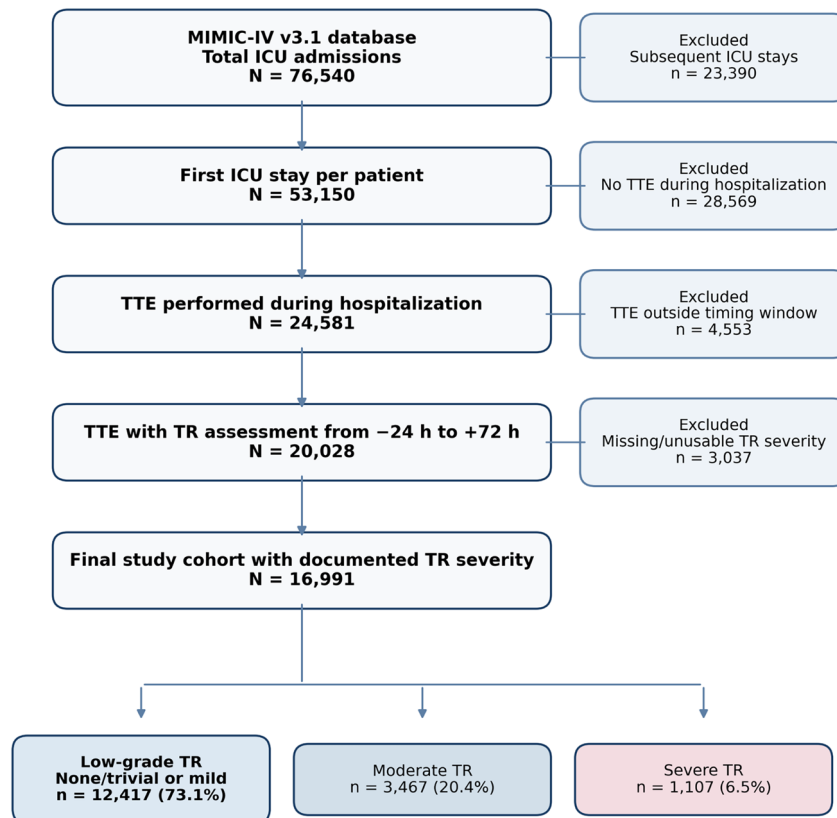


Figure 1. Patient selection and TR exposure groups. Final cohort: 16 991 echocardiography-selected adult ICU patients; none/trivial and mild TR were combined as low-grade TR. ICU, intensive care unit; MIMIC-IV, Medical Information Mart for Intensive Care-IV; TR, tricuspid regurgitation; TTE, transthoracic echocardiography.

Index, creatinine, hemoglobin, white blood cell count, mean arterial pressure, vasopressor use, invasive mechanical ventilation, congestive heart failure (CHF), chronic kidney disease (CKD), chronic pulmonary disease, and malignancy.¹⁸ Sodium was added in a sensitivity model rather than replacing the primary model.

Echocardiographic variables were routine structured or report-derived fields. Right ventricular function was a qualitative report label (normal/low-normal, mild/moderate/severe global hypokinesis, depressed, regional abnormalities, hyperdynamic, not well seen, or not assessable), not a composite measurement. Right ventricular chamber size was separately recorded. Tricuspid annular plane systolic excursion (TAPSE) was stored in centimeters and was available in 3494 patients (20.6%). Tissue Doppler S', RV fractional area change, and RV strain were not available as reproducible structured cohort variables. Inferior vena cava (IVC) diameter and report-level collapse descriptions were analyzed separately. Tricuspid annular plane systolic excursion availability and variable-level missingness are reported in Supplementary Table 1 and Supplementary Table 2.

The same covariate set was retained across primary and sensitivity models; it was selected according to clinical

relevance and expected mortality association rather than stepwise or outcome-driven procedures.

Missing Data and Data Quality Checks

Data validation confirmed the cohort flow, 1-record-per-patient structure, TR mapping, exposure-group counts, outcome counts, and model-specific complete-case counts. The analysis dataset contained 16 991 patients; the 365-day primary model contained 16 842 complete cases and 5412 events, whereas the full cohort contained 5459 deaths by 365 days.

Primary-model covariate missingness was low and complete-case analysis was used. Sodium was available in 16 884 patients (99.4%). Tricuspid annular plane systolic excursion was available in 20.6%, TR pressure gradient in 78.4%, and left ventricular ejection fraction (LVEF) in 81.0%. Biomarker measurement was selective: troponin T was available at any time during admission in 7805 patients (45.9%), N-terminal pro-B-type natriuretic peptide (NT-proBNP) in 2195 (12.9%), and D-dimer in 592 (3.5%).

Tricuspid annular plane systolic excursion was not included in the primary model because it was missing in 79.4% and is load dependent. Tricuspid annular plane systolic excursion

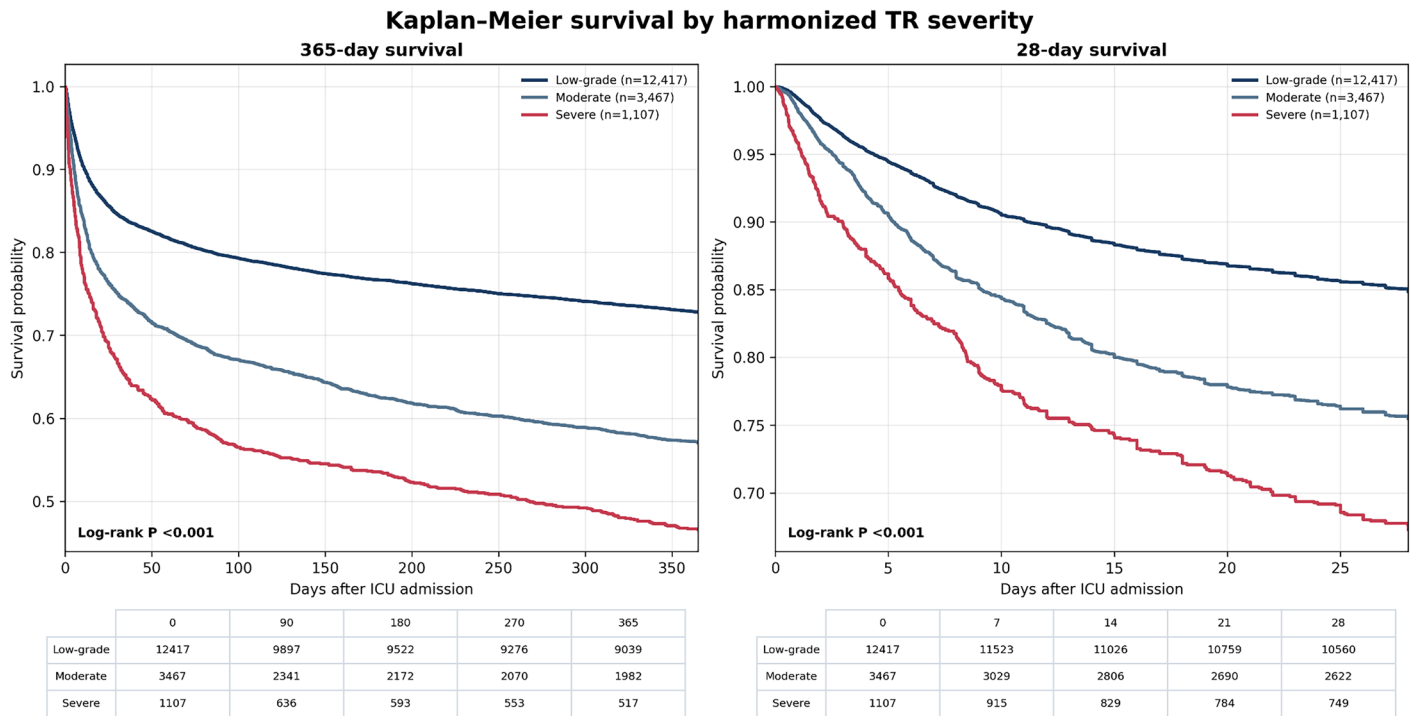


Figure 2. Unadjusted Kaplan–Meier survival by harmonized TR severity. Panels show 365-day and 28-day survival with numbers at risk; log-rank $P < .001$ for both horizons. ICU, intensive care unit; TR, tricuspid regurgitation.

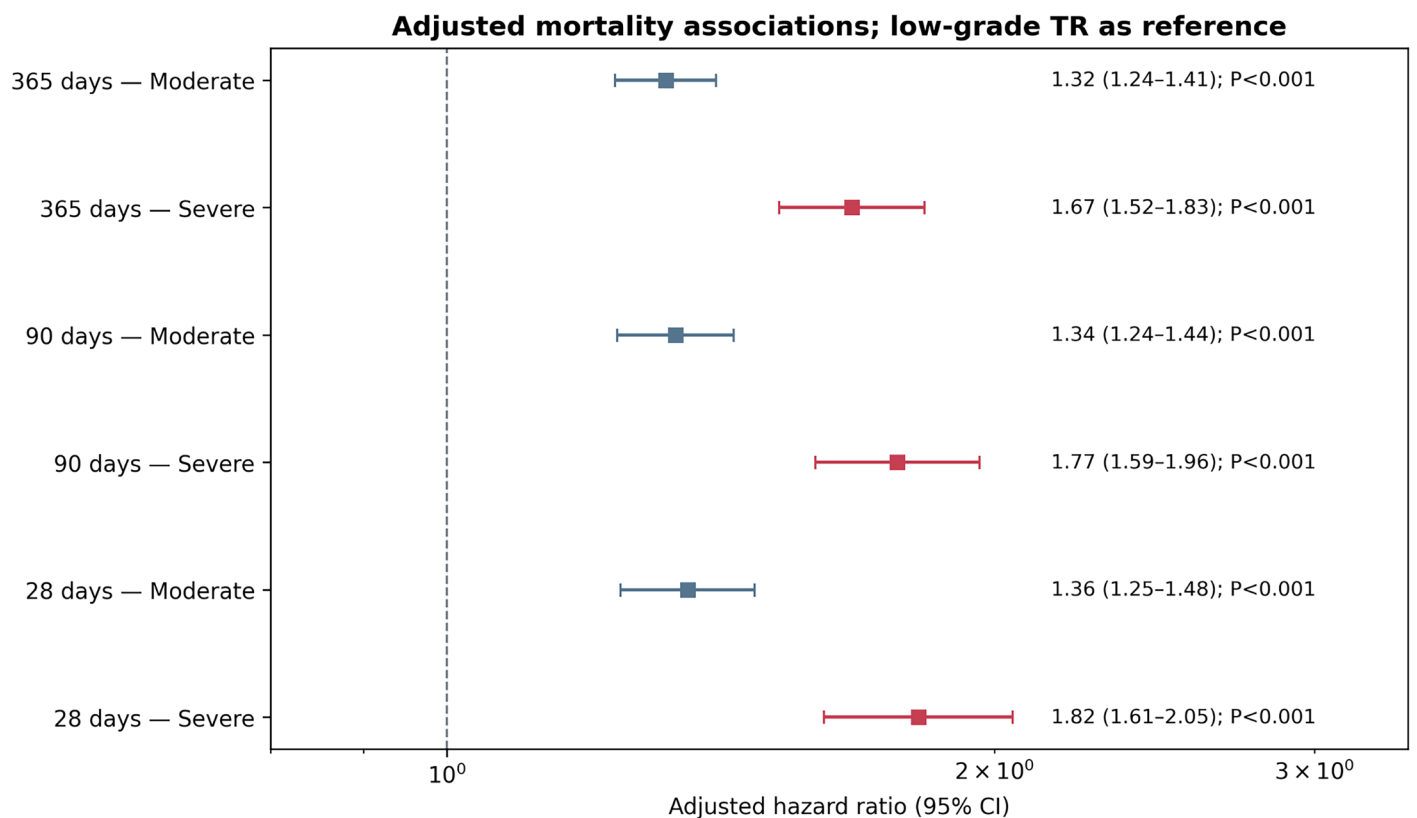


Figure 3. Multiple-adjusted mortality associations. HRs and 95% CIs compare moderate and severe TR with low-grade TR at 28, 90, and 365 days. HR, hazard ratio; TR, tricuspid regurgitation.

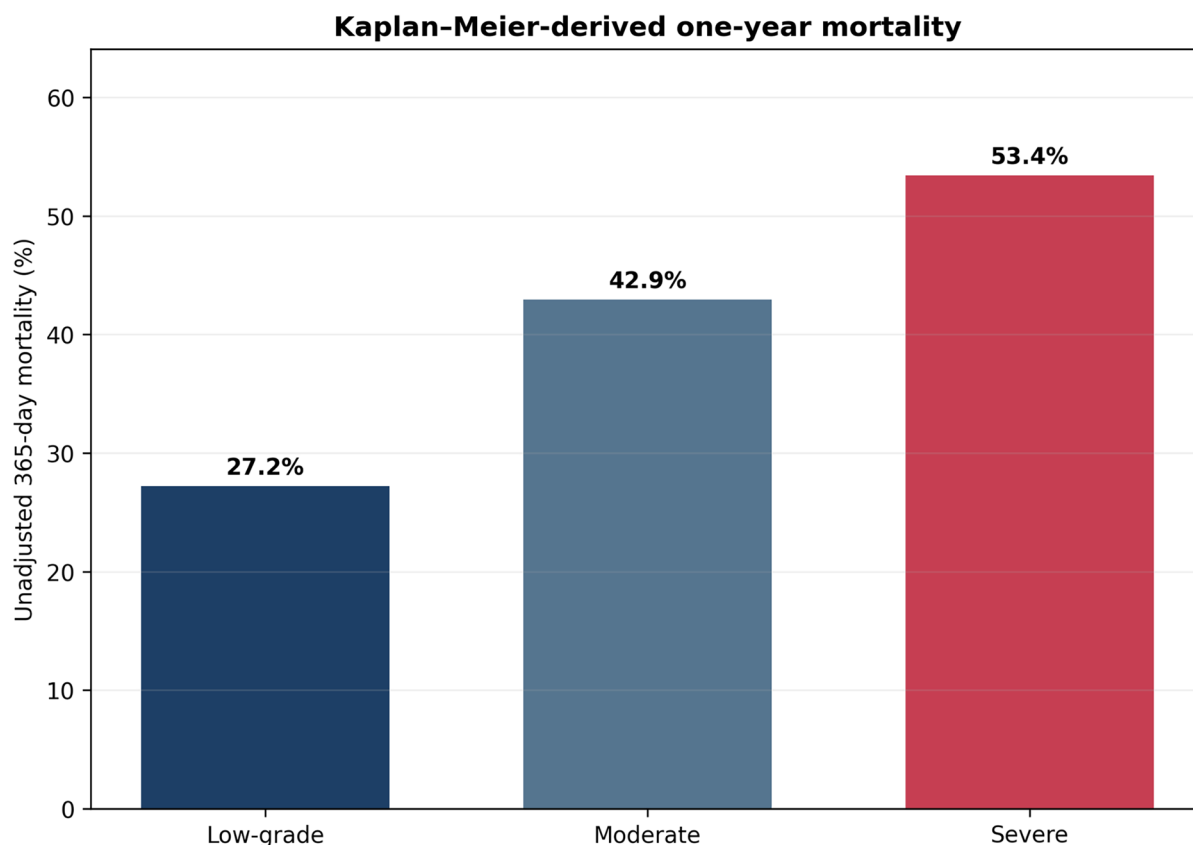


Figure 4. Unadjusted Kaplan–Meier-derived 365-day mortality; values are not model-adjusted probabilities.

availability was evaluated descriptively (Supplementary Table 1). Troponin T was evaluated only in a measured-subcohort sensitivity analysis; NT-proBNP and D-dimer coverage was insufficient for stable full-cohort adjustment, and their selective ordering could introduce collider and selection bias. Missing biomarker values were not imputed. Missingness and measurement availability are reported with explicit denominators in the supplementary material.

Statistical Analysis

Continuous variables were summarized as mean $\pm$ SD or median (interquartile range [IQR]), and categorical variables as n (%). Across the 3 TR groups, continuous and categorical comparisons used Kruskal–Wallis and chi-square tests, respectively.

Multiple Cox regression analyses estimated adjusted associations for 28-, 90-, and 365-day mortality. Nineteen low-grade TR was the reference. Hazard ratios (HRs), 95% CIs, *P* values, model N, and events were recorded for each model.

Term-specific scaled Schoenfeld residual tests assessed proportional hazards (PH).¹⁹ The severe-TR term and several adjustment covariates showed PH deviations in the 365-day model; the reported HRs were therefore interpreted as average associations over follow-up, with shorter-horizon models used to assess temporal consistency.

Exploratory 28-day subgroup analyses evaluated ordinal TR across age (<65/ $\geq$ 65 years), sex, SOFA (<6/ $\geq$ 6), CHF,

CKD, vasopressor use, and invasive mechanical ventilation. Multiplicative interaction terms generated interaction *P* values. No multiplicity correction was applied (Supplementary Table 3).

Additional exploratory 365-day analyses were stratified by sex and age to compare moderate and severe TR with low-grade TR. Sex-stratified models omitted sex from the adjustment set. Age strata used non-overlapping categories <30, 30–49, 50–69, and $\geq$ 70 years; continuous age was retained within age strata as residual adjustment. Primary and sodium-adjusted versions were fitted. If a sparse TR-specific event cell precluded reliable joint estimation within an age stratum, coefficients from the entire stratum-specific model were withheld rather than selectively reporting terms from that model.

Additional sensitivity models added sodium to the primary Cox adjustment set at each 28-, 90-, and 365-day mortality horizon and added log-transformed troponin T to the 365-day model in the subgroup with a numeric value from 24 hours before to 24 hours after ICU admission. Biomarker availability is detailed in Supplementary Table 4. Intensive care unit admission causes were grouped from principal discharge diagnosis codes (Supplementary Table 5). Among patients with CHF codes, early-transthoracic echocardiography (TTE) LVEF was described as $\leq$ 40%, 41%–49%, or $\geq$ 50%; these are echocardiographic EF categories and not clinically adjudicated heart failure with reduced ejection fraction/heart

failure with mildly reduced ejection fraction/heart failure with preserved ejection fraction diagnoses (Supplementary Table 6). Inferior vena cava—description availability and full-cohort mortality counts are reported in Supplementary Table 7 and Supplementary Table 8.

Data extraction used Structured Query Language (SQL) in Google BigQuery. Statistical analyses were performed in Python using pandas, SciPy, and lifelines 0.30.1; figures were generated with Matplotlib. Tests were 2-sided, $P < .05$ was considered nominally significant, and $P < .001$ was reported for smaller values.

RESULTS

Study Population and Baseline Characteristics

The study cohort contained 16 991 patients (Figure 1): 12 417 (73.1%) had low-grade TR, 3467 (20.4%) moderate TR, and 1107 (6.5%) severe TR. The low-grade group comprised 7661 none/trivial and 4756 mild labels. Mean age was 67.7 ± 16.3 years; 9249 patients (54.4%) were male. Tricuspid annular plane systolic excursion was available in 3494 (20.6%), LVEF in 13 768 (81.0%), and TR pressure gradient in 13 317 (78.4%). Median time from ICU admission to echocardiography was 17.0 hours (IQR, 9.0–33.0).

Baseline characteristics are shown in Table 1. Mean sodium was 136.6 ± 5.1 mEq/L in the low-grade group, 136.3 ± 5.5 mEq/L in moderate TR, and 135.7 ± 5.8 mEq/L in severe TR ($P < .001$). Higher TR grade was also accompanied by older age, more CHF and CKD, lower TAPSE, higher TR pressure gradient, and larger IVC diameter.

Mortality at 28, 90, and 365 Days

Crude mortality increased stepwise across low-grade, moderate, and severe TR at each sequential horizon: 1880/12 417 (15.1%), 850/3467 (24.5%), and 362/1107 (32.7%) at 28 days; 2523/12 417 (20.3%), 1128/3467 (32.5%), and 473/1107 (42.7%) at 90 days; and 3380/12 417 (27.2%), 1488/3467 (42.9%), and 591/1107 (53.4%) at 365 days, respectively (Table 2). Overall, 5459 patients (32.1%) died within 365 days.

In the primary complete-case multiple Cox models ($n=16\,842$), moderate and severe TR were associated with higher mortality at 28 days (moderate: HR=1.36, $P < .001$, 95% CI: 1.25–1.48; severe: HR=1.82, $P < .001$, 95% CI: 1.61–2.05), 90 days (moderate: HR=1.34, $P < .001$, 95% CI: 1.24–1.44; severe: HR=1.77, $P < .001$, 95% CI: 1.59–1.96), and 365 days (moderate: HR=1.32, $P < .001$, 95% CI: 1.24–1.41; severe: HR=1.67, $P < .001$, 95% CI: 1.52–1.83) (Table 2 and Figure 3). Model event counts were 3070, 4090, and 5412 at 28, 90, and 365 days, respectively.

Sodium adjustment produced nearly identical estimates at 28 days (moderate: HR=1.36, $P < .001$, 95% CI: 1.24–1.48; severe: HR=1.81, $P < .001$, 95% CI: 1.61–2.04), 90 days (moderate: HR=1.33, $P < .001$, 95% CI: 1.24–1.44; severe: HR=1.76, $P < .001$, 95% CI: 1.59–1.96), and 365 days (moderate: HR=1.32, $P < .001$, 95% CI: 1.23–1.40; severe: HR=1.66, $P < .001$, 95% CI: 1.52–1.82) (Table 2). The sodium-adjusted models included 16 824 patients, with 3068, 4088, and 5409 events at 28, 90, and 365 days, respectively. In the selectively measured

troponin T subgroup ($N=4793$; 1874 events), estimates remained directionally consistent (Table 3).

Kaplan–Meier Survival Analysis

Unadjusted Kaplan–Meier curves separated across low-grade, moderate, and severe TR through 365 and 28 days (both log-rank $P < .001$; Figure 2). Kaplan–Meier-derived 365-day mortality was 27.2%, 42.9%, and 53.4%, respectively (Figure 4).

Exploratory Subgroup Analyses

The ordinal TR association was positive in every exploratory 28-day stratum (Supplementary Figure 1 and Supplementary Table 3). For age <65 and ≥ 65 years, HRs per grade were 1.18 and 1.23 (interaction $P < .001$). For no vasopressor versus vasopressor use, HRs were 1.32 and 1.17 (interaction $P < .001$). For no mechanical ventilation versus ventilation, HRs were 1.35 and 1.15 (interaction $P < .001$). Nominal interactions were also observed for SOFA category ($P=.013$), CHF ($P=.027$), and CKD ($P=.047$); sex was not an effect modifier ($P=.546$). Because no multiplicity correction was applied, these findings are exploratory.

At 365 days, the associations were directionally consistent in women and men (Supplementary Table 9). Among women, moderate TR was associated with HR=1.28, $P < .001$, 95% CI: 1.17–1.40 and severe TR with HR=1.62, $P < .001$, 95% CI: 1.43–1.84; among men, corresponding estimates were HR=1.36, $P < .001$, 95% CI: 1.25–1.49 and HR=1.74, $P < .001$, 95% CI: 1.52–2.00. Sodium-adjusted estimates were nearly unchanged. In age-stratified analyses, adjusted Cox estimates were not reported for patients aged < 30 years because the moderate- and severe-TR cells were sparse. In the 30–49-year stratum, the severe-TR cell was sparse (51 patients/16 deaths); because moderate and severe TR were estimated jointly, the stratum-specific model was withheld as a whole, and the moderate-TR coefficient was not reported separately despite 53 deaths among 191 patients with moderate TR. For ages 50–69 years, adjusted estimates were HR=1.41, $P < .001$, 95% CI: 1.24–1.60 for moderate TR and HR=1.55, $P < .001$, 95% CI: 1.25–1.92 for severe TR; for ages ≥ 70 years, the corresponding estimates were HR=1.27, $P < .001$, 95% CI: 1.17–1.37 and HR=1.66, $P < .001$, 95% CI: 1.50–1.85 (Supplementary Table 10). Sodium-adjusted estimates were similar.

Supportive and Sensitivity Analyses

Sodium-adjusted estimates are presented alongside the primary models in Table 2. The troponin-measured subgroup 365-day sensitivity analysis is summarized in Table 3; its direction was consistent with the primary association, while the troponin result remained subject to measurement-selection bias.

Term-specific Schoenfeld testing identified a PH signal for severe TR ($P=.004$) and several adjustment covariates. The 90-day and 28-day models showed the same direction of association; 1-year HRs were interpreted as average associations over follow-up.

Principal-diagnosis categories were cardiovascular (37.1%), neurologic/cerebrovascular (13.9%), sepsis/infection (13.7%),

Table 1. Baseline Characteristics Stratified by Harmonized Tricuspid Regurgitation Severity

Variables	Overall (n = 16 991)	Low-Grade (n = 12417)	Moderate (n = 3467)	Severe (n = 1107)	P
Demographics					
Age, years	67.7 ± 16.3	65.2 ± 16.2	74.1 ± 14.5	76.1 ± 14.2	<.001
Male sex, n (%)	9249 (54.4)	7215 (58.1)	1580 (45.6)	454 (41.0)	<.001
White race, n (%)	10 189 (60.0)	7394 (59.5)	2140 (61.7)	655 (59.2)	.059
Physiologic measurements					
Heart rate, bpm	84.7 ± 17.3	84.4 ± 17.0	85.1 ± 17.8	86.5 ± 18.0	<.001
MAP, mm Hg	79.7 ± 11.8	80.6 ± 11.9	77.5 ± 11.2	76.1 ± 10.8	<.001
SpO ₂ , %	96.7 (95.3-98.0)	96.7 (95.3-98.0)	96.6 (95.3-98.0)	96.6 (95.0-97.8)	.007
Laboratory values					
WBC, 10 ³ /uL	12.1 (8.8-16.7)	12.1 (8.8-16.7)	12.1 (8.8-16.8)	11.8 (8.6-16.7)	.383
Hemoglobin, g/dL	10.5 ± 2.4	10.7 ± 2.4	10.1 ± 2.2	9.9 ± 2.3	<.001
Creatinine, mg/dL	1.1 (0.8-1.8)	1.1 (0.8-1.7)	1.3 (0.9-2.1)	1.5 (1.0-2.5)	<.001
BUN, mg/dL	23 (16-38)	21 (15-34)	29 (19-46)	34 (22-55)	<.001
Sodium, mEq/L	136.5 ± 5.2	136.6 ± 5.1	136.3 ± 5.5	135.7 ± 5.8	<.001
Bicarbonate, mEq/L	21.2 ± 5.0	21.3 ± 4.9	21.1 ± 5.3	20.9 ± 5.7	<.001
Severity scores					
SOFA score	4.0 (2.0-7.0)	4.0 (2.0-6.0)	5.0 (2.0-7.5)	5.0 (3.0-8.0)	<.001
APS III	42.0 (31.0-56.0)	40.0 (29.0-54.0)	47.0 (36.0-61.0)	49.0 (38.0-64.0)	<.001
CCI	5.5 ± 3.0	5.1 ± 3.0	6.4 ± 2.8	6.8 ± 2.6	<.001
Comorbidities					
CHF, n (%)	6865 (40.4)	4067 (32.8)	1970 (56.8)	828 (74.8)	<.001
CKD, n (%)	4149 (24.4)	2600 (20.9)	1111 (32.0)	438 (39.6)	<.001
COPD, n (%)	4464 (26.3)	2919 (23.5)	1160 (33.5)	385 (34.8)	<.001
Diabetes, n (%)	5368 (31.6)	3936 (31.7)	1095 (31.6)	337 (30.4)	.690
Prior MI, n (%)	4649 (27.4)	3401 (27.4)	951 (27.4)	297 (26.8)	.918
Malignancy, n (%)	2059 (12.1)	1497 (12.1)	441 (12.7)	121 (10.9)	.261
ICU treatments					
Vasopressor, n (%)	5727 (33.7)	3863 (31.1)	1367 (39.4)	497 (44.9)	<.001
Mechanical ventilation, n (%)	6360 (37.4)	4680 (37.7)	1323 (38.2)	357 (32.2)	<.001
RRT, n (%)	942 (5.5)	643 (5.2)	212 (6.1)	87 (7.9)	<.001
Echocardiographic parameters					
LVEF, %	52.4 ± 15.6	53.7 ± 14.5	49.3 ± 17.3	47.0 ± 18.3	<.001
TAPSE, cm	1.97 ± 0.54	2.06 ± 0.51	1.78 ± 0.55	1.50 ± 0.54	<.001
TR gradient, mm Hg	31 (24-39)	27 (22-34)	38 (31-46)	43 (34-55)	<.001
E/e' ratio	10.0 (8.0-13.0)	9.0 (8.0-13.0)	12.0 (9.0-16.0)	12.0 (8.2-16.0)	<.001
LA volume, mL	58 (42-79)	54 (40-72)	70 (50-95)	79 (58-99)	<.001
IVC diameter, cm	2.0 ± 0.5	1.9 ± 0.5	2.1 ± 0.5	2.4 ± 0.5	<.001

Continuous variables are mean ± SD or median (IQR); categorical variables are n (%). Low-grade TR combines none/trivial and mild routine-report labels. P values compare low-grade, moderate, and severe groups. Variable-specific denominators apply to echocardiographic measures; missingness is reported in Supplementary Table 2.

APS III, Acute Physiology Score III; BUN, blood urea nitrogen; CCI, Charlson Comorbidity Index; CHF, congestive heart failure; CKD, chronic kidney disease; COPD, chronic obstructive pulmonary disease; ICU, intensive care unit; IQR, interquartile range; IVC, inferior vena cava; LA, left atrial; LVEF, left ventricular ejection fraction; MAP, mean arterial pressure; MI, myocardial infarction; RRT, renal replacement therapy; SOFA, Sequential Organ Failure Assessment; SpO₂, peripheral oxygen saturation; TAPSE, tricuspid annular plane systolic excursion; TR, tricuspid regurgitation; WBC, white blood cell count.

trauma/injury/poisoning (9.8%), respiratory (7.0%), and other categories. Among 6865 patients with CHF codes, early-TTE LVEF was ≤40% in 2676 (39.0%), 41%-49% in 373 (5.4%), ≥50% in 2614 (38.1%), and unavailable in 1202 (17.5%). Report-level IVC collapse information was absent in 76.6% overall and was not used as a complete-cohort adjustment variable.

DISCUSSION

Two principal findings emerged. First, moderate and severe TR were associated with higher 365-day mortality compared with a combined low-grade reference group. Second, the associations persisted at 90 and 28 days and after sodium adjustment.

Table 2. Mortality According to Tricuspid Regurgitation Severity at Sequential Follow-Up Intervals

Endpoint	TR Severity	Crude Mortality, n/N (%)	Multiple-Adjusted HR	Sodium-Adjusted HR
28 days	Low-grade	1880/12 417 (15.1)	Reference	Reference
28 days	Moderate	850/3467 (24.5)	HR=1.36 $P < .001$ 95% CI: 1.25-1.48	HR=1.36 $P < .001$ 95% CI: 1.24-1.48
28 days	Severe	362/1107 (32.7)	HR=1.82 $P < .001$ 95% CI: 1.61-2.05	HR=1.81 $P < .001$ 95% CI: 1.61-2.04
90 days	Low-grade	2523/12 417 (20.3)	Reference	Reference
90 days	Moderate	1128/3467 (32.5)	HR=1.34 $P < .001$ 95% CI: 1.24-1.44	HR=1.33 $P < .001$ 95% CI: 1.24-1.44
90 days	Severe	473/1107 (42.7)	HR=1.77 $P < .001$ 95% CI: 1.59-1.96	HR=1.76 $P < .001$ 95% CI: 1.59-1.96
365 days	Low-grade	3380/12417 (27.2)	Reference	Reference
365 days	Moderate	1488/3467 (42.9)	HR=1.32 $P < .001$ 95% CI: 1.24-1.41	HR=1.32 $P < .001$ 95% CI: 1.23-1.40
365 days	Severe	591/1107 (53.4)	HR=1.67 $P < .001$ 95% CI: 1.52-1.83	HR=1.66 $P < .001$ 95% CI: 1.52-1.82

Crude mortality uses the full cohort. Low-grade TR is the reference for Cox models. Multiple-adjusted models included age, sex, SOFA score, CCI, creatinine, hemoglobin, white blood cell count, mean arterial pressure, vasopressor use, mechanical ventilation, CHF, CKD, chronic pulmonary disease, and malignancy. Primary model N/events were 16 842/3070 at 28 days, 16 842/4090 at 90 days, and 16 842/5412 at 365 days. Sodium-adjusted models added sodium; N/events were 16 824/3068, 16 824/4088, and 16 824/5409, respectively. HR, hazard ratio; TR, tricuspid regurgitation.

The findings align with non-ICU TR literature but extend the population context. Nath et al analyzed 5223 echocardiography patients and reported stepwise lower 1-year survival with increasing TR severity. Topilsky et al^{2,3} studied 353 patients with isolated TR and reported adverse long-term outcomes despite preserved LVEF and right ventricular systolic pressure below 50 mm Hg. Wang et al⁴ described contemporary etiologies and outcomes in patients with isolated TR, and Zahr et al⁵ found higher mortality with severe baseline and residual TR after transcatheter aortic valve replacement. The present ICU cohort is concordant in direction, but it anchors TR severity to an early critical-care echocardiography window and 1-year mortality after ICU admission.

In this cohort, higher TR severity accompanied larger IVC diameter, lower TAPSE, and higher illness severity. These associations support the interpretation of TR as a marker within a broader right-sided congestion and cardiopulmonary reserve phenotype.^{20,21} However, the study did not include standardized volume-status measurements, central image re-reading, or complete IVC collapsibility data, and it cannot separate chronic structural TR from transient load-dependent ICU changes.

Combining none/trivial and mild TR addresses the limited reproducibility of visually distinguishing low-grade jets, especially when jets are eccentric or loading conditions change rapidly. This grouping avoids assigning prognostic

meaning to a potentially unstable boundary and shows that risk separation is concentrated at moderate and severe grades.

Tricuspid annular plane systolic excursion reflects longitudinal RV shortening but is angle dependent, load dependent, and influenced by overall heart motion; it does not represent RV systolic performance alone. Its 20.6% availability also creates measurement-selection concerns. Qualitative RV function was available more often but derived from routine report labels. Tissue Doppler S', RV fractional area change, and RV strain could not be incorporated because reproducible structured values were unavailable.

Troponin T, NT-proBNP, and D-dimer were not suitable as complete-cohort covariates because any-admission availability was 45.9%, 12.9%, and 3.5%, respectively, and ordering was clinically selective. The troponin-measured subgroup result was directionally consistent but does not eliminate selection bias. Similarly, early-TTE EF categories among patients with CHF codes should not be interpreted as adjudicated heart-failure phenotypes.

Exploratory interaction estimates were strongest in patients without vasopressors or invasive ventilation. One interpretation is that when acute organ-support requirements dominate short-term mortality, ordinal TR adds less relative prognostic information. Nominal SOFA and CHF interactions and the absence of a sex interaction underscore that

Table 3. Additional 365-Day Biomarker Sensitivity Analysis

Analysis (N; Events)	Moderate TR	Severe TR
Troponin-T measured subgroup (N=4793; 1874)	HR=1.27 $P < .001$ 95% CI: 1.14-1.41	HR=1.62 $P < .001$ 95% CI: 1.39-1.88

The troponin-T model is restricted to selectively measured patients and adds log-transformed troponin T to the primary adjustment set. Model-specific N represents the intersection of biomarker availability with complete primary-model covariates. Low-grade TR is the reference. HR, hazard ratio; TR, tricuspid regurgitation.

the subgroup pattern is not reducible to 1 clinical dimension; these analyses require external confirmation. The additional sex-stratified estimates were similar in direction, whereas age-stratified estimates in the youngest categories were limited by sparse events; these exploratory findings should not be overinterpreted.

The results support TR as a prognostic marker within an echocardiography-selected ICU population, not as evidence that treating TR during critical illness improves outcome. Clinical use should remain contextual and should integrate loading conditions, RV assessment, ventilation, and the reason for ICU admission.

Study Limitations

This retrospective single-health-system study used routine clinical echocardiography reports without central image re-reading. Reader identity, reader count, and intraobserver/interobserver reproducibility were unavailable. Tricuspid regurgitation was semi-quantitatively reported; vena contracta and reproducible massive/torrential categories were not available, so a 5-grade scheme could not be reconstructed from the available data. Misclassification is therefore possible, particularly at low grades.

The qualifying ECHO could occur up to 72 hours after ICU admission, creating survivor and indication selection. Results therefore apply to patients selected for early echocardiography and not to all ICU admissions. Tricuspid annular plane systolic excursion was available in 20.6%; IVC collapse description in 23.4%; LVEF in 81.0%. Mechanical ventilation further limits interpretation of IVC respiratory variation.

Complete-case analysis excluded 149 patients from the primary model. Biomarker measurements were selective, and NT-proBNP and D-dimer coverage was too low for full-cohort adjustment. Residual confounding from volume status, pulmonary vascular load, acute RV afterload, treatment decisions, and unmeasured disease severity remains possible.

The severe-TR term and several adjustment covariates showed PH deviations; the 365-day HRs should therefore be interpreted as average associations over follow-up. Subgroup analyses were exploratory, had no multiplicity correction, and should not be used for confirmatory treatment targeting. In the 365-day age-stratified analyses, sparse TR-specific events in patients aged <30 years and the sparse severe-TR cell in patients aged 30–49 years prevented reliable joint stratum-specific Cox estimation; coefficients from those age-stratum models were therefore not reported.

CONCLUSION

Moderate and severe TR on early echocardiography were associated with higher 365-day mortality in echocardiography-selected adult ICU patients compared with low-grade TR. The association persisted at shorter horizons and after sodium adjustment. External validation with standardized image acquisition, quantitative TR/RV measures, reader reproducibility, and prospectively defined subgroup testing is required.

Ethics Committee Approval: This study analyzed de-identified data from the MIMIC-IV database. The institutional review boards of the Massachusetts Institute of Technology and Beth Israel Deaconess Medical Center approved the original data collection and waived the requirement for individual informed consent. Additionally, the Academic Board of the Department of Cardiology approved this secondary-data analysis (Meeting No. 5, Decision No. 1, March 31, 2026); therefore, additional local ethics committee approval was not required.

Informed Consent: Informed consent was waived because this study involved a retrospective secondary analysis of de-identified data.

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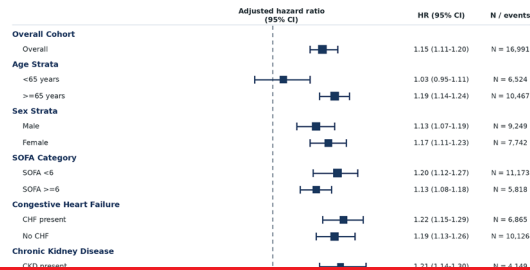
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SUPPLEMENTARY MATERIAL

Supplementary Figure S1. TR Severity and 28-Day Mortality

Subgroup estimates for the acute 28-day mortality horizon.



Supplementary Figure 1. Exploratory 28-day subgroup analyses. Adjusted HRs and 95% CIs are shown per one-grade increase in ordinal TR, with interaction P values. No multiplicity correction was applied. CHF, congestive heart failure; CI, confidence interval; CKD, chronic kidney disease; HR, hazard ratio; SOFA, Sequential Organ Failure Assessment; TR, tricuspid regurgitation.

Supplementary Table 1. Comparison of patients with and without TAPSE data

Variables	TAPSE Available (n = 3,494)	TAPSE Missing (n = 13,497)	P
Age, years	67.0 ± 16.1	67.9 ± 16.3	0.003
SOFA score	4 (2-7)	4 (2-7)	0.212
APS III	40 (29-54)	42 (31-56)	<0.001
CCI	5.5 ± 3.1	5.5 ± 2.9	0.864
MAP, mm Hg	83.1 ± 12.1	78.8 ± 11.5	<0.001
Creatinine, mg/dL	1.1 (0.8-1.8)	1.1 (0.8-1.8)	0.512
Hemoglobin, g/dL	10.5 ± 2.5	10.5 ± 2.4	0.961
LVEF, %	51.5 ± 16.6	52.5 ± 15.5	0.035
TR gradient, mm Hg	29 (23-38)	31 (24-40)	<0.001
Male sex, n (%)	1,983 (56.8)	7,266 (53.8)	0.002
CHF, n (%)	1,354 (38.8)	5,511 (40.8)	0.027
CKD, n (%)	812 (23.2)	3,337 (24.7)	0.072
COPD, n (%)	733 (21.0)	3,731 (27.6)	<0.001
Vasopressor, n (%)	827 (23.7)	4,900 (36.3)	<0.001
Mech. ventilation, n (%)	1,238 (35.4)	5,122 (37.9)	0.007
RRT, n (%)	242 (6.9)	700 (5.2)	<0.001
28-day mortality, n (%)	668 (19.1)	2,424 (18.0)	0.119

Continuous variables are mean ± SD or median (IQR); categorical variables are n (%). APS III, Acute Physiology Score III; CCI, Charlson Comorbidity Index; CHF, congestive heart failure; CKD, chronic kidney disease; COPD, chronic obstructive pulmonary disease; LVEF, left ventricular ejection fraction; MAP, mean arterial pressure; RRT, renal replacement therapy; SOFA, Sequential Organ Failure Assessment; TAPSE, tricuspid annular plane systolic excursion; TR, tricuspid regurgitation.

Supplementary Table 2. Variable-level missingness and availability

Variables	Available n (%)	Missing n	Missing %
Age	16,991 (100.0)	0	0.0
SOFA score	16,991 (100.0)	0	0.0
APS III	16,991 (100.0)	0	0.0
CCI	16,991 (100.0)	0	0.0
Heart rate	16,963 (99.8)	28	0.2
Mean arterial pressure	16,961 (99.8)	30	0.2
Creatinine	16,903 (99.5)	88	0.5
Hemoglobin	16,889 (99.4)	102	0.6
White blood cell count	16,887 (99.4)	104	0.6
Platelet count	16,893 (99.4)	98	0.6
Sodium	16,884 (99.4)	107	0.6
Blood urea nitrogen	16,907 (99.5)	84	0.5
TAPSE	3,494 (20.6)	13,497	79.4
TR pressure gradient	13,317 (78.4)	3,674	21.6
LVEF	13,768 (81.0)	3,223	19.0
Biplane LVEF	2,578 (15.2)	14,413	84.8
E/e' ratio	4,017 (23.6)	12,974	76.4
LA volume	6,433 (37.9)	10,558	62.1
IVC diameter	4,121 (24.3)	12,870	75.7

Counts refer to the 16,991-patient study cohort. Biomarker availability is reported separately below. APS III, Acute Physiology Score III; CCI, Charlson Comorbidity Index; IVC, inferior vena cava; LA, left atrial; LVEF, left ventricular ejection fraction; SOFA, Sequential Organ Failure Assessment; TAPSE, tricuspid annular plane systolic excursion; TR, tricuspid regurgitation.

Supplementary Table 3. Exploratory 28-day subgroup analyses

Subgroup	Level	N	Events	Adjusted HR per TR grade (95% CI)	Interaction P
Age	<65 years	6,463	868	1.18 (1.09-1.27)	<0.001
Age	≥65 years	10,379	2,202	1.23 (1.18-1.29)	<0.001
Sex	Female	7,671	1,443	1.23 (1.16-1.30)	0.546
Sex	Male	9,171	1,627	1.22 (1.15-1.29)	0.546
SOFA	SOFA <6	11,042	1,119	1.26 (1.18-1.35)	0.013
SOFA	SOFA ≥6	5,800	1,951	1.19 (1.14-1.25)	0.013
Congestive heart failure	No CHF	10,025	1,704	1.21 (1.15-1.28)	0.027
Congestive heart failure	CHF present	6,817	1,366	1.24 (1.17-1.31)	0.027
Chronic kidney disease	No CKD	12,719	2,133	1.21 (1.16-1.27)	0.047
Chronic kidney disease	CKD present	4,123	937	1.25 (1.16-1.33)	0.047
Vasopressor use	No vasopressor	11,133	1,349	1.32 (1.24-1.40)	<0.001
Vasopressor use	Vasopressor use	5,709	1,721	1.17 (1.11-1.23)	<0.001
Mechanical ventilation	No mechanical ventilation	10,514	1,184	1.35 (1.27-1.43)	<0.001
Mechanical ventilation	Mechanical ventilation	6,328	1,886	1.15 (1.09-1.21)	<0.001

Subgroup analyses were restricted to the primary complete-case cohort (N=16,842). Ordinal TR retains the original four-grade ordering. Interaction P values are from multiplicative terms; no multiplicity correction was applied. CHF, congestive heart failure; CI, confidence interval; CKD, chronic kidney disease; HR, hazard ratio; SOFA, Sequential Organ Failure Assessment; TR, tricuspid regurgitation.

Supplementary Table 4. Biomarker measurement availability

Biomarker	Any admission n (%)	-24 to +72 h n (%)	Numeric -24 to +24 h n (%)	Disposition
Troponin T	7,805 (45.9)	7,188 (42.3)	4,808 (28.3)	Measured-subgroup sensitivity
NT-proBNP	2,195 (12.9)	1,591 (9.4)	1,324 (7.8)	Coverage insufficient for cohort adjustment
D-dimer	592 (3.5)	436 (2.6)	276 (1.6)	Coverage insufficient for cohort adjustment
Fibrin degradation products	291 (1.7)	202 (1.2)	0 (0.0)	Coverage insufficient for cohort adjustment

Ordering was clinically selective. NT-proBNP and D-dimer were not used for full-cohort adjustment; no missing biomarker was imputed. NT-proBNP, N-terminal pro-B-type natriuretic peptide.

Supplementary Table 5. Principal-diagnosis categories

Principal ICU/hospitalization diagnosis category	n	%
Cardiovascular	6,303	37.1
Neurologic/cerebrovascular	2,363	13.9
Sepsis/infection	2,324	13.7
Trauma/injury/poisoning	1,659	9.8
Respiratory	1,187	7.0
Gastrointestinal/hepatic	1,012	6.0
Neoplasm/hematologic	822	4.8
Other	519	3.1
Endocrine/metabolic	313	1.8
Renal/genitourinary	242	1.4
Musculoskeletal	103	0.6
Symptoms/other	88	0.5
Obstetric	53	0.3
Principal diagnosis unavailable	3	0.0

Categories were grouped from principal discharge diagnosis codes and describe hospitalization context; they are not adjudicated causal ICU admission diagnoses. ICU, intensive care unit.

Supplementary Table 6. Early-TTE EF categories among patients with CHF codes

Early-TTE LVEF category among CHF-coded patients	n	% of CHF-coded patients
Reduced EF ($\leq 40\%$)	2,676	39.0
Preserved EF ($\geq 50\%$)	2,614	38.1
LVEF unavailable	1,202	17.5
Mildly reduced EF (41–49%)	373	5.4

These are early echocardiographic EF categories, not clinically adjudicated HFrEF, HFmrEF, or HFpEF phenotypes. CHF, congestive heart failure; EF, ejection fraction; HFmrEF, heart failure with mildly reduced ejection fraction; HFpEF, heart failure with preserved ejection fraction; HFrEF, heart failure with reduced ejection fraction; LVEF, left ventricular ejection fraction; TTE, transthoracic echocardiography.

Supplementary Table 7. Availability of report-level IVC collapse descriptions

IVC collapse-description category	n	%
Unavailable	13,012	76.6
>50% collapse documented	1,751	10.3
<50% collapse documented	734	4.3
Cannot assess RA pressure (including intubation)	586	3.4
Not visualized/unavailable	405	2.4
Normal IVC description without numeric collapse	329	1.9
Other structured IVC description	174	1.0

Structured collapse descriptions were absent in 76.6% and were not used as a complete-cohort covariate. Mechanical ventilation limits respiratory-variation interpretation. IVC, inferior vena cava; RA, right atrial.

Supplementary Table 8. Full-cohort mortality counts at sequential follow-up intervals

TR group	28-day deaths / n (%)	90-day deaths / n (%)	365-day deaths / n (%)
Low-grade	1,880/12,417 (15.1)	2,523/12,417 (20.3)	3,380/12,417 (27.2)
Moderate	850/3,467 (24.5)	1,128/3,467 (32.5)	1,488/3,467 (42.9)
Severe	362/1,107 (32.7)	473/1,107 (42.7)	591/1,107 (53.4)

These full-cohort counts differ from complete-case Cox-model event counts reported in the main tables. TR, tricuspid regurgitation.

Supplementary Table 9. Sex-stratified 365-day mortality according to TR severity

Sex	TR severity	Crude mortality, n/N (%)	Multiple-adjusted HR	Sodium-adjusted HR
Female	Low-grade	1,466/5,202 (28.2%)	Reference	Reference
Female	Moderate	767/1,887 (40.6%)	HR = 1.28 $P < 0.001$ 95% CI: 1.17–1.40	HR = 1.28 $P < 0.001$ 95% CI: 1.17–1.40
Female	Severe	336/653 (51.5%)	HR = 1.62 $P < 0.001$ 95% CI: 1.43–1.84	HR = 1.61 $P < 0.001$ 95% CI: 1.42–1.83
Male	Low-grade	1,914/7,215 (26.5%)	Reference	Reference
Male	Moderate	721/1,580 (45.6%)	HR = 1.36 $P < 0.001$ 95% CI: 1.25–1.49	HR = 1.36 $P < 0.001$ 95% CI: 1.24–1.49
Male	Severe	255/454 (56.2%)	HR = 1.74 $P < 0.001$ 95% CI: 1.52–2.00	HR = 1.74 $P < 0.001$ 95% CI: 1.52–2.00

Low-grade TR is the reference. Sex-stratified models use the primary adjustment set with sex omitted; sodium-adjusted models additionally include sodium. Primary model N/events: women 7,671/2,547 and men 9,171/2,865. Sodium-adjusted N/events: women 7,662/2,545 and men 9,162/2,864. CI, confidence interval; HR, hazard ratio; TR, tricuspid regurgitation.

Supplementary Table 10. Age-stratified 365-day mortality according to TR severity

Age group	TR severity	Crude mortality, n/N (%)	Multiple-adjusted HR	Sodium-adjusted HR
<30	Low-grade	53/396 (13.4%)	Reference	Reference
<30	Moderate	5/33 (15.2%)	Not reliably estimable	Not reliably estimable
<30	Severe	0/12 (0.0%)	Not reliably estimable	Not reliably estimable
30–49	Low-grade	294/1,600 (18.4%)	Reference	Reference
30–49	Moderate	53/191 (27.7%)	Not reported*	Not reported*
30–49	Severe	16/51 (31.4%)	Not reliably estimable*	Not reliably estimable*
50–69	Low-grade	1,180/5,068 (23.3%)	Reference	Reference
50–69	Moderate	326/898 (36.3%)	HR=1.41 <i>P</i> < 0.001 95% CI: 1.24–1.60	HR=1.40 <i>P</i> < 0.001 95% CI: 1.24–1.59
50–69	Severe	103/236 (43.6%)	HR=1.55 <i>P</i> < 0.001 95% CI: 1.25–1.92	HR=1.48 <i>P</i> < 0.001 95% CI: 1.19–1.84
≥70	Low-grade	1,853/5,353 (34.6%)	Reference	Reference
≥70	Moderate	1,104/2,345 (47.1%)	HR=1.27 <i>P</i> < 0.001 95% CI: 1.17–1.37	HR=1.27 <i>P</i> < 0.001 95% CI: 1.17–1.37
≥70	Severe	472/808 (58.4%)	HR=1.66 <i>P</i> < 0.001 95% CI: 1.50–1.85	HR=1.66 <i>P</i> < 0.001 95% CI: 1.50–1.85

Age strata use non-overlapping categories <30, 30–49, 50–69, and ≥70 years. Continuous age was retained within age strata as residual adjustment. For patients aged <30 years, standard adjusted Cox estimates were not considered reliably estimable because the moderate-TR group included 33 patients/5 deaths and the severe-TR group 12 patients/0 deaths. In the 30–49-year stratum, moderate and severe TR were estimated jointly; because the severe-TR cell was sparse (51 patients/16 deaths), the stratum-specific Cox model was withheld as a whole. Accordingly, the moderate-TR coefficient was not reported separately despite 191 patients and 53 deaths in that group. For ages 50–69, primary model N/events were 6,145/1,594 and sodium-adjusted N/events 6,137/1,592; for ages ≥70, corresponding values were 8,434/3,402 and 8,426/3,401. CI, confidence interval; HR, hazard ratio; TR, tricuspid regurgitation. *The asterisk denotes the 30–49-year stratum-level model decision described above.