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Association between CMR-derived hepatic T1-time, tricuspid regurgitation and survival

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Abstract

Background: Cardiac magnetic resonance (CMR) derived hepatic T1-time is associated with outcome. However, the interplay between tricuspid regurgitation (TR), which can cause congestive hepatopathy and liver T1-time is unclear.

Methods: We measured hepatic T1-time in CMR all-comers, who underwent echocardiography within 3 weeks of CMR. Kaplan–Meier estimates and Cox regression models were used to investigate the association between hepatic T1-time, TR severity and a composite endpoint of heart failure hospitalisation and all-cause death.

Results: 1029 participants (67 ± 17 y/o, 44% female) had a mean hepatic T1-time of 605 ± 79 ms. Overall, 41% (417) presented with non/trace, 38% (391) with mild, 13% (135) with moderate and 8% (85) with severe/massive/torrential TR. Liver T1-time was significantly associated with TR severity (no/trace: 586 ± 72 ms; mild: 601 ± 74 ms; moderate: 634 ± 84 ms; severe/massive/torrential: 665 ± 83 ms; $\beta = 25.4$ ms, [95% CI:19.7–31.2, $p < .001$]). After adjustment for serum NT-proBNP and right ventricular function in a linear regression model, TR severity remained significantly associated with hepatic T1-time ($p < .001$). During follow-up (mean 53 ± 36 months) 326 (32%) events occurred. Hepatic T1-time (adj.HR 1.69 [95% CI: 1.49–1.92] per 100 ms increase, $p < .001$) and TR (adj.HR 1.66 [95% CI: 1.49–1.84], $p < .001$) were both associated with outcome. Even after adjustment for serum NT-proBNP, cardiac structure and function, age, sex and TR severity, hepatic T1-time remained significantly associated with event-free survival (adj.HR 1.42 [95% CI: 1.20–1.68] per 100 ms increase, $p < .001$).

Conclusion: TR exerts a notable influence on hepatic T1-time. Nevertheless, after adjustment for serum NTproBNP, cardiac function and TR severity, hepatic T1-time still independently predicts outcomes. This underscores the importance of hepatic T1-time both as a marker of TR and prognosis.

KEYWORDS
CMR, hepatic-T1 time, T1-time, tricuspid regurgitation

1 | BACKGROUND

Liver damage is frequently observed in patients with cardiovascular disease. Hepatic T1-time derived from standard cardiovascular magnetic resonance (CMR) maps has been shown to predict clinical outcomes.[1] However, up to date, the various causes contributing to changes in CMR-derived hepatic T1-time are not fully understood.[2] Strong associations with liver congestion and hepatic fibrosis have been demonstrated.[2,3]

The increased pressure in the hepatic veins promoted by tricuspid regurgitation (TR) causes liver congestion and, consecutively, liver dysfunction.[4] Research has shown that patients with severe TR exhibit increased liver stiffness, as measured by transient elastography.[5] Moreover, *Model for End-Stage Liver Disease* (MELD) scores have been observed to significantly improve following TR reduction in patients with liver cirrhosis.[4]

The challenge of accurately assessing liver dysfunction in patients with TR lies in the fact that significant liver injury can manifest before any abnormalities appear in serum liver biochemistry. Patients with heart failure (HF) experience elevated hepatic venous pressures and congestion, leading to liver cell damage over time.[6] In non-congenital HF, there is no reliable data on the prevalence and natural history of congestive hepatopathy (CH), with even fewer solid data concerning any stage of liver disease.[7]

CMR is the current gold standard for assessing cardiac size and function.[8,9] T1-mapping further enables the detection of fibrosis, fat, myocardial infiltration (such as amyloid), edema and inflammation and iron overload conditions like hemochromatosis, and has found its way into daily clinical practice. On standard cardiac T1-maps, a segment of liver tissue is almost always depicted. To date, only a few studies have investigated the use of T1-mapping to assess liver tissue.[10,11] These studies showed a strong association of hepatic T1-time with both congestion and hepatic fibrosis,[12–14] as well as clinical outcome.[2,15]

In the present study, we investigated the impact of TR on hepatic T1-time and its prognostic implications. Changes in hepatic T1-time could serve as an early imaging biomarker for HF as well as valvular heart disease.

2 | METHODS

2.1 | Study design

This post-hoc analysis of a prospective registry study included consecutive all-comers for CMR, who underwent echocardiography within 3 weeks of CMR between January 2013 and January 2023. The study was carried out at the Vienna General Hospital, a university-affiliated tertiary care centre with a high-volume multimodality-imaging facility. All participants gave written informed consent, and the Institutional Review Board approved the study protocol (EK #2036/2015). All patients were stable without the need for intravenous diuretic treatment for at least 3 months prior to the CMR scan. In addition, patients with chronic viral hepatitis, autoimmune liver disease, liver cirrhosis or hereditary liver disease (e.g. hemochromatosis) were excluded. Due to overlapping metabolic risk factors, patients with metabolic dysfunction-associated steatotic liver disease (MASLD) were not excluded. Patients with excessive alcohol consumption were excluded.

2.2 | Patient population

At the time of CMR, demographic data (age, sex, body mass index [BMI], body surface area [BSA]) and comorbidities were assessed. These included hypertension (≥140/90 mmHg or antihypertensive treatment), atrial fibrillation (AF, present at the time of CMR or documented in the medical history), diabetes (fasting blood glucose level >126 mg/dL, HbA1c ≥ 6.5%, or use of anti-diabetic medication), hyperlipidemia (total serum cholesterol 240 mg/dL or use of cholesterol-lowering medication), chronic obstructive pulmonary disease (COPD, documented in the medical history), peripheral artery disease (PAD, documented in the medical history), chronic kidney disease (CKD, eGFR <40 mL/min, MRDR formula) and previous myocardial infarction, which was defined both by medical history and CMR. In addition, we conducted blood tests with the most important cardiac enzyme parameters before CMR (including NT-pro BNP).

2.3 | Cardiac magnetic resonance imaging

All patients underwent the CMR examination on a 1.5-T scanner (Avanto Fit, Siemens Healthineers, Erlangen, Germany) following standard protocols that included LGE imaging [.15 mmol/kg gadobutrol (Gadovist; Bayer Vital GmbH, Leverkusen, Germany)] if the estimated glomerular filtration rate was >30 mL/min/1.73 m^2. At the time of insertion of the intravenous cannula, blood was drawn for haematocrit and serum creatinine measurement. For the determination of LGE and other measurements, a semiautomatic approach was used, applying dedicated software (cmr42, Circle Cardiovascular Imaging Inc., Calgary, Alberta, Canada). A threshold of five standard deviations (SD) above the mean signal intensity of the reference myocardium was defined.

Electrocardiographically triggered Modified Look-Locker Inversion Recovery (MOLLI) using a 5(3)3 prototype (five acquisition heartbeats followed by three recovery heartbeats and further three acquisition heartbeats) was applied for pre-contrast T1-mapping. This method generates an inline, pixel-based T1-map by acquiring a series of images over several heartbeats with shifted T1-time, inline motion correction and inline calculation of the T1 relaxation curve within one breath-hold. T1 sequence parameters were as follows: starting inversion time 120 ms, inversion time increment 80 ms, reconstructed matrix size 256 × 218 and measured matrix size 256 × 144 (phase-encoding resolution 66% and phase-encoding field of view 85%). T1-maps were created both before and 15 min after contrast agent application. For post-contrast T1-mapping, a 4(1)3(1)2 prototype was used.

In addition to standard cardiac T1-mapping, regions of interest were defined in the liver parenchyma, avoiding the great hepatic vessels. The liver was depicted in the short axis view frontally. T1-values were assessed in 4 regions of interest (Figure 1). For inter-reader variability, a second reader blinded to previous results measured hepatic T1-time in a randomly chosen sample of 100 patients. Intraclass correlation coefficient (ICC) was calculated.

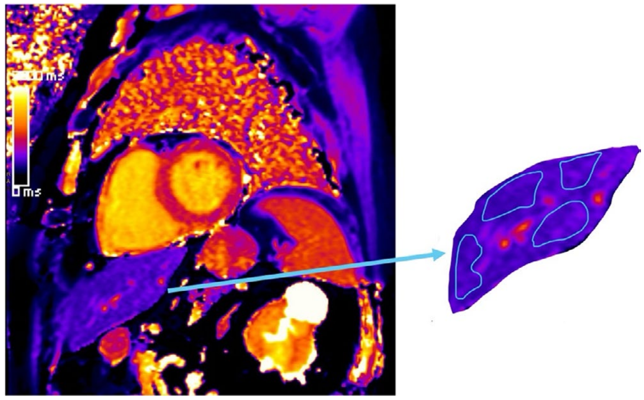

FIGURE 1 Standard cardiac modified Look-Locker inversion recovery sequence derived T1 maps were used to measure liver T1-time. The midventricular short-axis slice and myocardial region of interest were traced manually. For the liver, we chose four regions of interest and calculated the mean value of these areas.

2.4 | Echocardiography

All patients underwent a standard transthoracic echocardiogram by board-certified cardiologists using commercially available equipment (Vivid E95, GE Healthcare, Acuson Sequoia, Siemens). Cardiac morphology was assessed using standard parasternal long-axis, short-axis and apical 4- and 2-chamber views, in accordance with current recommendations.[16] Tricuspid regurgitation was graded according to the 2020 ACC/AHA guidelines using an integrated approach, which included assessment of vena contracta width, hepatic vein flow, jet area and right heart chamber dimensions and was graded as none, mild, moderate, severe, massive and torrential.[17] Quantitative measurements such as effective regurgitant orifice area (EROA) or regurgitant volume were not systematically available. The aetiology of TR (e.g., atrial, ventricular or device-associated) was not routinely classified. Grading was performed by board-certified cardiologists as part of routine clinical care.

2.5 | Outcome

The primary endpoint was defined as a composite of HF hospitalization and all-cause death. We retrieved dates and causes of death from in-hospital charts, the national death registry and nationwide medical records. Two study team members (BP and CN) served as the adjudication committee for each event and were blinded to CMR and echocardiography data.

2.6 | Statistical analysis

All statistical analyses were performed using Stata SE (Stata 18). Continuous variables are shown as mean ± standard deviation. Categorical variables are presented with both absolute and relative frequencies and are

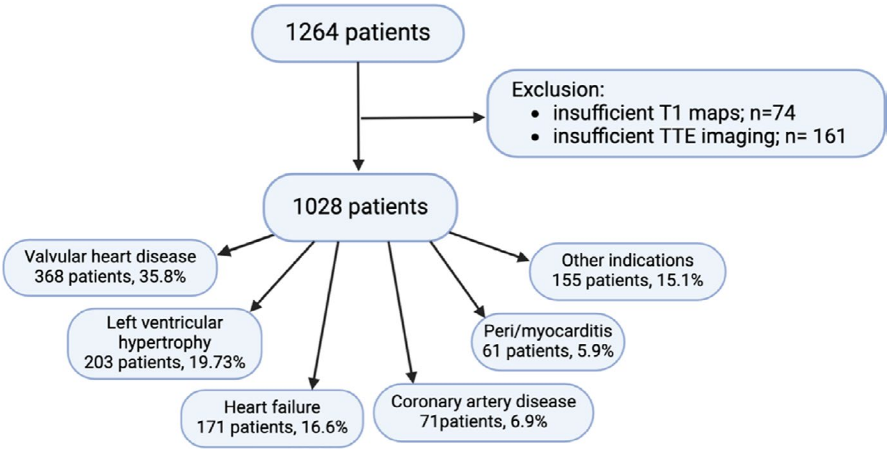

FIGURE 2 Demonstrates in a flowchart the patient population and CMR indications.

visualised by bar charts. Metric variables are expressed as arithmetic mean and standard deviation or median with corresponding interquartile range. Group-wise comparisons were performed using the student *t*-test, Fisher exact test, Kruskal–Wallis test and one-way analysis of variance. Additionally, linear regression models were used to analyse the association between hepatic T1 time and continuous variables such as NT-proBNP and ventricular parameters. Logistic regression was used to assess associations between hepatic T1 time and categorical clinical variables such as pre-existing conditions, including atrial fibrillation and COPD. For all analyses, the level of significance was set to .05.

Survival and freedom from death and hospitalization for HF are displayed using the Kaplan–Meier method and compared using the log-rank test. Correlation and regression models were used to test the association between hepatic T1-time and clinical as well as imaging variables. Kaplan–Meier estimates and Cox regression models were used to investigate the association between hepatic T1-time, TR and the primary endpoint. We chose a predefined model for multivariable testing based on existing literature[18,19] adjusted for age, sex, NT-proBNP values (in 1000 mg/dL increase) and ventricular function.

3 | RESULTS

Figure 2 shows the study flow. The mean age was 67 ± 17 years, and 44% of participants were female. The mean overall liver T1-time was 605 ± 79 ms. Inter-observer reproducibility was excellent for hepatic T1-time in a subset of 100 patients (ICC .882). Out of the 1029 patients, 417 (41%) had no/trace TR, 391 (38%) mild, 135 (13%) moderate and 85 (8%) severe, massive and torrential TR.

3.1 | Association of liver T1-time with baseline characteristics

Baseline characteristics, stratified by TR severity (<moderate and ≥moderate) and hepatic T1-time (divided by the median of 594 ms) are displayed in Table 1.

TR severity was significantly associated with hepatic T1 time on age and sex-adjusted linear regression (adj. beta = 25.58, $p < .001$). Additionally, hepatic T1 time was associated with age (adj.beta = 1.04; $p = .0012$), but not sex (adj.beta = −6.71, $p = .18$) (Table 2).

Logistic regression adjusted for age and sex revealed a significant association between hepatic T1-time and atrial fibrillation (adj.beta = .015, $p < .001$); COPD (adj.beta = .24, $p = .025$); chronic kidney disease (adj.beta = .17, $p = .032$); and hyperlipidemia (adj.beta = −.162, $p = .06$) (depicted in Table 2).

Significant correlations were observed between hepatic T1-time and serum parameters including alkaline phosphatase (adj.beta = .45, $p < .001$); aspartate aminotransferase (AST) (adj.beta = .23, $p = .046$); gamma-glutamyl transferase (gGT) (adj.beta = .19, $p < .001$); NT-proBNP values (adj.beta = .002, $p < .001$); INR (adj. beta = 38.01, $p < .001$); and bilirubin (adj.beta = 34.52, $p < .001$).

Of the 1028 patients, 93 (8%) were diagnosed with cardiac amyloidosis. Patients with cardiac amyloidosis presented with significantly higher liver T1 time than the remainder (671 ± 102 ms versus 601 ± 74 ms, $p < .001$).

The estimated systolic pulmonary artery pressure (sPAP) on echocardiography was significantly associated with both hepatic T1-time (adj.beta = .19, $p < .001$) and TR severity (adj.beta = .10, $p = .012$) on age and sex-adjusted regression.

TABLE 1 Baseline characteristics, stratified by hepatic T1-time and TR.

	Total *N* = 1029	T1-time <594 ms, TR < moderate *N* = 450	T1-time <594 ms, TR ≥ moderate *N* = 63	T1-time ≥594 ms, TR < moderate *N* = 359	T1-time ≥594 ms, TR ≥ moderate *N* = 157	*p*-Value
Age (years)	66.7 ± 16.7	63.9 ± 16.8	75.3 ± 8.8	65.5 ± 17.8	73.8 ± 12.3	**<.001**
Female sex (%)	44.12	39.78	61.90	40.95	56.69	**<.001**
BSA	1.91 ± .23	1.93 ± .22	1.84 ± .22	1.91 ± .23	1.87 ± .25	**.008**
BMI (kg/m^2)	26.8	26.8	26.9	26.6	27.1	.72
Referral diagnosis						
VHD (%)	35.76	34.89	34.54	44.44	37.58	**<.001**
Storage disease (%)	19.73	19.11	22.84	12.70	17.20	
HF (%)	16.62	16.67	12.81	15.87	25.48	
CAD (%)	6.90	9.33	7.24	.00	1.91	
Inflammation (%)	5.93	5.56	8.91	1.59	1.91	
Others (%)	15.06	14.44	13.65	25.40	15.92	
Comorbidities						
Previous myocardial infarction (%)	28.47	25.78	25.40	32.59	28.03	.18
Atrial fibrillation (%)	24.88	13.78	36.51	23.40	55.41	**<.001**
Hypertension (%)	56.85	56.00	57.14	56.82	59.24	.92
Diabetes mellitus (%)	17.49	14.44	22.22	18.38	22.29	.087
Chronic obstructive lung disease (%)	6.61	4.22	4.76	8.08	10.83	**.016**
Hyperlipidemia (%)	30.81	32.67	30.16	30.92	25.48	.42
PAD (%)	1.07	.44	.00	1.95	1.27	.17
Chronic kidney disease (%)	13.31	10.00	12.70	16.16	16.56	**.041**
Blood parameters						
eGFR, mL/min/1.73 m^2	69.68 ± 30.84	74.15 ± 31.14)	58.29 ± 25.40)	71.45 ± 33.18)	59.55 ± 22.91)	**<.001**
Haematocrit, %	39.09 ± 5.40	40.31 ± 5.22)	38.20 ± 5.99)	38.37 ± 5.27)	37.59 ± 5.25)	**<.001**
Platelet count, g/L	230.62 ± 82.10	224.85 ± 73.81)	212.20 ± 66.17)	242.40 ± 87.25)	227.30 ± 92.93)	**.013**
INR	1.20 ± .42	1.10 ± .27)	1.23 ± .49)	1.19 ± .41)	1.40 ± .56)	**<.001**
Bilirubin mg/dL	.68 ± .59	.58 ± .41)	.69 ± .32)	.65 ± .45)	.97 ± 1.04)	**<.001**
Albumin, g/L	40.99 ± 5.11	42.17 ± 4.88)	41.41 ± 4.45)	39.75 ± 5.62)	40.54 ± 4.02)	**<.001**
Cholinesterase	6.86 ± 2.06	7.54 ± 1.99)	6.56 ± 2.02)	6.55 ± 2.05)	5.94 ± 1.74)	**<.001**

(Continues)

TABLE 1 (Continued)

	Total *N* = 1029	T1-time <594 ms, TR < moderate *N* = 450	T1-time <594 ms, TR ≥ moderate *N* = 63	T1-time ≥594 ms, TR < moderate *N* = 359	T1-time ≥594 ms, TR ≥ moderate *N* = 157	*p*-Value
AP, U/L	80.73 ± 46.05	70.64 ± 29.62)	85.48 ± 52.30)	84.07 ± 52.67)	96.88 ± 55.77)	**<.001**
AST, U/L	29.25 ± 23.82	28.25 ± 20.39)	28.13 ± 12.15)	30.35 ± 30.24)	29.90 ± 19.67)	.69
ALT, U/L	28.30 ± 27.73	29.81 ± 28.64)	25.85 ± 16.10)	28.36 ± 31.70)	25.39 ± 18.51)	.39
gGT, U/L	62.24 ± 90.25	44.08 ± 66.07)	80.54 ± 116.67)	64.73 ± 92.49)	94.50 ± 112.69)	**<.001**
NT-proBNP, pg/mL	2976.77 ± 5929.06	1613.02 ± 4132.27)	4723.54 ± 8408.09)	3713.09 ± 6910.93)	4044.91 ± 5669.44)	**<.001**
HbA1C, %	5.81 ± .83	5.67 ± .80)	5.96 ± .74)	5.90 ± .93)	5.89 ± .71)	**.008**
Total Cholesterol, mg/dL	164.24 ± 47.66	173.59 ± 45.10)	160.98 ± 43.32)	160.17 ± 51.51)	151.61 ± 42.86)	**<.001**
Hämoglobin	12.83 ± 2.01	13.33 ± 1.91)	12.25 ± 2.25)	12.59 ± 1.99)	12.31 ± 1.92)	**<.001**
CRP, mg/dL	.94 ± 1.73	.66 ± 1.26)	1.75 ± 3.69)	1.09 ± 1.72)	1.17 ± 1.66)	**.003**
Concomitant medication						
Betablocker (%)	61.14	55.88	59.26	60.69	79.69	**.009**
ACE-Inhibitors (%)	31.58	30.39	22.22	34.48	32.79	.61
Angiotensin-Receptor-Inhibitors (%)	27.52	25.37	22.22	27.03	37.31	.25
Calcium-Inhibitors (%)	21.00	20.69	14.81	20.69	25.40	.71
T-ASS (%)	36.47	37.75	33.33	39.58	26.23	.31
Coumadin (%)	15.94	10.84	29.63	12.59	35.00	**<.001**
NOACS (%)	19.32	12.81	25.93	17.24	41.54	**<.001**
Diuretics (%)	43.67	33.01	48.15	42.76	78.12	**<.001**

Note: Bold values indicate $p \leq .05$.

Abbreviations: BSA, body surface area; CAD, coronary artery disease; CMR, cardiac magnetic resonance tomography; HF, heart failure; IVS, intraventricular septum; LA, left atrium; LV, left ventricular; LVCO, left ventricular cardiac output; LVEDV, left ventricular end-diastolic volume; LVEF, left ventricular ejection fraction; LVESV, left ventricular end-systolic volume; LVSV, left ventricular stroke volume; RA, right atrium; RVCO, right ventricular cardiac output; RVEDV, right ventricular end-diastolic volume; RVEF, right ventricular ejection fraction; RVESV, right ventricular end-systolic volume; RVSV, right ventricular stroke volume; sPAP, estimated systolic pulmonary artery pressure; VHD, valvular heart disease.

3.2 | Association between liver T1-time and TR

Overall, 41% (417) presented with non/trace, 38% (391) mild, 13% (135) moderate and 8% (85) severe, massive and torrential TR. On linear regression analysis, liver T1-time was significantly associated with TR severity (no/trace: 587 ± 72 ms; mild: 601 ± 74 ms; moderate: 634 ± 84 ms; severe/massive/torrential: 664 ± 83 ms, $p < .001$) (Figure 3).

TR severity exerted a notable influence on hepatic T1-time in an Analysis of Variance (ANOVA) ($p < .001$). Finally, in a multivariable linear regression model, TR severity was significantly associated with hepatic T1-time after adjustment for age, sex, serum NT-proBNP and left and right ventricular function ($p < .001$).

3.3 | Association between liver T1-time, TR severity and cardiac magnetic resonance parameters

Detailed results of CMR-determined measurements are provided in Table 3.

Hepatic T1-time was significantly associated with left ventricular (LV) and right ventricular (RV) systolic function on CMR (LV ejection fraction: adj.beta = −1.11, p = <.001; RV ejection fraction: adj.beta = −2.18, $p < .001$) in age and sex-adjusted linear regression. In addition, T1 time correlated significantly with RV and LV size (indexed RV end-diastolic volume (RVEDV) adj.beta = .53, $p = .001$; indexed LV end-diastolic volume (LVEDV) adj.beta = .08, $p = .023$) and increased atrial volumes (indexed left atrium (LA) volume: adj.beta = 2.04, $p = .003$; right atrium (RA) volume: adj.beta = 1.89, $p = .003$).

A significant correlation was observed between myocardial and hepatic T1-time ($r = .55$, $p < .001$), which remained consistent after the exclusion of patients with cardiac amyloidosis ($r = .50$, $p < .001$).

3.4 | Association between liver T1-time and outcome

During a mean follow-up period of 53 ± 36 months, a total of 326 (32%) events occurred. These included 235 deaths (72%; 69 due to CAD, 61 cancer, 29 liver disease, 8 amyloidosis, 8 infections and 60 due to other causes). Additionally, 91 (28%) HF hospitalisations occurred.

Patients with higher hepatic T1-time were more likely to experience adverse events (adj.HR 1.05 [95% CI: 1.04–1.07] per 10 ms increase, $p < .001$) (Figure 4). Likewise, individuals with more severe TR faced worse outcomes (adj. HR 1.66 [95% CI: 1.49–1.84], $p < .001$) (Figure 4). Despite both variables being significant predictors of adverse clinical outcomes, native hepatic T1-time held prognostic value even after adjustment for TR severity, NT-proBNP levels, right and left ventricular structure and function, age and sex (adj.HR 1.42 [95% CI: 1.20–1.68] per 100 ms increase, $p < .001$) (Figure 5, Central Illustration, Table 4). Hepatic T1-time remained significantly associated with outcomes after the exclusion of cardiac amyloidosis patients (adj.HR 1.57 [95% CI:1.34–1.83] per 100 ms increase, $p < .001$).

4 | DISCUSSION

Determining the prognosis of patients within the broad spectrum of HF often is a challenge. In addition to clinical and serum parameters, imaging can be of great help. In particular, the non-invasive assessment of the liver parenchyma with magnetic resonance imaging has recently received a lot of attention.[20,21] Liver T1-time reflects even subtle parenchymal changes in the liver and can be determined from routine CMR scans.[22] In patients undergoing CMR for cardiovascular diseases, liver T1-time was found to be strongly and independently associated with event-free survival,[2,23,24] indicating a strong link between cardiac function and parenchymal liver changes.

In this prospective cohort study of 1029 patients referred for CMR, hepatic T1-time derived from standard cardiac T1-maps was found to be strongly associated with TR severity—a marker of heart failure burden—and independently predictive of clinical outcome.

The exact relationship between heart disease and pathological liver changes remains largely unclear, although patients with HF frequently present with liver fibrosis or even cirrhosis. Only recently, liver stiffness, as determined by non-invasive transient elastography, which can be used to measure the degree of liver stiffness as a marker of fibrosis, was found to be associated with the severity of HF and outcome in 171 patients.[3] Also, in a small observational study of patients with constrictive pericarditis, Fenstad and coworkers reported significantly increased levels of liver stiffness as determined by magnetic resonance elastography and attributed this observation to chronic hepatic venous congestion.[25]

It is commonly assumed that TR links HF and liver fibrosis; however, the respective evidence is scarce. A study on 131 patients with various degrees of TR (48 with mild–moderate; 83 with severe TR) aimed to evaluate the relation between TR severity and liver stiffness as assessed by transient elastography.[5] Patients with severe TR had higher liver stiffness than those with mild–moderate TR. Furthermore, liver stiffness independently correlated with right atrial pressure and inferior vena cava diameter.[5] This is in line with our study, showing higher T1 time in

TABLE 2 Linear and logistic regression on the association between liver T1-time and baseline variables.

Liver T1-time	Linear regression			Age- and sex-adjusted		
Parameter	**Coef.**	**95% Conf-Interval**	***p*-Value**	**Coef.**	**95% Conf-Interval**	***p*-Value**
Age, years (in 10-years increase)	3.33	.44 to 6.22	**.024**	1.04	1.03 to 1.05	**<.001**
Serum parameter						
eGFR, mL/min/1.73 m^2	−.27	−.54 to −.09	**.003**	−.25	−.45 to −.06	**.012**
Haematocrit, %	−2.96	−3.84 to −2.08	**<.001**	−2.87	−3.80 to −1.95	**<.001**
Platelet count, g/L	.07	.002 to .13	**.043**	.07	.004 to .14	.04
INR	37.84	24.02 to 51.65	**<.001**	38.01	24.12 to 51.89	**<.001**
Bilirubin, mg/dL	33.04	23.91 to 42.17	**<.001**	34.52	25.31 to 43.74	**<.001**
Albumin, mg/L	−3.18	−4.26 to −2.19	**<.001**	−3.16	−4.27 to −1.06	**<.001**
AP, U/L	.45	.35 to .57	**<.001**	.45	.34 to .57	**<.001**
AST, U/L	.23	.04 to .46	**.046**	.26	.03 to .50	**.026**
gGT, U/L	.19	.13 to .25	**<.001**	.19	.13 to .25	**<.001**
Serum NT-proBNP, pg/mL	.002	.002 to .003	**<.001**	.002	.002 to .003	**<.001**
HbA1C, %	7.87	.31 to 15.44	**.04**	7.22	−.53 to 14.98	.36
Imaging parameter						
LA volume/BSA, mL/m^2	2.26	1.01 to 3.51	**<.001**	2.04	.71 to 3.36	**.003**
RA volume/BSA, mL/m^2	1.94	.57 to 3.31	**.006**	1.89	.51 to 3.27	**.007**
LVEF, %	1.05	−1.41 to −.68	**<.001**	−1.11	−1.49 to −.74	**.001**
RVEF, %	−2.1ß	−2.52 to −1.68	**<.001**	−2.18	−2.61 to −1.76	**<.001**
LVEDV/BSA, mL/m^2	.038	−.13 to .21	.66	.08	.04 to .65	**.023**
RVEDV/BSA, mL/m^2	.53	.35 to .71	**<.001**	.58	.07 to .66	**<.001**
CMR-ECV, %	2.63	2.04 to 3.23	**<.001**	2.81	2.21 to 3.41	**<.001**
Cardic muscle native, ms	.55	.46 to .63	**<.001**	.55	.47 to .63	**<.001**
Tricusid regurgitation	24.66	19.66 to 29.66	**<.001**	25.58	20.20 to 30.96	**<.001**
	Logistic regression			**Age and sex-adjusted**		
	Odds ratio	**95% Conf-Interval**	***p*-Value**	**Coef.**	**95% Conf-Interval**	***p*-Value**
Sex, male, %	−7.76	−17.47 to 1.94	.12	−6.71	16.44–3.03	.18
Comorbidities						
Hypertension, %	−2.51	−12.25 to 7.23	.61	−6.05	−16.09 to 4.00	.24
Atrial fibrillation, %	37.26	26.33 to 48.18	**<.001**	36.19	24.96 to 47.41	**.001**
Diabetes mellitus, %	10.62	−2.01 to 23.25	.10	8.86	−3.90 to 21.61	.17
Hyperlipidemia, %	−7.84	−18.28 to 2.60	.14	−10.37	−21.00 to .26	.06
COPD, %	23.49	4.12 to 42.86	**.017**	22.09	2.69 to 41.49	**.03**
CAD, %	−6.17	−23.78 to 11.45	.49	−.35	−18.89 to 18.19	.97
Previous PCI, %	−3.23	−27.58 to 21.11	.79	4.54	−20.42 to 29.50	.72
PAD (%)	35.36	−11.51 to 82.22	.14	31.03	−15.87 to 77.93	.19
Chronic kidney disease (%)	17.42	3.26 to 31.58	**.02**	17.13	2.96 to 31.30	**.02**

Note: Bold values indicate $p \leq .05$.

Abbreviations: ALT alanine aminotransferase; AP, alcalic phosphate; AST aspartate aminotransferase; BMI indicates body mass index; CAD, coronary artery disease; CMR-ECV, cardiac magnetic resonance–extracellular volume; CRP, C-reactive protein; eGFR, estimated glomerular filtration rate; gGT, gamma-glutamyltransferase; HbA1C, glycated haemoglobin; INR, international normalized ratio; INR, International Normalized Ratio; IVS, intraventricular septum; LA, left atrium; LVEDV, left ventricular end-diastolic volume; LVEF, left ventricular ejection fraction; NT-proBNP, N-terminal prohormone of brain natriuretic peptide; PAVK, periphery artery disease; PCI, percutaneous coronary intervention; RA, right atrium; RVEDV, right ventricular end-diastolic volume; RVEF, right ventricular ejection fraction.

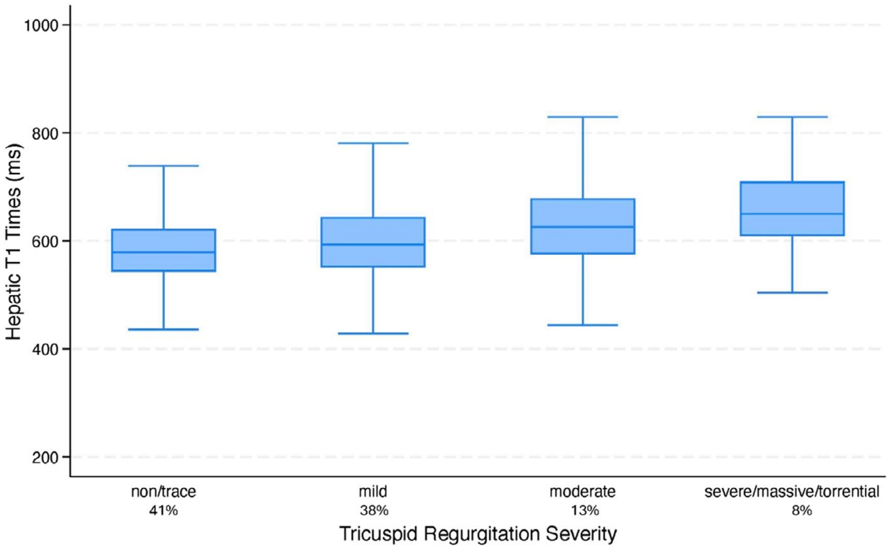

FIGURE 3 Interplay between TR severity and hepatic T1-time. Liver T1-time was significantly correlated with TR severity. (no/trace: 587 ± 72 ms; mild: 601 ± 74 ms; moderate: 634 ± 84 ms; severe/massive/torrential: 664 ± 83 ms; $\beta = 25.4$ ms per TR grade, 95% CI:19.7–31.2, $p < .001$).

patients with higher graded TR. The close link between TR and liver function was also supported by the work of Lim and coworkers, including 159 patients (101 with moderate to severe TR) who underwent surgery for TR between 2008 and 2017.[4] After successful TR intervention, measures of hepatic function, such as the MELD score, improved significantly, indicating a relevant association between TR severity and hepatic function. Taken together, although the above-mentioned studies are all limited by their limited sample size, they indicate a strong association of heart failure, TR and fibrosis of the liver.

The diagnosis of liver fibrosis at an early stage is challenging, but hepatic native T1 mapping can be used as a measure of fibrosis with high accuracy.[14,21,22] Prior research showed that elevated hepatic T1-time correlated with cardiac dimensions and performance, also when adjusted for clinical confounders.[2,23] Additionally, liver T1-time was demonstrated to be independently associated with the risk of hospitalization for HF and cardiovascular death.[2,23,24] A more recent investigation of 37 patients with pulmonary hypertension who underwent both right heart catheterization and CMR demonstrated a close association between invasively measured right atrial pressure and hepatic T1-time on CMR.[26] In line with our results, the authors concluded that the assessment of liver T1-time provided valuable information with regard to the presence of passive liver congestion.

The present study is the first one to investigate the interplay between TR and CMR-derived hepatic T1-time in a large contemporary sample of more than 1 000 patients referred for CMR for cardiovascular diseases. Our results show that hepatic T1-time remained independently associated with prognosis after adjustment for NT-proBNP serum levels, cardiac function and TR.

As the existing evidence shows, liver damage in patients with cardiac disorders is most commonly attributed to congestion due to backward failure or metabolic-associated fatty liver disease, the most rapidly increasing cause of liver-related mortality worldwide. Metabolic-associated fatty liver disease is strongly associated with CVD risk[26] sharing similar risk factors, such as hypertension, diabetes, obesity and metabolic syndrome[27,28] A study in 5121 asymptomatic individuals undergoing coronary computed tomography angiography demonstrated that metabolic-associated fatty liver disease was associated with non-calcified coronary plaques and sudden and unexpected cardiac events[29] Several studies were able to demonstrate a close relationship between scores and actual fibrosis occurrence[30] They additionally showed that the FIB-4 index might serve as a simple biomarker of the severity of RV dysfunction and is a poor prognostic marker for heart failure in patients with preserved ejection fraction[31,32]

Following, liver T1-time seems to be strongly influenced by cardiac pathologies and, as such, contains important prognostic information.

Overall, our study sheds light on the interaction between liver damage and TR severity. However, the fact that liver T1-time was associated with the outcome even after adjustment for TR severity and NT-proBNP as a surrogate for HF severity warrants further investigation.

5 | STUDY LIMITATIONS

The present series is the largest to date investigating the association between hepatic T1-time and TR in HF patients. Despite the large sample size, several issues merit comment. Data have been collected in a single-center setting; however, our single-center setting allowed for consistency of CMR and echocardiography exams and reporting as well as patient work-up throughout the study period, including

TABLE 3 Baseline CMR characteristics, stratified by TR and hepatic T1-time.

	Total N = 1029	T1-time <594 ms, TR <moderate N = 450	T1-time <594 ms TR ≥moderate N = 63	T1-time ≥594 ms, TR <moderate N = 359	T1-time ≥594 ms, TR ≥moderate N = 157	*p*-Value
CMR parameters						
LVEDV, mL	160.82 ± 60.62	161.51 ± 59.38	147.47 ± 49.77	164.17 ± 57.90	156.67 ± 72.55	.18
LVEDV/BSA, mL/m^2	84.19 ± 29.19	83.45 ± 27.14	80.94 ± 28.30	86.14 ± 28.90	83.25 ± 35.17	.44
LVEF, %	57.58 ± 13.35	60.36 ± 11.73	56.52 ± 15.16	56.77 ± 13.63	52.18 ± 14.31	**<.001**
LVSV, mL	88.14 ± 28.63	93.35 ± 29.59	78.41 ± 23.45	89.28 ± 28.44	74.84 ± 22.68	**<.001**
LVSV/BSA, mL/m^2	46.18 ± 13.63	48.35 ± 13.78	43.06 ± 13.15	46.76 ± 13.47	40.07 ± 11.75	**<.001**
IVS, mm	12.67 ± 3.97	12.57 ± 3.58	11.61 ± 3.04	13.11 ± 4.59	12.35 ± 3.71	**.018**
LVCO, mL	5.65 ± 1.98	5.90 ± 2.12	5.29 ± 1.98	5.69 ± 1.86	5.00 ± 1.70	**<.001**
LVCO/BSA, mL/m^2	2.96 ± .97	3.06 ± 1.01	2.89 ± 1.08	2.99 ± .92	2.66 ± .84	**<.001**
RVEDV, mL	157.85 ± 54.37	149.60 ± 44.45	165.42 ± 61.38	156.17 ± 51.75	181.69 ± 72.68	**<.001**
RVEDV/BSA, mL/m^2	82.97 ± 26.95	77.49 ± 20.10	90.64 ± 34.11	81.95 ± 24.34	97.47 ± 38.07	**<.001**
RVEF, %	52.77 ± 11.22	56.48 ± 9.10	49.12 ± 11.78	52.57 ± 10.90	44.46 ± 12.07	**<.001**
RVSV, mL	80.47 ± 26.60	83.00 ± 24.85	76.58 ± 23.44	79.68 ± 27.25	76.75 ± 30.27	**.038**
RVEDV, mL	157.85 ± 54.37	149.60 ± 44.45	165.42 ± 61.38	156.17 ± 51.75	181.69 ± 72.68	**<.001**
RVEDV/BSA, mL/m^2	82.97 ± 26.95	77.49 ± 20.10	90.64 ± 34.11	81.95 ± 24.34	97.47 ± 38.07	**<.001**
RVCO, mL	5.18 ± 2.42	5.21 ± 1.82	5.02 ± 1.59	5.08 ± 1.77	5.40 ± 4.55	.53
RVCO/BSA, mL/m^2	2.72 ± 1.25	2.69 ± .85	2.74 ± .87	2.67 ± .87	2.92 ± 2.47	.21
RVSV, mL	80.47 ± 26.60	83.00 ± 24.85	76.58 ± 23.44	79.68 ± 27.25	76.75 ± 30.27	**.038**
RVSV/BSA, mL/m^2	42.27 ± 13.07	42.96 ± 11.46	42.11 ± 13.62	41.79 ± 13.11	41.47 ± 16.58	.53
LV-Mass (g)	147.71 ± 58.65	146.74 ± 58.67	133.56 ± 54.87	155.45 ± 60.51	138.49 ± 53.88	**.014**
LA volume, mL	37.91 ± 6.58	37.12 ± 6.47	38.57 ± 5.79	37.69 ± 6.39	40.29 ± 7.05	**<.001**
LA volume/BSA, mL/m^2	20.09 ± 3.98	19.44 ± 3.94	21.18 ± 3.66	19.98 ± 3.68	21.71 ± 4.36	**<.001**
RA volume, mL	35.74 ± 9.84	33.73 ± 4.94	39.56 ± 7.09	35.10 ± 6.79	42.21 ± 20.39	**<.001**
RA volume/BSA, mL/m^2	18.58 ± 5.56	17.26 ± 2.63	21.41 ± 4.12	18.37 ± 3.55	22.23 ± 11.86	**<.001**
Myocardial native T1, ms	1026.38 ± 58.42	1008.83 ± 60.21	1021.40 ± 44.07	1040.97 ± 52.25	1045.31 ± 57.21	**<.001**
Blood native T1, ms	1618.04 ± 123.38	1575.86 ± 114.62	1631.37 ± 126.62	1645.29 ± 118.10	1671.25 ± 119.81	**<.001**
Liver native T1, ms	605.61 ± 81.92	545.19 ± 40.67	550.17 ± 34.14	654.88 ± 62.48	688.40 ± 76.02	**<.001**
Echocardiographic parameters						
sPAP, mmHg	47.39 ± 17.26	39.60 ± 12.33	61.83 ± 19.23	43.80 ± 14.28	60.86 ± 16.93	**<.001**

Note: Bold values indicate $p \leq .05$.

Abbreviations: ALT, alanine aminotransferase; AP, alkalic phosphate; AST, aspartate aminotransferase; BMI, body mass index; CMR indicates cardic magnetic resonance tomography; CRP, C-reactive protein; eGFR, estimated glomerular filtration rate; gGT, gamma-glutamyltransferase; HbA1C, glycated haemoglobin; INR, International Normalized Ratio; NT-proBNP, N-terminal prohormone of brain natriuretic peptide; PAD, peripheral artery disease.

post-processing workflows. Nevertheless, referral bias may influence the results in a single-center study. Besides fibrosis and edema, factors like amyloid deposition, iron or other proteins can influence hepatic T1-time. Iron-corrected T1 or T2-star sequences were not performed, which are the established MR sequences for iron overload. Additionally, proton density fat fraction (PDFF) mapping was not done. We included patients with cardiac amyloidosis in the study but excluded them from certain analyses. However, amyloid deposits can also occur solely in the liver.

The study is furthermore limited by the lack of systematic liver biopsies, which were not performed due to procedural risk. Although only patients in stable clinical condition without overt cardiac decompensation were included, subclinical congestion may have been present in a proportion of study participants. Furthermore, the use of transthoracic echocardiography limited our ability to determine the aetiology of TR, which we consider a significant limitation. Understanding whether specific TR etiologies cause more liver damage than others would be

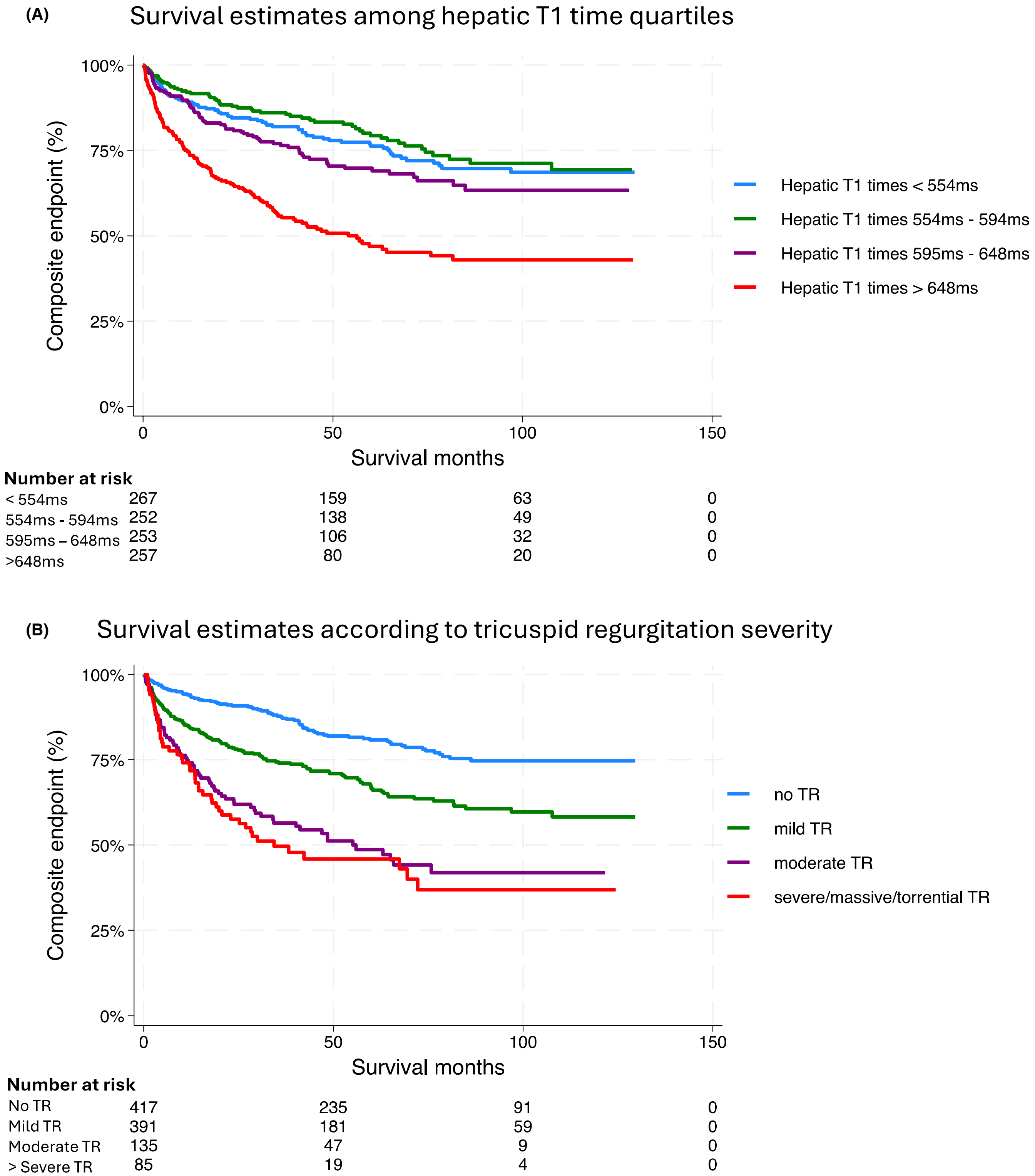

FIGURE 4 Kaplan–Meier estimates according to hepatic T1-time (panel A) and degree of tricuspid regurgitation (panel B). Panel (A) shows Kaplan–Meier survival estimates according to hepatic T1-time quartiles; panel (B) shows estimates according to the degree of tricuspid regurgitation. TR, tricuspid regurgitation.

particularly insightful. In addition, TR grading was performed by board-certified cardiologists as part of routine clinical care; therefore, a formal adjudication committee for TR severity was missing. The number of patients with severe to torrential TR was relatively low, which may limit subgroup analyses and interpretation of prognostic

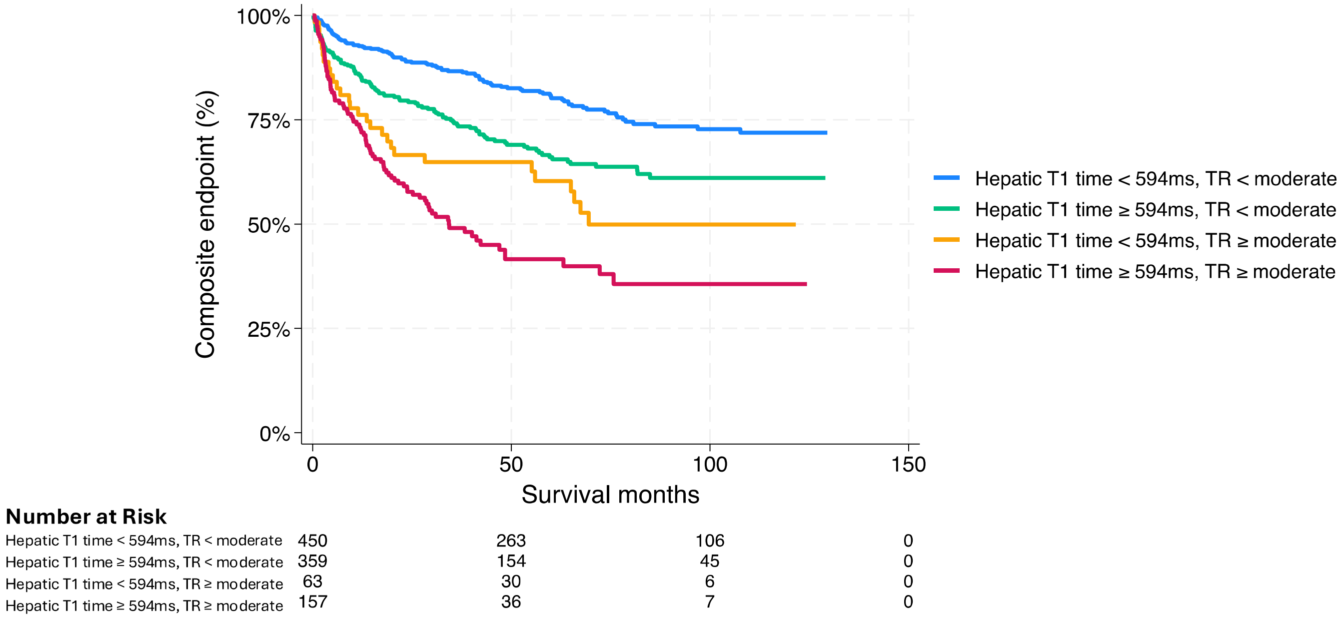

FIGURE 5 Showing Kaplan–Meier estimates stratified by TR severity and hepatic T1-time. TR, tricuspid regurgitation.

TABLE 4 Multivariable Cox-regression demonstrating an independent association of hepatic T1-time with outcome.

	HR	95% Conf-Interval	*p*-Value
Hepatic T1-time (ms)	1.003	1.001–1.005	**<.001**
Tricusid regurgitation severity	1.377	1.195–1.588	**<.001**
NT-proBNP levels (in 1000 mg/dL increase, mg/dL)	1.038	1.021–1.055	**<.001**
RVEF; %	1.001	.988–1.016	.851
LVEF, %	1.006	.995–1.018	.282
Myocardial T1 times (ms)	1.002	.987–1.004	.076
Age (years)	1.023	1.013–1.034	**<.001**
Sex, male, %	1.327	1.015–1.734	**.039**

Abbreviations: LVEF, left ventricular ejection fraction; RVEF, right ventricular ejection fraction.

associations in this group. A Bland–Altman analysis of TR grading was not performed due to the absence of repeat measurements by independent observers.

6 | CONCLUSIONS

The severity of TR and hepatic T1-time, which reflect liver congestion and function, are closely connected. In over 1000 individuals with cardiovascular conditions, liver T1-time independently predicted outcomes, underscoring its diagnostic ability. Hepatic T1-time could thus serve as an early imaging marker for HF and provide prognostic value for the assessment of valvular heart disease in the future. Our findings highlight the importance of hepatic imaging in the comprehensive assessment and management of cardiovascular disease.

AUTHOR CONTRIBUTIONS

Katharina Mascherbauer: conception, analysis and interpretation of data, manuscript drafting. Christina Kronberger: conception, analysis and interpretation of data, manuscript drafting. Manuel Gruber: conception, analysis and interpretation of data. Carolina Donà: conception, analysis and interpretation of data, manuscript drafting. Matthias Koschutnik: conception, analysis and interpretation of data, manuscript drafting. Varius Dannenberg: conception, analysis and interpretation of data, manuscript drafting. Michael Poledniczek: conception, analysis and interpretation of data, manuscript drafting. Laura Lunzer: conception, analysis and interpretation of data, manuscript drafting. Christian Nitsche: conception, analysis and interpretation of data, manuscript drafting. Franz Duca: conception, analysis and interpretation of data, manuscript drafting. Gregor

Heitzinger: conception, analysis and interpretation of data, manuscript drafting. Kseniya Halavina: conception, analysis and interpretation of data, manuscript drafting. Dietrich Beitzke: conception, analysis and interpretation of data. Christian Loewe: analysis and interpretation of data. Michael Trauner: analysis and interpretation of data. Philipp Bartko: conception, analysis. Julia Mascherbauer: conception, analysis and interpretation of data; revising the manuscript. Christian Hengstenberg: conception, analysis and interpretation of data, revising the manuscript. Andreas Kammerlander: conception, analysis and interpretation of data; revising the manuscript. Elisabeth Waldmann: conception, analysis and interpretation of data; revising the manuscript.

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None.

CONFLICT OF INTEREST STATEMENT

All authors have nothing to disclose.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

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