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RESEARCH ARTICLE

One-year follow-up of patients with severe tricuspid regurgitation: prognostic impact of right heart failure staging

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Abstract

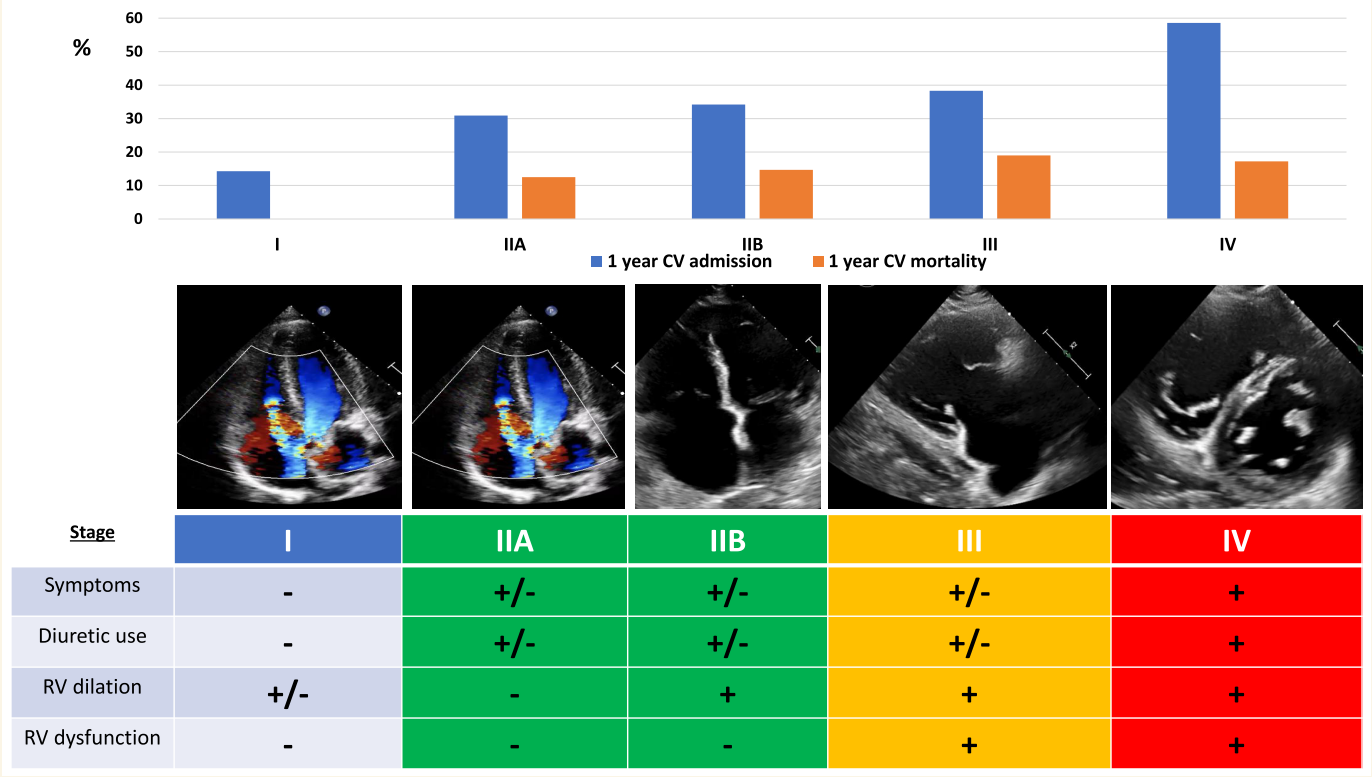
Introduction Severe tricuspid regurgitation (STR) is associated with adverse outcomes. This nationwide registry describes the prevalence, aetiology, clinical, and echocardiographic profile of STR using contemporary classifications, and evaluates the prognostic impact of a modified right heart failure staging (RHFS).

Methods We prospectively enrolled consecutive patients with STR from 48 centres over 6 months and followed them for 1 year. TR severity (severe, massive, torrential) and aetiology were defined using current recommendations. A modified RHFS incorporating RV remodelling and systolic dysfunction was applied.

Results We included 1247 patients (mean age 76.7 ± 10.5 years; 70.2% women): 64.9% had severe TR, 24.1% massive TR, and 11.0% torrential TR. V-STR due to left-heart disease was the most frequent aetiology (49.9%), followed by A-STR (19.8%). During follow-up, HF hospitalization occurred in 20.8%, cardiovascular death in 9.2%, and 12.3% underwent tricuspid intervention. In multivariable analysis, Stages III–IV of the modified RHFS were independently associated with HF hospitalization (HR 1.58; 95% CI 1.13–2.23; $P = .008$) and cardiovascular mortality (HR 2.01; 95% CI 1.27–3.18; $P = .003$). Massive (HR 1.72; 95% CI 1.02–2.91; $P = .041$) and torrential TR (HR 2.57; 95% CI 1.24–5.34; $P = .011$) were also associated with cardiovascular mortality.

Conclusion Massive and torrential TR identify patients at the highest risk. The modified RHFS stratifies patients with STR into prognostic categories and may help guide the timing of intervention.

Graphical Abstract



Stage	I	IIA	IIB	III	IV
Symptoms	-	+/-	+/-	+/-	+
Diuretic use	-	+/-	+/-	+/-	+
RV dilation	+/-	-	+	+	+
RV dysfunction	-	-	-	+	+

Central Illustration: New proposed staging system for RHF in patients with severe TR and its prognostic impact. CV: cardiovascular. RHF: right heart failure. RV: right ventricle. TR: tricuspid regurgitation.

Keywords Atrial fibrillation • Right heart failure • Right ventricle • Severe tricuspid regurgitation • Cardiovascular mortality

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Introduction

Significant (moderate to severe) tricuspid regurgitation (TR) has a prevalence between 1.5% and 15% of echocardiographic studies and is directly related to age.[1]

Traditionally, TR has been classified in a binary way as primary or organic and secondary or functional. Recently, three different morphological types have been described: primary TR due to organic valvular involvement; TR secondary to the presence of pulmonary hypertension (PHT), which leads to right ventricular (RV) remodelling and traction of the subvalvular apparatus on the leaflets (increased tenting area); and idiopathic or atriogenic TR, typical of patients with atrial fibrillation (AF) who develop dilatation of the tricuspid annulus (normal leaflets and absence of pulmonary hypertension).[2]

Another relevant and relatively recent TR classification quantifies and divides the degree of severe TR into severe, massive, and torrential.[3] Apparently, the latter carries a worse prognosis.[4] Severe tricuspid regurgitation (TR) leads to progressive right ventricular dilation and failure if left untreated.[1]

Studies highlighting the prognostic importance of severe TR are scarce. Those that have already been published either have a small number of patients,[2] are retrospective[3–5] or have a mix of patients with moderate and severe TR. In fact, in these series, most patients had moderate TR.[6,7] In practice, moderate and severe TR are managed differently.[1]

Regarding prognosis, previous classifications in patients with TR have been published,[6] but focusing solely on the RV systolic dysfunction (without considering RV dilation) and including patients with moderate TR. Our hypothesis was that RV dilation secondary to TR occurs before RV systolic dysfunction as a previous sign of RV overload. Therefore, in these patients, to better stratify their prognosis, the presence of RV remodelling with or without RV systolic dysfunction could be considered.

The herein study is a nationwide prospective registry dedicated exclusively to severe TR and to their different degrees of severity.

This registry has a main objective: to describe the prevalence, aetiology, clinical profile, and echocardiographic findings of patients with severe TR according to the latest classifications and to assess 1 year outcomes, and two secondary objectives: (i) to assess the potential benefit of a modified version of right heart failure staging in patients with severe TR, and (ii), to assess the patients prognosis according to the degree of TR.

Methods

Study population

In this national, multicentre, observational, and prospective registry, all patients with severe TR diagnosed in the Cardiac Imaging Units of the 48 participating centres have been consecutively collected over a period of 6 months (from November 2021 to April 2022). Informed consent was obtained from all patients. At the time of inclusion, demographic data, electrocardiographic features, clinical findings, echocardiographic variables, medications, as well as the results of blood tests were collected in all patients. A prospective follow-up of the recruited patients was carried out.

Echocardiographic, clinical, and analytical variables

TR severity was quantified by transthoracic echocardiography and categorized in three grades: severe (Vena contracta (VC) $\geq$ 0.7 cm, effective regurgitant orifice (ERO) $\geq$ 0.40 cm^2, and/or regurgitant volume $\geq$ 45 ml), massive (VC 14–20 mm, ERO 0.60–0.79 cm^2) and torrential (VC $\geq$ 21 mm, ERO $\geq$ 0.80 cm^2) according to current classifications.[8,9]

Most of the population was found in AF (85%), so the measurements were obtained as the average of 3 echocardiographic measurements.[9]

From an aetiological point of view, TR was predefined into five groups: primary TR (organic disease of the tricuspid leaflets); ventricular functional TR (V-STR) due to left heart disease (morphologically normal tricuspid leaflets with RV remodelling due to PHT secondary to left heart disease); V-STR due to lung disease (normal tricuspid leaflets with RV remodelling due to PHT secondary to lung disease); functional A-STR (diagnosis reached by exclusion; leaflets are morphologically normal and TR is secondary to dilation of the tricuspid annulus (TA) from permanent AF; they do not have significant PHT, and if there is RV remodelling, it is more prominent at basal level, in the RV inflow tract, with no or mild leaflet tethering, rather than the RV longitudinal remodelling associated with PHT, generating severe tethering), and cardiac implantable electronic device (CIED)-related TR (lead-associated type A).[9] Although the Tricuspid Valve Academic Research Consortium[9] classification includes TR secondary to primary right ventricular diseases, no patients fulfilling this mechanism as the dominant cause of severe TR were identified in our cohort. For clarity, this category has been explicitly acknowledged, although it was not represented in our study population. In case of confluence of several aetiologies in the same patient, the most relevant one was chosen at the discretion of the principal investigator of each hospital.

Clinically, patients were initially stratified according to the classification proposed by Haddad et al[10] and adapted by Dietz et al.[6] We proposed a new RHF staging in patients with severe TR, defining five stages: Stage I: asymptomatic without diuretic treatment, no right ventricular (RV) dysfunction and with or without RV dilatation; Stage IIA: signs/symptoms of RHF and/or taking diuretics without RV dilatation or dysfunction, Stage IIB: Dilated RV with signs/symptoms of RHF and/or taking diuretics without RV dysfunction; Stage III: RV dysfunction with RV dilatation with or without signs/symptoms of RHF, and or taking diuretics, and Stage IV: RV dysfunction with refractory signs/symptoms of RHF.

RV remodelling was defined as the change in size (dilatation) and shape (conical or spherical) of the RV and the TA.[9] TR results in dilatation of the RV, which can subsequently lead to further annular dilatation and tethering of tricuspid valve leaflets.[9,11] To measure RV dimensions, RV focused apical 4-chamber view was used. An RV diameter of >41 mm at the base and >35 mm at the mid-level were considered abnormal. All chamber measurements were made from inner-edge to inner-edge. RV systolic function was assessed by a combination of the following parameters: tricuspid annular plane systolic excursion (TAPSE) and RV fractional area change (FAC). TAPSE was calculated on M-mode recordings of the lateral TA. FAC was calculated by the following formula: FAC = (end-diastolic area–end-systolic area/end-diastolic area)×100. RV systolic dysfunction was defined when TAPSE was <17 mm and FAC <35%.[12]

RV-free wall longitudinal strain was analysed in 427 patients (34% of the total population). This parameter was measured in the RV-focused apical 4-chamber view and calculated as the mean of the RV lateral-basal, mid, and apical segments, with exclusion of the septal segments. A longitudinal RV-free wall strain >−23% was considered abnormal.[13] The TA was measured at end-diastole in the RV focused apical 4-chamber view. The diameter of the TA was considered dilated if it measured > 40 mm or >21 mm/m^2.[12] Systolic pulmonary artery pressure (SPAP) was estimated through the maximum velocity of the TR jet using continuous wave Doppler.[9,11] Ventriculoarterial coupling is an index of RV contractility to afterload and may further characterize the RV systolic function compensation to RV overload. VAC was estimated by the TAPSE/SPAP ratio. A cut-off value of <0.31 mm/mmHg indicates RV-PA uncoupling.[14]

Kidney disease was defined according to the Tricuspid Valve Academic Research Consortium,[9] and chronic liver disease as elevation of at least two of three cholestatic hepatic enzymes.[15]

The data collection by the participating researchers has been carried out through a centralized telematic database. The Ethics Committee of each centre approved the protocol, which was carried out in accordance with the Declaration of Helsinki for studies in humans.

Statistical analysis

Continuous variables are described as mean ± SD or median [interquartile range] depending on whether the assumption of normality verified by the Kolmogorov–Smirnov test was met. Categorical variables are expressed with the absolute number and percentage. Comparisons between groups were made using the χ^2 test or Fisher's exact test for categorical variables. For continuous variables, Student's *t* test or Mann–Whitney *U* test were used to compare two groups. One-way ANOVA was used to compare

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Table 1 Clinical characteristics of the total population according to the different grades of severe tricuspid regurgitation

	Total population ($n = 1247$)	Severe TR ($n = 810$)	Massive TR ($n = 301$)	Torrential TR ($n = 136$)	P
Age (years)	76.7 (10.5)	77.5 (10.6)	75.8 (10.3)	74.5 (10.6)	.007*
Gender (male)	371 (29.8%)	207 (28.8%)	90 (32.8%)	40 (36.7%)	.106
HT	812 (65.1%)	502 (68.4%)	187 (68.2%)	79 (72.5%)	.677
HCOL	579 (46.4%)	358 (48.8%)	135 (49.3%)	49 (45%)	.728
DM	303 (24.3%)	194 (26.4%)	59 (21.5%)	31 (28.4%)	.212
BSI	28.2 (26.9–29.5)	28.8 (27–30.6)	27.6 (26–29.2)	25.9 (25.1–26.7)	.373
Stroke	159 (12.2%)	88 (12%)	38 (13.9%)	19 (17.4%)	.254
CKD	256 (20.7%)	134 (18.1%)	67 (23.4%)	34 (28.7%)	.094
COPD	214 (17.2%)	129 (17.5%)	54 (19.7%)	18 (16.5%)	.590
Chronic liver disease	68 (5.5%)	41 (5.6%)	16 (5.8%)	9 (8.3%)	.543
Coronary artery disease	148 (13.2%)	103 (14%)	30 (10.9%)	15 (13.8%)	.432
HF admission	459 (41.1%)	289 (39.4%)	120 (43.8%)	50 (45.9%)	.253
AF	948 (84.9%)	612 (83.4%)	245 (89.4%)	91 (83.5%)	.118
Pacemaker	191 (17.1%)	135 (18.1%)	35 (12.8%)	21 (19.3%)	.089
ICD/CRT	41 (3.6%)	25 (3.5%)	10 (3.7%)	6 (5.5%)	.402
Previous valve surgery	294 (26.3%)	187 (25.5%)	79 (28.8%)	28 (25.7%)	.553
Biologic prosthetic mitral valve	17 (1.5%)	12 (1.6%)	5 (1.8%)	0	.384
Mechanical prosthetic mitral valve	155 (13.9%)	91 (12.4%)	47 (17.2%)	17 (5.6%)	.130
Mitral valve repair	24 (2.1%)	21 (2.9%)	2 (0.7%)	1 (0.9%)	.124
Tricuspid annuloplasty	47 (4.2%)	27 (3.7%)	15 (5.5%)	5 (4.6%)	.451
TAVI	30 (2.7%)	7 (2.6%)	7 (2.6%)	1 (0.9%)	.440
Mitraclip	13 (1.2%)	3 (1.1%)	3 (1.1%)	1 (0.9%)	.954

AF, atrial fibrillation; BSI, body Surface index; CHF, congestive heart failure; CKD, Chronic kidney disease; COPD, chronic obstructive pulmonary disease; DM, diabetes mellitus; HCOL, hypercholesterolaemia; HT, hypertension; ICD, implantable cardioverter defibrillator; TAVI, transcatheter aortic valve implantation; TR, tricuspid regurgitation.
*Significant between severe TR and torrential TR.

differences among more than two groups, and multiple comparisons of continuous variables were tested with Bonferroni correction. The primary cardiovascular endpoint was a composite of death from cardiovascular causes and HF hospitalization.

A multivariable Cox proportional hazard regression analysis by backward stepwise (likelihood ratio) selection was performed to identify parameters independently associated with 1-year CV mortality and 1-year HF hospitalization. Entry criteria for the multivariable regression analysis were a significant association in univariable analysis and clinical relevance. Possible collinearity among the different introduced variables was evaluated and controlled for overfitting in the model. Verification of the proportional hazard assumption was performed by Schoenfeld residuals. Hazard ratios (HR) and 95% confidence intervals (CI 95%) were calculated. C-Index was used to measure the discriminatory capacity. Calibration was evaluated with the Gronesby–Borgan test. Internal validation was performed using time-dependent AUC at each time point across all bootstrap samples.

The 1-year CV mortality and HF admission event rates were estimated by Kaplan–Meier survival curves, which were compared using the log-rank test.

All tests were two-sided. Statistical significance was established at a P value of .05. All analyses were performed using R-software, V.3.6.1 (R Project for Statistical Computing).

Results

Patient characteristics

A total of 1247 consecutive patients with severe grades of TR were included in the Registry. Of these, 810 (64.9%) had criteria for severe TR, 301 (24.1%) massive TR, and 136 (11%) torrential TR. The frequency of detection of severe TR in Cardiac Imaging Units during the study period was 0.9% of all echocardiograms performed. This frequency is higher if we only consider those centres that had Cardiac Surgery (1.47%). Mean age was 76 ± 10.5 years, and the majority were women (70.2%). Regarding the baseline characteristics of the population, a high prevalence of classic cardiovascular risk factors and important comorbidities such as cerebrovascular disease, chronic kidney disease, lung disease, and liver disease stand out (*Table 1*).

When analysing the CV history, 459 patients (41.1%) had a history of HF and 948 (84.9%) were in AF (*Table 2*). A quarter of the population ($n = 294$; 26.3%) had undergone valve surgery before inclusion, the most frequent being mitral valve replacement with a mechanical prosthesis ($n = 155$; 13.9%). A total of 232 patients (20.7%) had pacemakers or defibrillators.

Regarding the aetiology of TR, the most frequent was V-STR due to left heart disease ($n = 621$; 49.9%), followed by A-STR ($n = 247$; 19.8%) (*Figure 1*). When analysing the severity of TR according to its aetiology, we found that, while in V-STR due to left heart disease, torrential TR is less frequent than severe and massive TR, in the case of A-STR occurs the opposite. In patients with primary TR, higher degrees of TR predominate (*Table 2*).

Clinical condition and echocardiographic characteristics

Clinical condition of the overall population at the time of inclusion is presented in *Table 3*. The most frequent symptom was exertional

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Table 2 Distribution of the aetiology of severe tricuspid regurgitation in the global population and according to grading stratification

	Total population (*n* = 1247)	Severe TR (*n* = 810)	Massive TR (*n* = 301)	Torrential TR (*n* = 136)	*P*
V-STR	621 (49.8%)	421 (51.9%)	147 (48.8%)	53 (38.8%)	*<.001**
Functional due to lung disease	82 (6.6%)	53 (6.5%)	18 (6.0%)	11 (8.1%)	*<.001**
A-STR	247 (19.8%)	157 (19.4%)	60 (19.9%)	30 (22.4%)	*<.001**
CIED-related	109 (8.7%)	74 (9.1%)	22 (7.3%)	13 (9.5%)	*<.001***
Organic disease	152 (12.2%)	80 (10%)	47 (15.6%)	25 (18.3%)	*<.001**
Other causes not defined	36 (2.9%)	25 (3.1%)	7 (2.4%)	4 (2.9%)	*NS*

Left heart disease was defined as: moderate or higher native or prosthetic, mitral or aortic valve regurgitation/stenosis or systolic LV dysfunction (LVEF < 40%).
Lung disease was defined as: chronic obstructive pulmonary disease or other restrictive lung diseases.
CIED-related: cardiac implantable electronic device-related TR.
A-STR, atrial functional; TR, tricuspid regurgitation; V-STR, ventricular functional TR due to left heart disease.
*Significant between severe TR and torrential TR.
**Significant between massive TR and torrential TR.
The values in italics are intended solely to highlight statistical significance.

dyspnoea (67.4%), followed by peripheral oedema in 40.9%. Except for seven patients, all the others were on diuretics; most were taking furosemide (24.2% of the total population received a dose higher than 80 mg/day), 446 (35.7%) potassium-sparing diuretics, and 181 (14,5%) thiazide diuretics.

As expected, signs of systemic venous congestion (elevated jugular venous pressure, oedema, hepatomegaly, and ascites) were present more frequently in patients with massive and torrential TR than in those with severe TR. In addition, the former were in a more advanced NYHA functional class and required higher doses of furosemide than those with severe TR.

Regarding the echocardiographic parameters analysed, the mean left ventricular ejection fraction (LVEF) was 54.6 ± 13.1% and 27.9% of the patients (*n* = 348) had a LVEF < 50%. (*Table 4*). The most common concomitant valve disease was mitral regurgitation (34.5%). PHT (SPAP >70 mmHg) was documented in 28.8% of the patients. When comparing the diameters of the RV and TA depending on the severity of the TR, patients with torrential TR had larger diameters than those with massive TR, and these were larger than those of patients with severe TR. Impaired RV free wall longitudinal strain was more frequently found in patients with torrential TR than in the other groups (*Table 4*).

At the time of diagnosis, and according to the classification of Haddad et al.,[10] most patients (95.9%) fall into stage 3 (RV dysfunction with symptoms of RHF or diuretic use) (*Table 4*, *Supplementary material*).

To better stratify the patient's clinical picture, we have slightly modified this classification considering that in RV volume overload conditions such as severe TR, an initial adaptive response of RV dilation is followed by progressive contractile dysfunction.[11] Accordingly, in addition to the presence or not of signs of RHF, we have distinguished the RV dilation from the RV with systolic dysfunction. Thus, the patients´ new RHF staging is as shown in *Table 5*.

Now, the largest group is the one that includes patients with a non-dilated RV and preserved systolic function, but with symptoms of RHF or diuretic use (44%). The aetiology of TR differed according to the different RHF stages (*Table 6*). Clinical and echocardiographic characteristics, and risk score (TRI-SCORE)[16] of patients according to their RHF stage are shown in *Table 7*.

Laboratory results are shown in *Table 8*. There were no significant differences in glomerular-filtration rate. A typical pattern of cholestasis (increased alkaline phosphatase and gamma-glutamyl transferase) was seen in 57% of the patients. The greater the degree of regurgitation, the more important the cholestatic pattern was.

Outcomes and TV interventions

A total of 1172 patients (94%) completed 1 year follow-up (75 patients refused to attend the protocol follow-up visits). One hundred 87 patients were asymptomatic at the time of inclusion and only 7 were symptom-free and not taking diuretics. Of the asymptomatic patients, 18.7% (*n* = 35) developed signs/symptoms of RHF at follow-up. Hospital admission was required in 403 patients (34,4%) during follow-up, 244 (20,8%) due to HF decompensation. Death occurred in 175 patients (14.9%) and 108 of them (9.2%) were due to refractory HF; stroke was the cause of death in 6 patients and 61 (5.2%) died due to non-CV causes. Univariable and multivariable regression analyses for the combined endpoint CV are shown in *Supplementary Table S5*.

TV interventions were performed in 145 patients (12,3%). Surgery was done in 71 patients (6,0%): surgical repair in 50 and surgical replacement in 21. Forty-eight had STR, 14 primary TR, and 9 CIED-related TR. Most surgical patients were in stages IIA (39.4%), IIB (30.9%), and III (23.9%). Transcatheter TV interventions were performed in 74 patients (6,3%): percutaneous edge-to-edge repair in 58, percutaneous annuloplasty in 1, and bicaval transcatheter prosthesis implantation in 15. Most patients (95%) had STR, and of them, 40% were in stage IIA, 27% in stage IIB, and 33% in stage III.

Univariable and multivariable regression analyses for CV mortality are shown in *Table 9*. Age, impaired renal function, previous HF history, massive and torrential TR, and the new proposed RHF staging were significantly associated with worse survival. Kaplan–Meier curves for CV mortality according to RHF staging are shown in *Figure 2*. Stages III and IV were identified as independent predictors of CV mortality. Mortality was not associated with TR aetiology (*Figure 3*).

Kaplan–Meier curves for a combined endpoint of all-cause death, new HF admission, or TV intervention are presented in *Figure 4A*. Women had greater 1-year survival than men (*Figure 4B*), but there were no differences in CV-mortality (*P* = .361). *Figure 4C* shows an analysis of overall survival comparing patients that underwent tricuspid intervention versus medical treatment. Kaplan–Meier curve for overall 1-year survival depending on the new RHF staging is presented in *Supplementary material*.

Independent predictors of HF hospital admission in the multivariable analysis are shown in *Table 10*. Age, chronic kidney disease, previous HF admission, haemoglobin levels <10 g/dl, and stages III and IV of the new RHF classification were associated with high risk of admission due to HF.

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Figure 1 Distribution of the aetiology of severe tricuspid regurgitation in the global population

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Table 3 Clinical condition of the global population at the time of inclusion according to the degree of TR

	Total population ($n = 1247$)	Severe TR ($n = 810$)	Massive TR ($n = 301$)	Torrential TR ($n = 136$)	*P*
Asymptomatic	187 (16.7%)	127 (17.3%)	43 (15.7%)	17 (15.6%)	*.785*
Peripheral oedema	457 (40.9%)	284 (38.7%)	122 (44.5%)	51 (46.8%)	*.104*
Ascites	72 (6.4%)	35 (4.8%)	24 (8.8%)	13 (11.9%)	*.004**
Dyspnoea	753 (67.4%)	492 (67%)	190 (69.3%)	71 (65.1%)	*.680*
Asthenia	172 (15.4%)	115 (15.7%)	36 (13.1%)	21 (19.3%)	*.306*
JVP	200 (17.9%)	112 (15.3%)	61 (22.3%)	27 (22.4%)	*<.005***
Hepatomegaly	116 (10.4%)	65 (8.9%)	38 (13.9%)	13 (11.9%)	*.005***
Rales	129 (11.5%)	75 (9.2%)	40 (14.6%)	14 (12.8%)	*.139*
NYHA III/IV	323 (29.8%)	197 (27.8%)	89 (33.2%)	37 (34.3%)	*.005***
Furosemide dose > 80 mg/day	197 (24.2%)	174 (23%)	78 (26.4%)	35 (27.1%)	*.047*

JVP, jugular venous pressure; NYHA, New York Heart Association; TR, tricuspid regurgitation.
*Significant between severe TR and torrential TR.
**Significant between severe TR and massive and torrential TR.
The values in italics are intended solely to highlight statistical significance.

Table 4 Echocardiographic characteristics of the global population according to the degree of TR

	Total population ($n = 1247$)	Severe TR ($n = 810$)	Massive TR ($n = 301$)	Torrential TR ($n = 136$)	*P*
LVEF (%)	54.6 (13.1%)	57.2 (11.4)	56 (10.9)	58.6 (11.3)	*.116*
Severe PHT	296 (28.8%)	215 (30.7%)	66 (26.8%)	15 (18.3%)	*.002**
VAC	0.40 (0.12)	0.38 (0.11)	0.40 (0.12)	0.50 (0.18)	*.452*
RV basal diameter (mm)	43.9 (7.1)	46.3 (7.2)	48.4 (7.8)	54.9 (8.7)	*<.005**
Tricuspid annulus diastolic diameter (mm)	41.6 (8.1)	40.3 (6.3)	41.9 (6.5)	44.5 (7.3)	*<.005**
TAPSE (mm)	21.8 (14–21)	18.4 (15–21)	18 (15.7–20)	19.4 (16–23)	*.013**
FAC (%)	41.2 (10.5%)	39.2 (9.8%)	40.3 (10.1)	40.6 (10.3)	*.256*
RV free wall LS > −23%[a]	310 (24.8%)	195 (24%)	72 (24%)	43 (31%)	*<.005**
MR mod/sev	441 (34.5%)	298 (41.6%)	102 (38%)	31 (29%)	*.004**
MS mod/sev	85 (6.8%)	63 (8.9%)	32 (12.2%)	4 (3.8%)	*.079*
AS mod/sev	99 (9.2%)	55 (7.8%)	26 (9.7%)	4 (3.8%)	*.12*
AR mod/sev	154 (14.2%)	100 (14.1%)	41 (15.4%)	13 (12.2%)	*.136*

AS, aortic stenosis; AR, aortic regurgitation; FAC, fractional area change; LVEF, left ventricle ejection fraction; MR, mitral regurgitation; MS, mitral stenosis; PHT, pulmonary hypertension; RV, right ventricle; RV free wall LS, right ventricle free wall longitudinal strain; TAPSE, tricuspid annular plane systolic excursion; VAC, ventriculo-arterial coupling.
[a]These data were only available in 427 patients.
*Significant between severe TR and torrential TR.
The values in italics are intended solely to highlight statistical significance.

Discussion

The main findings of this large real-life series of patients with severe TR are: (i) severe TR in cardiac imaging units is prevalent, especially in older women, and is accompanied by a high percentage of comorbidities; (ii) the most common aetiology is V-STR due to left heart disease, followed by A-STR; (iii) the higher the degree of TR, the greater the RV remodelling, (iv) traditional systolic RV function parameters did not show significant differences among the three groups of patients with severe TR; (v) massive and torrential TR were associated with a higher CV mortality; and (vi) RHF classical staging do not adequately stratify patients with severe TR. A better stratification with prognostic impact can be achieved with a slight modification of RHF stages distinguishing dilation from systolic dysfunction.

In this study, severe TR was documented in approximately 1% of the echocardiograms of the total cohort and in 1.5% of those performed in hospitals with cardiac surgery. Other studies[16] have found similar frequencies with a slight variation depending on whether the echocardiogram was performed in hospitalized patients (1%) or outpatients (0.85%).[17]

As in other series, most patients are older women with a high burden of cardiovascular risk factors and comorbidities.[17–19] In our series, the proportion of patients with massive or torrential TR (35.1%) was much higher than that detected in the study by Vieitez et al. (4.9%).[17] This difference can be justified because in our series patients with moderate TR were not included and most of our patients (85%) had AF compared with only 48% of those in the series by Vieitez et al.[17] Regarding prognosis, overall mortality was higher in men ($P < .001$), but no differences were observed in cardiovascular mortality.

Regarding the aetiology, the most frequent cause of severe TR was V-STR due to left heart disease, followed by A-STR. The latter type of TR deserves special attention; it was present in 20% of the

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Table 5 Distribution of the global population and the different degrees of TR according to the new right heart failure staging

	Total population ($n = 1247$)	Severe TR ($n = 810$)	Massive TR ($n = 301$)	Torrential TR ($n = 136$)	*P*
Stage I	7 (0,6%)	6 (0.8%)	1 (0.3%)	0	<.005
Stage IIA	550 (44,1%)	370 (45.6%)	120 (39.9%)	43 (31.6%)	<.005
Stage IIB	313 (25,1%)	189 (23.3%)	82 (27.3%)	55 (40.5%)	<.005
Stage III	346 (27,7%)	227 (28.1%)	90 (29.9%)	34 (25%)	<.005
Stage IV	31 (2,5%)	18 (2.2%)	8 (2.6%)	4 (2.9%)	<.005

Stage I: asymptomatic without diuretic treatment, no right ventricular (RV) dysfunction, and with or without RV dilatation.
Stage IIA: signs/symptoms of RHF and/or taking diuretics without RV dilatation or dysfunction.
Stage IIB: Dilated RV with signs/symptoms of RHF and/or taking diuretics without RV dysfunction.
Stage III: RV dysfunction with RV dilatation with or without signs/symptoms of RHF, and or taking diuretics.
Stage IV: RV dysfunction with refractory signs/symptoms of RHF.

Table 6 Distribution of the global population of patients according to the aetiology of TR in the proposed new right heart failure staging

	Functional ventricular due to left heart disease ($n = 621$, 49,9%)	Functional due to lung disease ($n = 82$, 6,5%)	Pacemaker lead ($n = 109$, 19,8%)	Functional atriogenic ($n = 247$, 8,7%)	Organic TR ($n = 90$, 7,3%)	*P*
Stage I	1 (0,2%)	0	0	3 (1,2%)	3 (3,3%)	<.005
Stage IIA	254 (40,9%)	32 (39%)	52 (47,7%)	104 (42,1%)	55 (61,1%)	<.005
Stage IIB	150 (24,1%)	25 (30,5%)	24 (22%)	72 (29,2%)	7 (7,8%)	<.005
Stage III	196 (31,6%)	22 (26,8%)	33 (30,3%)	62 (25%)	23 (25,6%)	<.005
Stage IV	20 (3,2%)	3 (3,7%)	0	6 (2,4%)	2 (2,2%)	<.005

Table 7 Clinical and echocardiographic characteristics of the total population, classified according to the proposed stages of right heart failure in patients with severe TR

	Stage I ($n = 7$)	Stage IIA ($n = 550$)	Stage IIB ($n = 313$)	Stage III ($n = 346$)	Stage IV ($n = 31$)
Peripheral oedema	0	168 (30.5%)	118 (37.7%)	165 (47.6%)	24 (77.4%)
Ascites	0	22 (4%)	17 (5.4%)	31 (8.9%)	6 (19.3%)
Dyspnoea	0	303 (55.1%)	220 (70.2%)	241 (69.6%)	27 (87%)
Elevated JVP	0	70 (12.7%)	56 (17.9%)	70 (20.2%)	13 (41.9%)
Hepatomegaly	0	43 (7.8%)	35 (11.2%)	35 (11.1%)	10 (32.2%)
NYHA III/IV	0	105 (19.1%)	95 (30.3%)	109 (31.5%)	31 (100%)
Furosemide dose > 80 mg/day	0	58 (10.5%)	52 (9.4%)	81 (14.7%)	11 (35.4%)
LVEF	62% (12)	53.7% (16)	58.4% (13)	56.5% (16)	50% (14)
Severe PHT	0	103 (18.7%)	83 (26.5%)	104 (18.9%)	15 (48.4%)
Tricuspid annulus (mm)	38 (3)	36.6 (3)	46.6 (3)	47.6 (4)	37 (4)
TAPSE (mm)	24 (19–26)	19 (18–22)	19 (18–22)	14 (11–17)	14 (8–16)
FAC	45 (5)	39.2 (4)	37.2 (3)	32.8 (5)	33.4 (4)
RV free wall LS (427 patients)	24 (2)	21.5 (5)	20.4 (5)	15.5 (7)	14.5 (7)
Tri-score (mean)	1 (0)	2.3 (1.3)	3.7 (1.5)	4.3 (1.4)	5.3 (1.2)

FAC, fractional area change; JVP, jugular venous pressure; LVEF, left ventricular ejection fraction; NYHA, New York Heart Association; PHT, pulmonary hypertension; RV free wall LS, right ventricular free wall longitudinal strain; TAPSE, tricuspid annular plane systolic excursion; VAC, ventriculo-arterial coupling.

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Table 8 Laboratory values of the global population and according to the different degrees of severe TR

	Total population ($n = 1247$)	Severe TR ($n = 810$)	Massive TR ($n = 301$)	Torrential TR ($n = 136$)	*P*
Hb (g/dl)	12.4 (2.2)	12.4 (2.1)	12.5 (2.1)	12.2 (3.4)	*NS*
GFR (CKD-EPI)	57.1 (20.8)	56.9 (21.7)	58.1 (21.3)	58.4 (21.9)	*NS*
Urea (mg/dl)	65.3 (40.5)	65.2 (32.7)	64.5 (38.2)	70.9 (35.8)	*NS*
Na (mmol/L)	139 (8)	139 (7)	138 (12)	139 (5)	*NS*
K (mmol/L)	4.7 (0.4)	4.7 (0.5)	4.1 (0.5)	4.4 (0.7)	*NS*
AST (UI/L)	31.3 (8–1871)	31.6 (9–1871)	31.5 (8–591)	30.3 (12–88)	*NS*
ALT (UI/L)	26.7 (4–2065)	27.3 (4–2065)	27.3 (4–799)	22.5 (6–118)	*NS*
GGT (UI/L)	90.7 (1–709)	80.9 (7–709)	105.6 (8–619)	117.3 (10–522)	*<.005**
AP (UI/L)	109.6 (12–672)	103.7 (12–672)	117.5 (26–513)	132 (39–431)	*<.005**
Total bilirubin (mg/dl)	2.3 (1–16)	2.1 (1–16)	2.81 (1–11)	1.2 (0.8–3.3)	*NS*
Nt-ProBnp (pg/ml)	4733 (5–259 000)	4956 (5–259 000)	4607 (50–98 419)	3515 (221–18 606)	*NS*
Fe (mcg/dL)	67 (8–558)	65 (8–255)	72 (11–558)	72.6 (21–285)	*NS*
Transferrin (mg/dL)	259 (12–778)	254 (12–499)	267 (59)	273.6 (92.4)	*NS*
TS (%)	20 (13)	19.8 (11.6)	19.8 (12.3)	23.7 (11.2)	*NS*

ALT, alanine aminotransferase; AP, alkaline phosphatase; AST, aspartate aminotransferase; GFR, glomerular filtration rate; GGT, gamma-glutamil transferase; TR, tricuspid regurgitation; TS, transferrin saturation.
*Significant between severe TR and torrential TR.
The values in italics are intended solely to highlight statistical significance.

Table 9 Univariable and multivariable predictors of cardiovascular mortality

	Univariable		Multivariable	
	HR (95%CI)	***P* value**	**HR (95%CI)**	***P* value**
Age	1.070 (1.043–1.098)	<0.001	1.062 (1.029–1.096)	<.001
Gender (male)	1.210 (0.803–1.825)	0.362		
CKD	2.124 (1.394–3.236)	<0.001	1.613 (0.983–2.648)	.059
NYHA III–IV	2.476 (1.652–3.710)	<0.001	1.380 (0.785–2.249)	.314
Previous HF admission	2.444 (1.652–3.616)	<0.001	1.956 (1.194–3.204)	.008
Severe PHT	1.677 (1.088–2.587)	0.019	1.179 (0.635–2.192)	.602
Grade TR				
Severe	Ref		Ref	
Massive	1.055 (0.658–1.693)	0.823	1.726 (1.022–2.915)	.041
Torrential	1.658 (0.942–2.917)	0.079	2.575 (1.240–5.346)	.011
TR aetiology	1.262 (0.73–2.18)	0.643		
VAC ≤ 0.31	1.747 (1.116–2.733)	0.015		
Haddad classification Stages 3–4	1.559 (0.217–11.174)	0.659		
Proposed classification Stages III–IV	1.932 (1.310–2.849)	0.001	2.011 (1.270–3.182)	.003
Tricuspid intervention	0.414 (0.169–1.018)	0.055	0.159 (0.022–1.162)	.070
Furosemide dose >160 mg/day	2.87 (1.83–4.49)	<0.001	4.49 (1.06–19.15)	.042

CKD, chronic kidney disease; CI, confidence Interval; HF, heart failure; HR, hazard ratio; NYHA, New York Heart Association; PHT, pulmonary hypertension; TR, tricuspid regurgitation; VAC, ventriculo-arterial coupling.
Gronesby–Borgan test, $P = .795$; C-Index 0.742 [0.685–0.798].

patients in this series and is associated with permanent AF and TA dilation (18–20). These patients are older than those with other TR aetiologies (mean age: 80.4 (7.7) years; $P < .05$), which, together with a high presence of comorbidities, makes them less candidates for surgery and more suitable for transcatheter repair techniques.[20,21] It is important to mention that aetiology was not a predictor of mortality in the multivariable analysis, but in the univariable analysis, A-STR was associated with mortality ($P = .03$).

We included all patients with severe A-STR, of all ages, and our results support their bad prognosis, just as poor as other forms of TR. In contrast to previous studies, atrial TR in our cohort showed a prognosis comparable to ventricular secondary TR. This discrepancy may be explained by the older age, higher burden of comorbidities, and the exclusive inclusion of severe forms of TR in our registry. Notably, 22% of patients with atrial TR presented with torrential TR, a proportion substantially higher than previously reported,

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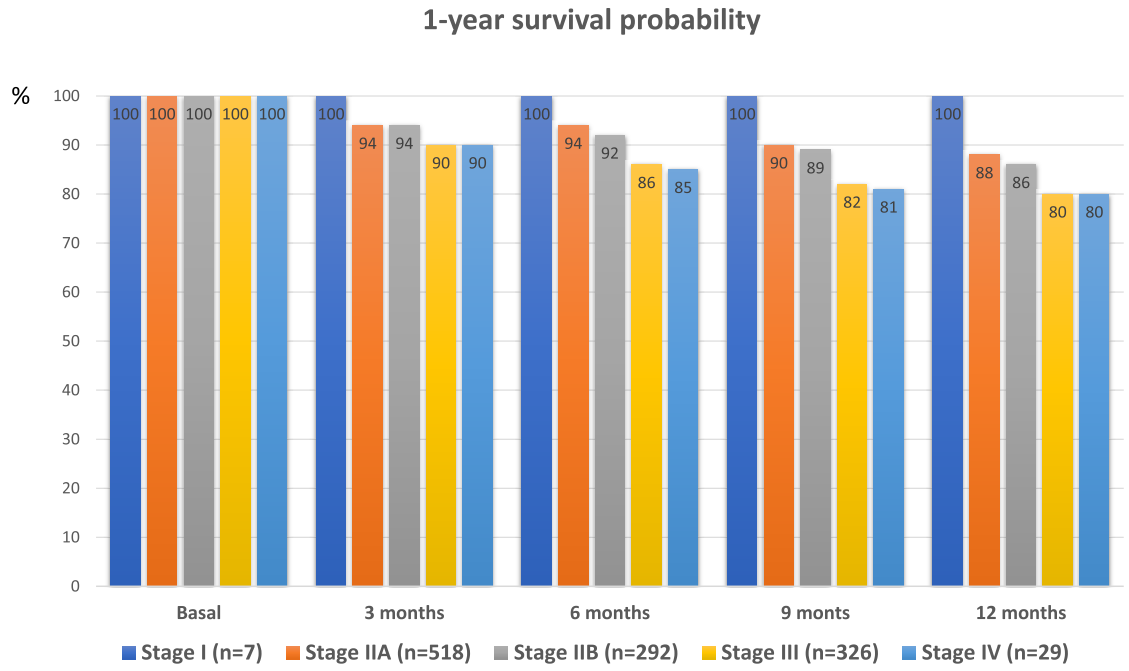

Figure 2 Cardiovascular mortality analysis and Kaplan–Meier curves depending on the proposed staging system. Restricted range from 0.8 to 1.0

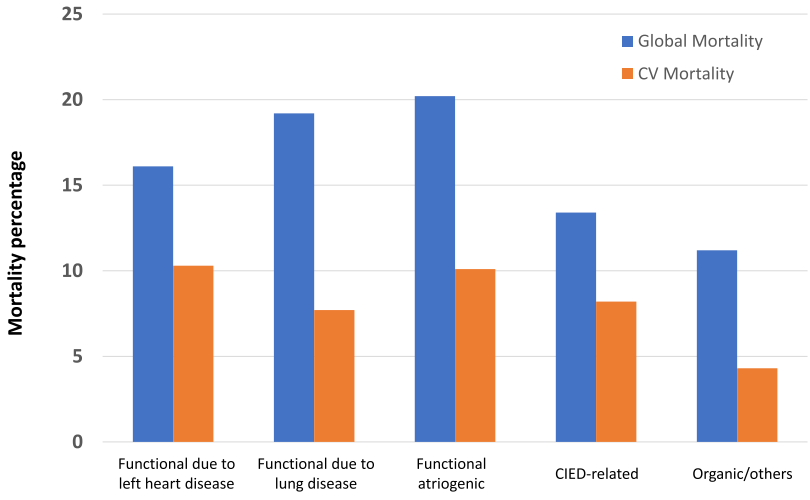

Figure 3 Mortality analysis depending on TR aetiology. CV: cardiovascular; TR, tricuspid regurgitation.

which likely contributes to their adverse outcomes. We believe that the difference with previous studies regarding the prognosis of A-STR lies in the fact that most of them are retrospective and include non-severe forms of A-STR. Furthermore, in the literature, there are previous studies showing a worse prognosis in patients with A-STR.[19,21]

Because PASP estimation is often unreliable in patients with torrential TR, some degree of misclassification between A-STR and V-STR cannot be excluded. This limitation is particularly relevant given that 22% of A-STR patients presented with torrential TR.

Regarding echocardiographic findings, when comparing patients with massive and torrential TR with those with severe TR, the former had a more dilated RV and TA, but there were no differences in the parameters (TAPSE, S′ wave, FAC, and VAC) that classically evaluate RV systolic function. It should be noted that TAPSE paradoxically had higher values in patients with torrential TR (*Table 4*). It is a volume-dependent parameter and does not mean that these patients have better systolic function, but rather that it is the result of the decrease in afterload in torrential TR. Impaired RV free wall longitudinal strain was more frequently found in patients with torrential TR than in those with severe and massive TR.

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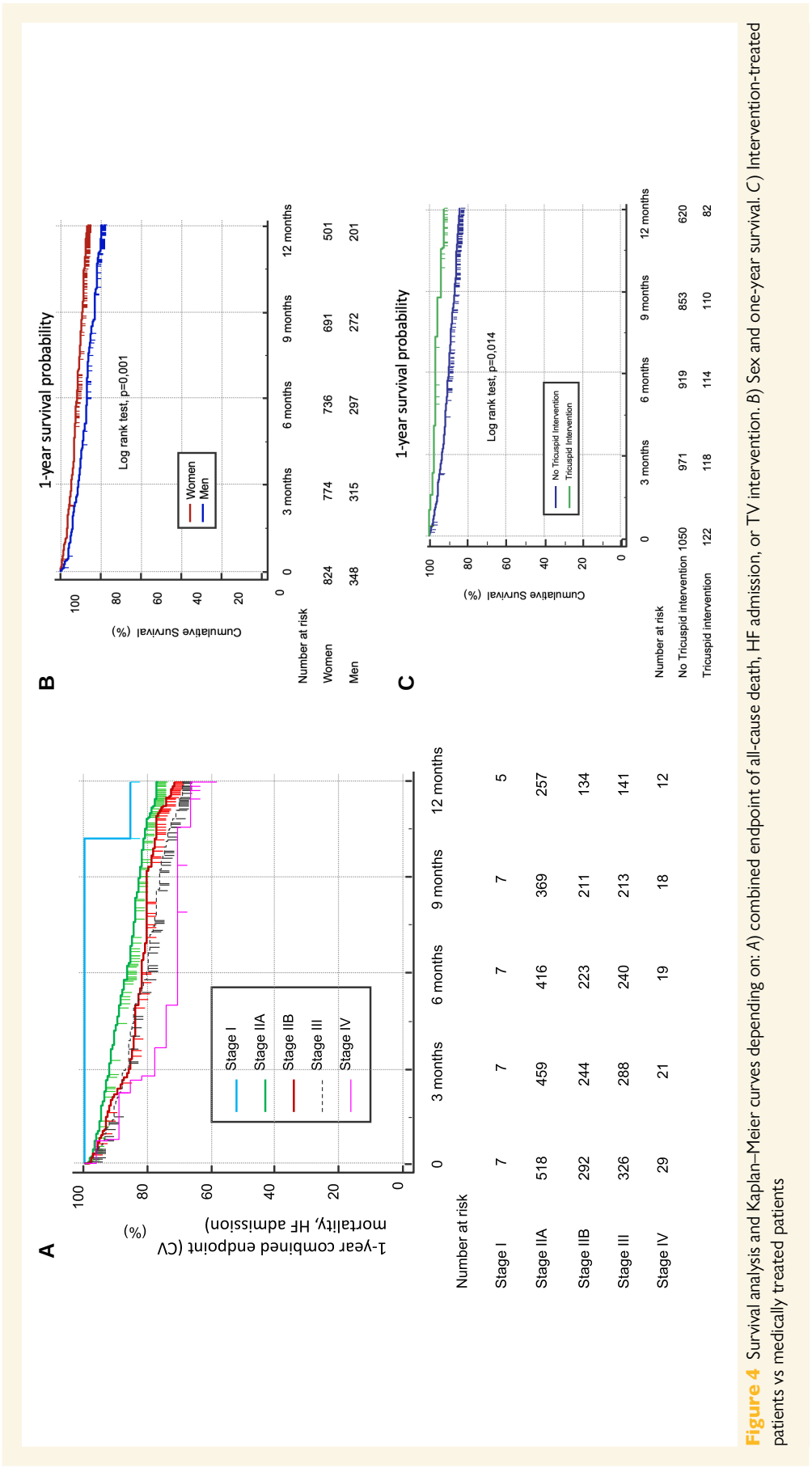

Figure 4 Survival analysis and Kaplan–Meier curves depending on: *A*) combined endpoint of all-cause death, HF admission, or TV intervention. *B*) Sex and one-year survival. *C*) Intervention-treated patients vs medically treated patients

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Table 10 Univariable and multivariable predictors of heart failure admission

	Univariable		Multivariable	
	HR (95%CI)	*P* value	HR (95%CI)	*P* value
Age	1.034 (1.017–1.051)	<.001	1.020 (1.000–1.040)	.045
Gender (male)	1.317 (0.975–1.778)	.072	1.301 (0.910–1.861)	.148
CKD	1.770 (1.279–2.451)	.001	1.424 (0.988–2.053)	.058
NYHA III–IV	2.703 (2.013–3.631)	<.001	1.490 (1.043–2.129)	.029
Previous HF admission	2.534 (1.899–3.382)	<.001	1.968 (1.359–2.851)	<.001
Severe PHT	2.175 (1.590–2.974)	<.001	1.481 (1.041–2.107)	.029
Grade TR				
Severe	Ref			
Massive	1.254 (0.900–1.748)	.182		
Torrential	1.714 (1.125–2.612)	.012	1.281 (0.690–2.377)	.432
Hb < 10 g/dl	2.278 (1.572–3.301)	<.001	1.836 (1.209–2.788)	.004
VAC ≤ 0.31	2.061 (1.484–2.863)	<.001	1.026 (0.650–1.619)	.913
Haddad classification Stages 3–4	3.051 (0.427–21.769)	.266		
Proposed classification Stages III–IV	1.640 (1.226–2.194)	.001	1.589 (1.131–2.231)	.008
Tricuspid intervention	1.182 (0.764–1.828)	.453		

CKD, chronic kidney disease; CI, confidence interval; HR, hazard ratio; HF, heart failure; HB, haemoglobin; NYHA, New York Heart Association; PHT, pulmonary hypertension; TR, tricuspid regurgitation; VAC, ventriculo-arterial coupling.
Gronesby–Borgan test, $P = .100$; C-Index 0.690 [0.651–0.730].

Both TAPSE and the S' wave may be falsely elevated if the left ventricle is hyperdynamic.[22] Our findings confirm the incremental value of RV free wall longitudinal strain for the detection of RV systolic dysfunction.[13]

The series of Dietz et al.,[6] included more patients with moderate (79%) TR than severe TR. Those patients in more advanced stages had a worse prognosis and greater surgical risk. In our study, prognosis was also worse among the more severe degrees of TR.

As in patients with left heart failure, prognostic staging has been proposed in patients with RHF.[10] This staging combines the presence of RV dysfunction with the existence of RHF signs and symptoms. It is based on the progressive nature of the disease in the presence of risk factors such as TR. In the herein series, classic four RHF staging failed to stratify patients and was not a predictor of mortality or HF-admission. Patient stratification was not much better in the series of Dietz et al.,[6] 83% of patients were in Stages 3 and 4. Surprisingly, there were no significant differences in staging distribution between moderate and severe TR.[6]

To achieve a better patient stratification and knowing that RV remodelling occurs before RV systolic dysfunction,[11] patients were classified into five stages (stage II was divided into IIA and IIB) instead of four. It is notorious that many patients with severe TR are in Stage IIA (no RV dilatation or systolic dysfunction, but with symptoms/signs of RHF or taking diuretics). The Stages IIA and IIB differ in the presence or absence of right ventricular dilation. In our opinion, this difference is conceptually important when discussing patients with severe tricuspid regurgitation, where significant volume overload occurs. We believe it is important to consider the presence or absence of right ventricular dilation in this analysis.

Thus, many patients with RHF and on diuretic treatment will not have RV dilation since the RV may remain well adapted to right-sided valvular regurgitant lesions for extended periods of time.[11] RHF is a clinical diagnosis with signs and symptoms of systemic venous congestion in combination with functional or structural RV abnormalities.[23] Therefore, it is imperative to carry out an exhaustive study of RV diameters and function, as well as an exquisite physical examination to detect patients in the early stages of the disease.[24]

This new staging system not only provides a better patient stratification but, unlike the Haddad et al classification,[10] also showed a linear association with mortality and new HF hospital admissions. In the multivariate analysis, only Stages III and IV (which include patients with right ventricular systolic dysfunction) were found to be significant. Therefore, the data obtained in this study indicate that the key factor is the presence of systolic dysfunction.

Stratification of patients into five stages could be especially relevant to detect RV remodelling in time. The appropriate time to intervene on a patient with severe TR is not well established, but progressive RV dilation (before systolic dysfunction occurs), together with the presence of signs of systemic venous congestion could be sufficient to consider intervention in most patients.[23] The potential prognostic impact of this new RHF staging of patients with severe TR has been documented during patient follow-up, becoming an independent predictor of CV mortality and HF hospital admission (*Graphical Abstract*, *Tables 9 and 10*) in patients with severe TR.

TV interventions were mostly performed in Stages IIA, IIB, and III. There are limited data addressing the role of TV interventions on the outcomes of patients with severe TR.[23] Based on our results, it appears that patients with severe TR might benefit from timely interventions. Further studies are needed to identify the patient profile that will benefit most from both surgical procedures and catheter-based treatment options.

Study limitations

First, although all the participating cardiac imaging units have adhered to the quantitative parameters established to determine the severity of TR and to the predetermined classification to define its aetiology, interobserver variability might be present.

In this study, SPAP was estimated by Doppler echocardiography through the maximum velocity of the TR jet using continuous wave Doppler. In patients with torrential TR, Doppler-based PASP estimation may be inaccurate, especially in the A-STR subgroup, where 22% of patients had torrential TR. Therefore, some degree of aetiologic misclassification cannot be ruled out. The inaccuracy in calculating SPAP by

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continuous Doppler in patients with torrential TR might partially explain the low ratio of severe PHT in these patients.[9] This also applies to the estimation of VAC.

As TR is a highly load-dependent valvular disease, some patients may have been assigned to a different grade of severe TR based on whether they are taking diuretics or have volume overload. The diagnosis of RHF is clinical and, therefore, carries a certain degree of subjectivity.

The new proposed RHF staging requires external validity.

Conclusions

This is a large prospective observational study that tries to assess the prevalence and characteristics of patients with severe TR. Severe TR is a prevalent valve disease, especially in older women. The most common aetiology is V-STR to left heart disease, followed by A-STR. Up to 44% of patients have signs or symptoms of RHF or take diuretics and have not undergone RV remodelling. Classic, volume-dependent methods to assess RV systolic function are not trustworthy in severe TR. One-third of patients are in the most severe stages (III and IV) of the disease and mortality was high (14,9%) at 1-year follow-up. The new RHF staging system stratifies patients into prognostic categories, and it will be of value in the design of forthcoming clinical trials to ascertain the proper time to treat these patients.

What is known about the topic?

There is limited understanding of the prevalence, natural history, and aetiology of tricuspid regurgitation, traditionally assessed by retrospective and heterogeneous studies.

What does this study add?

This contemporary and prospective registry will provide relevant clinical and morphological information on this prevalent disease according to the current classifications. The modified staging system of right heart failure could better stratify patients with severe tricuspid regurgitation.

Supplementary data

Supplementary data are available at *ESC Heart Failure* online.

Author contributions

Juan Carlos Gómez Polo (Conceptualization; Data curation; Formal analysis; Investigation; Methodology; Project administration; Resources; Software; Supervision; Validation; Visualization; Writing—original draft; Writing—review & editing), Pedro Morillas (Investigation), Daniel Gaitán Román (Investigation), María Victoria Mogollón (Investigation), Ana Payá Chaume (Investigation), Alberto Bouzas Mosquera (Investigation), Jana Pérez (Investigation), Jessica Sánchez Quiñones (Investigation), Angel López Cuenca (Investigation), Elvis Amao (Investigation), José Carlos Fernández Camacho (Investigation), Teresa Blasco (Investigation), Ildefonso Roldán (Investigation), Catherine Graupner (Investigation), Carmen Manzano (Investigation), Amparo Martínez Monzonis (Investigation), Alex Ruiz Majoral (Investigation), Luca Vannini (Investigation), Mario Baquero (Investigation), Lizandro Rodríguez (Investigation), Fernando Cabrera (Investigation), Maria Jesús Rollán (Investigation), Raúl Reyes (Investigation), Cristina Fernández Pérez (Data curation; Formal analysis; Investigation), Pilar Martín de la Fuente (Investigation), Samuel Del Castillo (Investigation), Ariana González Gómez (Investigation), Teresa Romero Delgado (Investigation), Itziar Gómez-Salvador (Data curation; Formal analysis), Patricia Mahía Casado (Conceptualization; Investigation; Methodology), Esther Pérez David (Conceptualization; Investigation), Pere Pericàs (Conceptualization; Investigation), Jose Antonio Parada Barcia (Investigation), Carlos Izurieta (Investigation), Ana Revilla Orodea (Investigation), Vanessa Moñivas (Investigation), Ana Testa (Investigation), Lara Miguel (Investigation), Irene Carrión Sánchez (Investigation), José Alberto San Román (Conceptualization; Investigation; Methodology; Supervision; Validation; Writing—review & editing), Laura Gutiérrez (Investigation), Nuria Hernández Vicente (Investigation), Lucía Torres Quintero (Investigation), Olatz Zaldua Irastorza (Investigation), Mónica Delgado Ortega (Investigation), Ana Osa Sáez (Investigation), María Victoria Millán (Investigation), María de los Ángeles Pérez (Investigation), Almudena Aguilera (Investigation), Isaac Pascual (Investigation), Irene Méndez Santos (Investigation), Marc Abuli Lluch (Investigation), José Ramón Ortega Trujillo (Investigation), and Isidre Vilacosta (Conceptualization; Investigation; Methodology; Project administration; Supervision; Validation; Writing—original draft; Writing—review & editing).

Declarations

Disclosure of Interest

All authors declare no disclosure of interest for this contribution. The data underlying this article are available in the article and in its online supplementary material.

Data Availability

No data were generated or analysed for this manuscript.

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Ethical Approval

Ethical approval was not required.

Pre-registered Clinical Trial Number

None supplied.

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