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ORIGINAL ARTICLE

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Right ventricular strain and tricuspid annular plane systolic excursion are associated with mortality in inferior ST-elevation myocardial infarction

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Abstract

Background: Patients with inferior ST-segment elevation myocardial infarction face a substantial risk for cardiovascular death. While left ventricular function is known to be associated with clinical outcomes in these patients, we evaluated the prognostic impact of tricuspid annular plane systolic excursion (TAPSE) and advanced measures of right ventricular function (free wall strain [FWS] and global longitudinal strain [RVGLS]).

Methods: Consecutive patients presenting with acute inferior ST-segment elevation myocardial infarction undergoing percutaneous coronary intervention between 01/2012 and 08/2015 were retrospectively analysed. Associations between RV strain measurements and all-cause mortality were evaluated using Cox regression analysis.

Results: 207 patients (69.6% male, median 59.0 [IQR: 52.1–70.7] years) were followed for 8.3 (IQR: 7.4–9.3) years, during which 49 patients (23.7%) deceased. Median right ventricular function parameters were significantly better in surviving patients (RVGLS: −17.5% vs. −13.3%, $p < .001$; FWS: −20.5% vs. −14.8%, $p < .001$; TAPSE 1.8 cm vs. 1.3 cm, $p < .001$). All 3 parameters were associated with mortality in univariate and multivariable analysis adjusted for age, sex and the number of comorbidities (chronic kidney disease, hypercholesterinaemia, diabetes mellitus) (adj. hazard ratio [HR] per 1 standard deviation: RVGLS: 1.68 [95% CI: 1.27–2.23, $p < .001$], FWS: 1.56 [95% CI: 1.56–2.00, $p < .001$], TAPSE: 1.55 [95% CI: 1.17–2.05, $p = .002$]). Additionally, right ventricular function was inversely associated with peak troponin T and creatine kinase levels.

Conclusions: Among patients with inferior ST-segment myocardial infarction, RVGLS, FWS and TAPSE convey crucial prognostic information and might help to identify patients at increased risk requiring intensified monitoring and therapy.

KEYWORDS
inferior ST-segment elevation myocardial infarction, right ventricular function, right ventricular strain imaging, speckle tracking

1 | INTRODUCTION

Inferior ST-elevation myocardial infarction (STEMI) represents a substantial portion of acute coronary syndromes and continues to pose a significant burden on global healthcare systems.[1]

While advancements in reperfusion therapy have considerably improved outcomes, risk stratification remains essential in identifying patients who may benefit from targeted interventions and closer monitoring.[2,3] Traditionally, the focus of STEMI management has predominantly revolved around assessing left ventricular (LV) dysfunction due to its well-established association with adverse clinical outcomes.[4] However, in recent years, there has been a growing interest in the role of right ventricular (RV) dysfunction as a prognostic marker in patients with inferior STEMI.[5–7]

Besides more conventional echocardiographic parameters, such as tricuspid annular plane systolic excursion (TAPSE), strain measurements have gained increasing importance for the echocardiographic assessment of RV function during the last decade.[8,9] RV strain parameters such as free wall strain (FWS), RV global longitudinal strain (RVGLS) and RV regional wall strain measures have not only demonstrated prognostic value in patients with STEMI but also in populations with various other cardiovascular pathologies.[10–13]

Although several authors have observed a significant association of diverse RV function parameters and clinical outcome parameters in patients with inferior STEMI,[7,14] data are inconsistent, and the independent prognostic value of RV strain parameters for the prediction of all-cause mortality remains to be established. Therefore, the aim of the present study was to assess the strength of the association between FWS, RVGLS, RV regional wall strain measures, and TAPSE after inferior STEMI and all-cause mortality. Furthermore, we aimed to assess the association between the degree of RV dysfunction and parameters of myocardial damage in patients with inferior STEMI.

2 | MATERIALS AND METHODS

2.1 | Study design, setting and population

This retrospective, observational, single-centre cohort study was conducted at the Vienna General Hospital (Austria), a large university-affiliated tertiary centre with more than 1700 beds and a high-volume 24-h coronary catheter laboratory. All patients who presented with (1) acute inferior STEMI and (2) underwent acute coronary intervention between 01/2012 and 08/2015 were eligible for study inclusion. Diagnosis of inferior STEMI was based on the Third Universal Definition of Myocardial Infarction.[1] Specifically, the ECG criteria for inferior STEMI were ST elevation ≥1 mV in more than one of leads II, III and aVF. Exclusion criteria were (1) age <19 years, (2) cardiogenic shock requiring catecholamine support, (3) the need for cardiopulmonary resuscitation and (4) no echocardiographic study within the first 3 days after myocardial infarction.

The study protocol complies with the Declaration of Helsinki and was approved by the local ethics committee. The local ethics committee determined that written informed consent was not required due to the retrospective nature of the study. Demographic, angiographic and laboratory data was extracted from patient records.

2.2 | Coronary angiography

Acute angiography and percutaneous coronary intervention were performed according to guidelines from the European Society of Cardiology[15] and the hospital's clinical practice and was left to the operators' discretion. We used the online calculator version (syntaxscore.org, version 2.28) to assess the Syntax-I score in all patients for quantification of coronary artery disease severity.

2.3 | Study endpoints

The study endpoint all-cause mortality was determined by screening the Austrian register of deaths (statistics Austria).

2.4 | Echocardiographic measurements

All patients underwent a comprehensive transthoracic echocardiographic study performed by an experienced echocardiographer within the first 3 days after myocardial infarction using a commercially available GE cardiovascular ultrasound system (General Electric Healthcare, Waukesha, WI, USA). These studies included apical 4-, 2- and 3-chamber views with a frame rate of 60–80 frames/

second and 3–5 cardiac cycles. Offline analysis of strain and function parameters was performed using dedicated software (EchoPAC Clinical Workstation Software, GE Vingmed Ultrasound, Version 206) on professional workstations. For the 2-dimensional longitudinal speckle tracking analysis of the LV and the RV, the GE EchoPAC Automated Function Imaging (AFI) software was used according to the vendor's instructions. The peak-systolic strain of the full myocardium (Echopac AFI strain reference layer "Full") was assessed. End-diastole was defined by ECG gating (peak of R wave). End-systole was defined by aortic valve closure (AVC) and pulmonary valve closure (PVC) measurements derived from Doppler measurements. In the 6 patients with atrial fibrillation a cycle with a medium cycle length was used for strain analysis. In patients with premature beats, only cycles between two regular beats were analysed. In brief, speckle tracking analysis of the LV was performed in all 3 apical views. The contouring and tracking of the myocardium through the cardiac cycle was accomplished by the automated imaging software, reassessed visually and adjusted manually if necessary. Myocardial segments with inadequate tracking after readjustment were excluded from analysis. The manual adjustment also included the modification of the region of interest to correctly cover the thickness of the myocardium. LVGLS, peak longitudinal systolic strain values of all 17 segments, and time-domain LV strain curves for each segment were calculated automatically by the software system. RVGLS, RV FWS and segmental RV strain values were assessed in a RV-focused apical 4-chamber view (Figure 1). Similar to the LV, the delineation and tracking of the RV myocardium was performed automatically by the software (after the placing of 3 endocardial markers) and adjusted manually if necessary.[16] The software automatically calculated RVGLS, FWS and 6 segmental strain values. Subjects in whom >1 segment per view could not adequately be tracked were excluded from strain analysis. TAPSE was assessed as previously

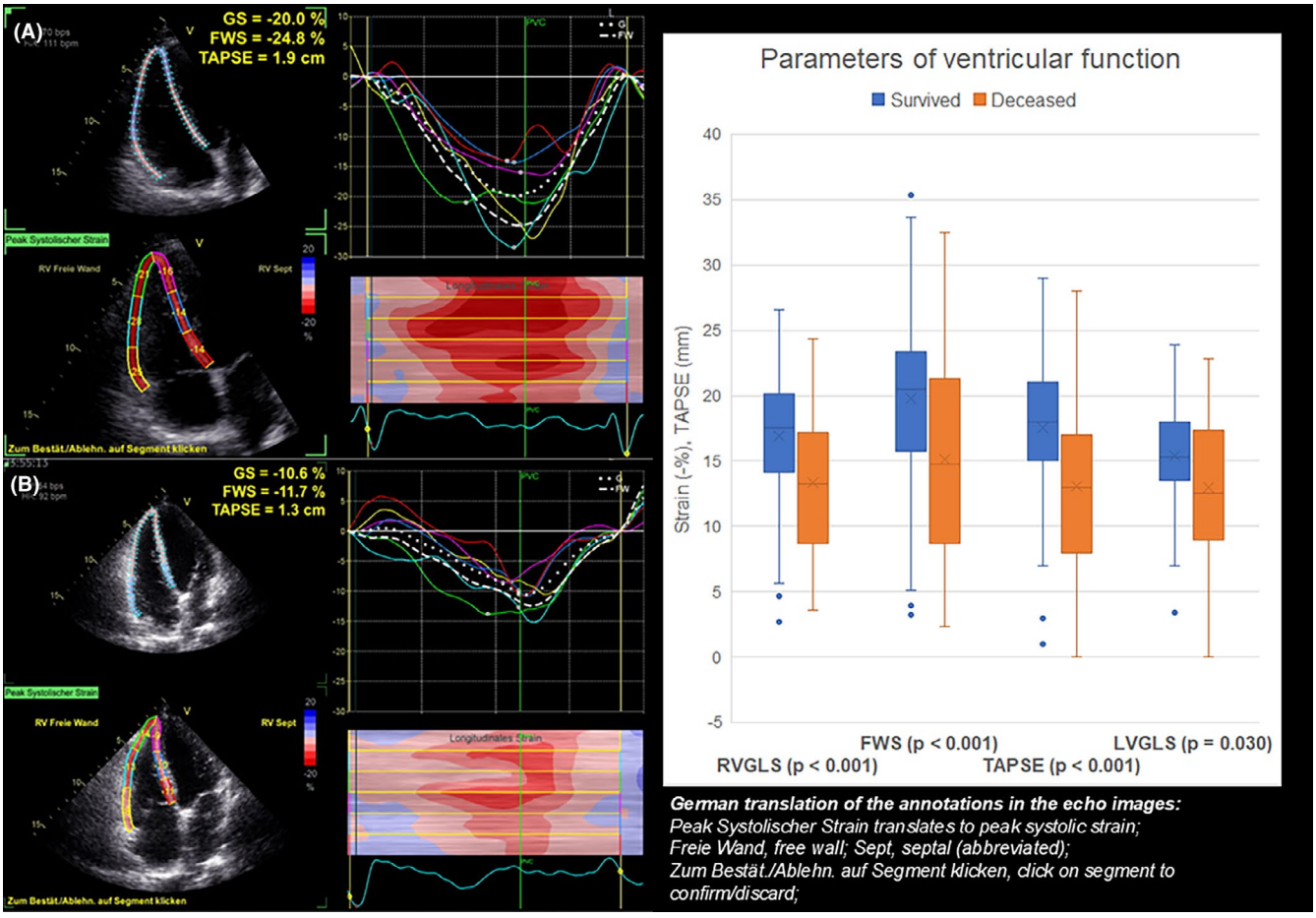

FIGURE 1 Right ventricle-focused 4-chamber view with right ventricle strain measurements and boxplots of ventricular function stratified by survival status; Measurement of right ventricular global longitudinal strain, free wall strain, and segmental RV strain measures in a RV-focused apical 4-chamber view using the EchoPAC Automated Function Imaging software. Peak-systolic strain values of the whole myocardium are reported. (A) example of a survivor versus (B) example of a non-survivor. FWS, free wall strain; LVGLS, right ventricular global longitudinal strain; TAPSE, tricuspid annular plane systolic excursion.

described.[17] LV ejection fraction was calculated using biplane Simpson's method.

2.5 | Statistical analysis

Continuous data is presented as mean ± standard deviation (SD) or as median and interquartile range (IQR) with distribution assessed utilizing the Shapiro–Wilk test. Differences between groups are assessed using the *t*-test for independent samples or the Mann–Whitney *U* test, respectively. Categorical data are presented as counts and percentages, with differences evaluated using the chi-square test. Correlation between the parameters of RV function and LVGLS was assessed using Spearman correlation analysis.

Univariate and multivariate Cox regression analyses were conducted to assess the association between ventricular functional parameters and all-cause mortality. Separate multivariate Cox regression models were created for each of the ventricular functional parameters, including the respective ventricular functional parameter, age, sex and a comorbidity score (from 0 to 3) according to the number of the following comorbidities: diabetes mellitus, hypercholesterinaemia and reduced kidney function defined as an eGFR of <60 mL/min/1.73m^2. These comorbidities are known to be associated with adverse outcomes in patients with myocardial infarction.[18–20] To allow for a better comparison between different RV function parameters, regression models report hazard ratios (HR) per 1-SD worsening of RV function and the 95% confidence interval (CI). To describe the respective models' goodness-of-fit, the Akaike information criterion, the log likelihood value and the chi-square values were calculated. The Youden index was used to obtain cut-off values for the ventricular functional parameters. Consecutively, Kaplan Meier estimates and log-rank tests were used to assess differences in survival. The discriminatory abilities of RVGLS, FWS and TAPSE for prediction of all-cause mortality were assessed using Harrell's C-statistic. Additionally, the association of different RV function parameters with peak Troponin T levels was assessed using linear regression models (crude and adjusted for LVGLS). Intra-observer and inter-observer reproducibility of strain and TAPSE measurements were assessed in a random sample of 20 patients by intraclass correlation coefficients (ICCs) as presented in Table S1. All ICCs were >.90, indicating a high level of agreement in line with previous publications.[21,22]

Statistical analysis was conducted using BlueSky Statistics 10.3.4, R package version 8.95 (BlueSky Statistics LLC, Chicago, IL, USA) and IBM SPSS Statistics 29 (IBM Corporation, Armonk, NY, USA), and $p < .05$ (two-sided) was considered statistically significant.

3 | RESULTS

3.1 | Study population and angiographic findings

A total of 297 consecutive patients undergoing acute percutaneous coronary intervention due to acute inferior STEMI were screened for eligibility, and 244 fulfilled inclusion criteria, as shown in the flow chart in Figure 2. Out of these patients, 207 had sufficient quality on echocardiography to perform speckle tracking analysis, representing the final study population. Mortality data were available in all 207 patients. The baseline patient characteristics of the final study population stratified by survival status are displayed in Table 1. Patients were predominantly male (69.6%) with a median age of 59.0 (IQR: 52.1–70.7) years.

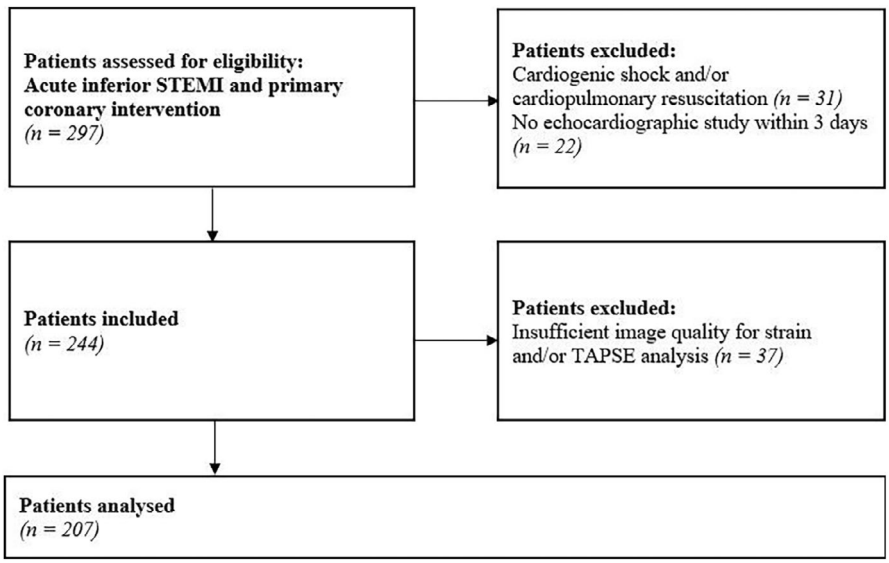

FIGURE 2 Patient recruitment flow chart; STEMI, ST-elevation myocardial infarction; TAPSE, tricuspid annular plane systolic excursion; Study design: The study is a retrospective, observational, single-centre cohort study.

TABLE 1 Baseline characteristics and coronary angiography data, stratified by survival status.

	Total	Alive	Deceased	
	N = 207	*N* = 158	*N* = 49	*p*-Value
Demographics and Medical History				
Age (years)	59.0 (52.1–70.7)	57.5 (51.0–66.4)	68.6 (59.0–77.7)	<.001
Male Sex	144 (69.6%)	114 (72.2%)	30 (61.2%)	.146
Systolic blood pressure (mmHg)	132 ± 26	136 ± 24	129 ± 32	.474
Diastolic blood pressure (mmHg)	77 ± 15	78 ± 14	73 ± 17	.053
Heart rate (bpm)	75 ± 18	75 ± 17	76 ± 19	.933
Body mass index (kg/m^2)	27.2 (24.8–30.1)	27.4 (25.1–30.4)	26.9 (24.7–29.0)	.293
COPD	14 (6.8%)	8 (5.1%)	6 (12.2%)	.080
Peripheral arterial disease	18 (8.7%)	13 (8.2%)	5 (10.2%)	.668
Previous stroke	12 (5.8%)	9 (5.7%)	3 (6.1%)	.911
Cerebrovascular disease	8 (3.9%)	8 (5.1%)	0 (.0%)	.108
Chronic kidney disease	15 (7.2%)	9 (5.7%)	6 (12.2%)	.122
Hypertension	152 (73.4%)	115 (72.8%)	37 (75.5%)	.706
Diabetes mellitus	47 (22.7%)	31 (19.6%)	16 (32.7%)	.057
Hyperlipidaemia	132 (63.8%)	106 (67.1%)	26 (53.1%)	.074
Current smoker	117 (56.5%)	90 (57.0%)	27 (55.1%)	.819
Laboratory evaluations				
GFR (mL/min/1.73m^2)	77.3 (64.2–91.7)	78.8 (68.6–94.3)	70.4 (51.3–85.1)	.001
LDL (mg/dL)	110 ± 40	111 ± 40	106 ± 39	.539
HbA1c (%)	5.6 (5.3–6.0)	5.6 (5.3–5.9)	5.8 (5.3–6.2)	.240
Peak creatine kinase (U/l)	1302 (590–2450)	1301 (605–2453)	1333 (509–2372)	.865
Peak CK-MB (U/l)	164 (90.3–273)	164 (91.6–273)	162 (81.2–262)	.770
Peak Troponin T (ng/mL)	3.04 (1.14–5.35)	2.70 (1.05–5.26)	3.47 (1.63–6.31)	.146
Coronary angiography data				
Right dominance	178 (86.0%)	135 (85.4%)	43 (87.8%)	.684
Coronary vessels diseased				
1-VD	113 (54.6%)	92 (58.2%)	21 (42.9%)	.153
2-VD	60 (29.0%)	43 (27.2%)	17 (34.7%)	
3-VD	34 (16.4%)	23 (14.6%)	11 (22.4%)	
Syntax score I	8.0 (3.5–12)	8.0 (3.0–10.5)	9.0 (7.0–15.5)	.016
Culprit lesion				
Right coronary artery	156 (75.4%)	120 (75.9%)	36 (73.5%)	.725
Circumflex artery	51 (24.6%)	38 (24.1%)	13 (26.5%)	
Discharge Medication				
Aspirin	205 (99.0%)	158 (100.0%)	47 (95.9%)	.011
Ticagrelor	46 (22.2%)	34 (21.5%)	12 (24.5%)	.662
Clopidogrel	40 (19.3%)	24 (15.2%)	16 (32.7%)	.007
Prasugrel	121 (58.5%)	100 (63.3%)	21 (42.9%)	.011
Beta blocker	197 (95.2%)	151 (95.6%)	46 (93.9%)	.629
RAAS inhibitor	202 (97.6%)	155 (98.1%)	47 (95.9%)	.385
Thiazide diuretics	24 (11.6%)	16 (10.1%)	8 (16.3%)	.236
Loop diuretics	18 (8.7%)	3 (1.9%)	15 (30.6%)	<.001
Aldosterone receptor antagonist	31 (15.0%)	17 (10.8%)	14 (28.6%)	.002

(Continues)

TABLE 1 (Continued)

	Total	Alive	Deceased	
	N = 207	*N* = 158	*N* = 49	*p*-Value
Calcium channel blocker	19 (9.2%)	15 (9.5%)	4 (8.2%)	.778
Statins	202 (97.6%)	156 (98.7%)	46 (93.9%)	.053

Note: Metric data are given as mean ± standard deviation in case of normal distribution, and as median (interquartile range) in case of non-normal distribution, which is assessed utilizing the Shapiro–Wilk test. Categorical data is displayed as counts (percentage). *p*-values for metric variables are derived from the *t*-test for independent samples and the Mann–Whitney-*U* test, respectively. For binary/categorical variables, the chi-square test is utilized.

Abbreviations: CK-MB, creatine kinase – muscle-brain type; COPD, chronic obstructive pulmonary disease; GFR, glomerular filtration rate; HbA1c; haemoglobin A1c; mmHg, millimetres of mercury; LDL, Low-density lipoprotein; RAAS; renin-angiotensin-aldosterone system; VD, vessel disease.

Cardiovascular risk factors were highly prevalent (hypertension in 73.4%, diabetes mellitus in 22.7%, hyperlipidaemia in 63.8% and current smoking status in 56.5% of patients, respectively). In 75.4% of patients, the culprit lesion was located in the right coronary artery (RCA). The majority of patients (54.6%) had single-vessel disease, and the median Syntax-I score was 8.0 (IQR: 3.5–12).

Table 2 and Figure 1 display detailed data on echocardiographic findings after inferior STEMI, stratified by survival status. LV and RV function according to strain parameters and TAPSE was significantly better in patients who survived the observation period. There were strong correlations among parameters of RV function and a weak association between parameters of RV function and LVGLS (RVGLS: $r = .39$ FWS: $r = .31$, TAPSE: $r = -.30$; all $p < .001$). Correlations between markers of ventricular function and clinical parameters are shown in the Table S2.

Atrial fibrillation and bundle branch block were present in 6 (2.9%) and 9 (4.3%) patients, respectively, at the time of the echocardiographic study (Table 2). Tables S3 and S4 display the baseline characteristics and echocardiographic measurements stratified by cardiac rhythm and presence of conduction abnormalities.

3.2 | RV function and mortality

During a median follow-up of 8.3 (IQR: 7.4–9.3) years, a total of 49 patients (23.7%) deceased. The global RV strain parameters RVGLS and FWS as well as the TAPSE were significantly associated with all-cause mortality in univariate and multivariable Cox regression analysis after adjustment for age, sex and a comorbidity score (including chronic kidney disease, hypercholesterinaemia and diabetes mellitus). All results of the univariate and multivariate Cox regression analysis are depicted in Table 3. Forest plots for the multivariate models are depicted in Figure 3A–D. According to the adjusted HR, all three RV function parameters had a rather comparable predictive value with the highest adjusted HR per 1-SD increase of 1.68 for RVGLS.

Additionally, we assessed the predictive value of all segmental RV longitudinal strain values. Although four out of the six segmental RV strain values predicted mortality in univariate analysis, only the basal free wall and the septal mid-cavity segment remained predictive after multivariable adjustment (HR per 1-SD increase: 1.69, 95% CI: 1.35–2.13, $p < .001$; HR per 1-SD increase: 1.36, 95% CI: 1.02–1.81, $p = .035$).

Figure 4 shows Kaplan–Meier survival curves stratified according to tertiles of RVGLS (1st: tertile: <−18.4%, 3rd tertile: >−14.1%), FWS (1st tertile: <−21.9%, 3rd tertile: >−16.0%), TAPSE (1st tertile: >2.1 cm, 3rd tertile: <1.5 cm) and LVGLS (1st: tertile: <−16.8%, 3rd tertile: >−13.8%). Interestingly, the survival curve of the mid tertile of TAPSE clearly separates from the first and third tertiles and seems to illustrate an intermediate risk group, while the risk of death in the first and second tertiles of RVGLS, FWS and LVGLS was still low and distinctly increased only in the third tertile. To better characterize this intermediate risk group, Tables S5 and S6 present the patient characteristics and echocardiographic parameters of subgroups stratified by TAPSE tertiles. Kaplan–Meier survival curves stratified by the optimal cut-off values according to the Youden index (RVGLS: −13.5%, FWS: −15.1%, TAPSE: 1.4 cm, LVGLS: −11.15%) are depicted in Figure 5.

As assessed by Harrell's C-statistic, the discriminatory abilities of RVGLS, FWS and TAPSE for the prediction of long-term mortality were .675 [95% CI .595–.754, $p < .001$], .667 [95% CI .586–.748, $p < .001$] and .688 [95% CI .609–.767, $p < .001$], respectively, and the addition of either RVGLS, FWS or TAPSE to our base model (including age, sex and comorbidity score) improved discrimination (Table S7).

3.3 | Association of RV function parameters with laboratory parameters of myocardial ischaemia

Table 4 shows the association between RV function parameters and peak Troponin T levels in the course of

TABLE 2 Echocardiographic parameters, stratified by survival status.

	Total	Alive	Deceased	
	N = 207	*N* = 158	*N* = 49	*p*-Value
Parameters of global right ventricular function				
RVGLS[a] (%)	−16.7 (−13.1 to −19.2)	−17.5 (−14.1 to −20.1)	−13.3 (−9.2 to −17.1)	<.001
FWS[a] (%)	−19.7 (−14.7 to −23.2)	−20.5 (−15.9 to −23.3)	−14.8 (−9.3 to −21.1)	<.001
TAPSE[a] (cm)	1.7 (1.4 to 2.1)	1.8 (1.5 to 2.1)	1.3 (.8 to 1.7)	<.001
Right ventricular segmental longitudinal strain				
Basal FW (%)	−26.0 (−19.8 to −31.0)	−27.0 (−21.0 to −33.0)	−19.0 (−8.0 to −27.0)	<.001
Mid FW (%)	−21.0 (−14.0 to −28.0)	−22.5 (−15.0 to −29.0)	−16.0 (−9.0 to −23.0)	.008
Apical FW (%)	−11.0 (−6.0 to −17.0)	−10.0 (−3.8 to −16.0)	−13.0 (−8.0 to −19.5)	.083
Basal IVS (%)	−10.0 (−6.0 to −13.0)	−10.0 (−6.0 to −14.0)	−9.0 (−4.5 to −12.5)	.183
Mid IVS (%)	−17.0 (−13.0 to −20.2)	−17.0 (−14.0 to −21.0)	−16.0 (−10.0 to −19.0)	.027
Apical IVS (%)	−15.0 (−10.0 to −19.0)	−16.0 (−12.0 to −19.0)	−11.0 (−8.0 to −16.0)	.007
Other echocardiographic parameters				
LVEF (%)	50.0 (45.0 to 55.0)	50.0 (46.5 to 55.0)	48.0 (39.0–54.5)	.077
LVGLS[b] (%)	−15.2 (−13.0 to −17.8)	−15.3 (−13.5 to −17.9)	−13.6 (−9.3 to −17.2)	.030
LVEDD (mm)	46.0 (42.0 to 49.0)	46.0 (42.0 to 49.0)	46.0 (41.0 to 49.0)	.600
RVEDD (mm)	32.0 (29.0 to 34.0)	32.0 (29.0 to 34.0)	32.0 (29.0 to 36.0)	.351
Left atrial diameter (mm)	52.0 (48.2 to 55.0)	51.0 (48.0 to 55.0)	52.0 (50.0 to 58.0)	.050
Right atrial diameter (mm)	51.0 (47.0 to 54.0)	50.0 (47.0 to 53.0)	51.0 (48.8 to 56.0)	.095
IVS (mm)	13.0 (12.0 to 14.0)	13.0 (12.0 to 14.0)	13.0 (12.0 to 14.2)	.890
LVEDVi (mL)	58.0 (47.8 to 67.6)	57.7 (47.7 to 66.6)	60.6 (49.4 to 68.3)	.373
TR ≥ moderate	8 (9.9%)	3 (5.5%)	5 (19.2%)	.052
MR ≥ moderate	16 (7.8%)	10 (6.3%)	6 (12.5%)	.162
AS ≥ moderate	1 (.5%)	0 (.0%)	1 (2.2%)	.067
Conduction disorders and atrial fibrillation during the echo study				
2nd/3rd degree AV block	0 (.0%)	0 (.0%)	0 (.0%)	
Bundle branch block	9 (4.3%)	6 (3.8%)	3 (6.1%)	.486
Atrial fibrillation	6 (2.9%)	2 (1.3%)	4 (8.2%)	.012

Note: Metric data are given as mean ± standard deviation in case of normal distribution, and as median (interquartile range) in case of non-normal distribution. Categorical data is displayed as counts (percentage). *p*-values for metric variables are derived from the *t*-test for independent samples and the Mann–Whitney-*U* test, respectively. For binary/categorical variables, the chi-square test is utilized.

Abbreviations: AS, aortic stenosis; AV, atrioventricular; FW, free wall; FWS, free wall strain; IVS, interventricular septum; LV, left ventricular; LVEDD, left ventricular end diastolic diameter; LVEDVi, left ventricular end diastolic volume indexed to body surface area; LVGLS, left ventricular global longitudinal strain; MR, mitral regurgitation; RVEDD, right ventricular end diastolic diameter; RVGLS, right ventricular global longitudinal strain; TR, tricuspid regurgitation.

[a]All segments were included in 191 patients and 1 segment was excluded in 16 patients due to visibility issues.

[b]All segments were included in 194 patients; 1 segment was excluded in 11 patients, and 2 segments were excluded in 2 patients due to visibility issues.

inferior STEMI. Peak Troponin T was 3.04 (1.14–5.35) ng/mL in the entire cohort. We found that TAPSE, RVGLS, FWS and LVGLS were significantly associated with peak Troponin T levels in the raw analysis. After further adjustment for LVGLS, the tested RV function parameters also demonstrated a statistically significant association with peak Troponin T levels. In line with these findings, we also observed significant associations between markers of RV function and creatine kinase muscle brain type levels (data not shown).

4 | DISCUSSION

The present study clearly demonstrates for the first time that RV strain parameters independently predict all-cause mortality in patients with inferior STEMI. FWS and RVGLS had the potential to predict all-cause mortality with a similar association as compared to TAPSE and remained significant after adjusting for a set of potential confounders. The optimal cut-off values to discriminate between good and bad survival according to the Youden

TABLE 3 Cox regression analysis assessing the univariate and multivariable associations of ventricular function and all-cause mortality.

	HR	95% CI	*p*-Value	HR	95% CI	*p*-Value	HR	95% CI	*p*-Value	HR	95% CI	*p*-Value
Variable	**Univariate**			**Multivariate**			**Multivariate**			**Multivariate**		
RVGLS	1.86	1.42–2.45	<.001	1.68	1.27–2.23	<.001						
FWS	1.82	1.41–2.35	<.001				1.56	1.21–2.00	<.001			
TAPSE	1.89	1.44–2.48	<.001							1.55	1.17–2.05	.002
Age[a]	1.58	1.29 – 1.93	<.001	1.46	1.16–1.83	.001	1.41	1.12–1.76	.003	1.33	1.05–1.69	.020
Male sex	.63	.36–1.13	.120	1.27	.65–2.50	.485	1.14	.59–2.19	.698	1.12	.58–2.16	.744
Comorbidity Score[b]	2.10	1.40–3.15	.003	1.48	.95–2.31	.084	1.42	.90–2.22	.128	1.54	.98–2.41	.061
			Log likelihood			**Chi-square**			***p*-Value**			**AIC**
Base model[c]			−238			27.1			<.001			482
Base model + RVGLS			−231			43.0			<.001			470
Base model + FWS			−232			42.1			<.001			472
Base model + TAPSE			−233			39.8			<.001			474

Note: The multivariable models contain age, sex, the comorbidity score and the respective ventricular functional parameter. Hazard ratios (HR) are reported as HR per 1 standard deviation increase. To enhance the comparability of parameters, an increase in HRs means more severe worsening of ventricular function (i.e. increase for strain parameters and decrease for tricuspid annular plane systolic excursion (TAPSE)).

Abbreviations: CI, confidence interval; FWS, free wall strain; HR, hazard ratio; RVGLS, right ventricular global longitudinal strain;

[a]Per 10 years.

[b]Comprising chronic kidney disease, hypercholesterolaemia and diabetes mellitus (one point each).

[c]Comprising age, sex and the comorbidity score.

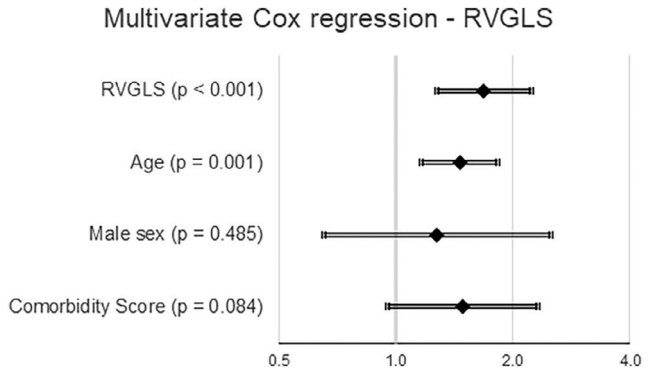

FIGURE 3 Forest plots of the adjusted Cox regression model for the right ventricular global longitudinal strain: RVGLS, right ventricular global longitudinal strain.

index were −13.5% for RVGLS, −15.1% for FWS and 1.4 cm for TAPSE, respectively. However, the risk of death was already elevated in the mid tertile of TAPSE between 2.0 and 1.6 cm, which could help to identify an intermediate risk group, whilst the risk in the mid tertile of RVGLS and FWS was still low and comparable to the first tertile. Moreover, we found significant associations between RVGLS, FWS and biochemical markers of myocardial damage.

The present study extends the current knowledge on echocardiographic strain measurements and survival after inferior myocardial infarction. While conventional measurements of RV function, including TAPSE and the RV fractional area change, have been previously identified as predictors of mortality in inferior STEMI,[23,24] the advanced speckle-tracking techniques FWS and RVGLS have so far only been demonstrated to independently predict a combined endpoint of major adverse cardiovascular events and mortality in inferior STEMI.[14] The present findings in patients with inferior STEMI are also in line with previous data showing an association of RV strain parameters and in-hospital mortality in a cohort of patients with diverse types of acute coronary syndromes.[7] One strength of the present study is the median long-term follow-up of 8.3 years, which is longer than in previous studies.[7,14,23,24]

Although RV function traditionally attracts less attention than LV function, the present findings in patients with inferior STEMI emphasize the important prognostic role of RV function. The detrimental effects of RV dysfunction are also known in numerous other cardiovascular pathologies, including chronic heart failure,[10] pulmonary hypertension,[11] pulmonary embolism,[12] and cardiac amyloidosis.[13]

Interestingly, all three assessed parameters of RV function, FWS, RVGLS and TAPSE, implied a roughly equal strength of association with dismal outcomes. According to the Akaike information criterion and the chi-square value, the multivariable models including either RVGLS, FWS or TAPSE had a comparable model fit, and c-statistics showed that the discriminatory ability to predict mortality was comparable across the 3 RV functional markers. While both strain parameters measure myocardial deformation by tracking speckles,[25] TAPSE is an M-mode measurement that describes the systolic displacement of the RV free-wall tricuspid valve annulus towards the RV apex.[26] The rather comparable prognostic potential might be explained by the fact that all three measures primarily assess RV longitudinal function. As RV muscle fibres predominantly align longitudinally, longitudinal function is the essential component of RV function and generates approximately 80% of the RV stroke volume.[27]

It might have been expected that RVGLS, owing to its global assessment of RV function, would be superior to TAPSE and FWS, which solely measure free wall function. A potential explanation for the rather comparable prognostic value might be the fact that RV infarction after RCA occlusion almost always affects the basal RV free wall areas near the atrioventricular groove, which are assessed by all three measures.[28,29] Of note, RCA occlusion and a right-dominant coronary system were present in the vast majority of patients with inferior STEMI in the present cohort, which is in line with previous studies.[30] This fact might have influenced the present results. To the best of our knowledge, specific data on RV function in patients with circumflex artery-related inferior STEMI and/or left-dominant systems are not yet available.

Contrary to FWS and TAPSE, RVGLS does not only cover the RV free wall, which mainly belongs to the RCA territory, but also septal segments and might therefore also include areas supplied by other coronary arteries. It is known that the RV is primarily supplied by the RCA, although little parts, especially in the anterior, anteroseptal and apical regions, may also be perfused by branches of the left anterior descending artery. In left dominant systems, parts of the RV, particularly the inferior free wall and the inferobasal septum, may also be supplied by the circumflex artery.[31]

All three parameters can be assessed with minor time effort, with a little time advantage for the TAPSE over the strain parameters. In contrast to TAPSE and RV tissue doppler imaging velocity, strain measurements are deemed to be more reproducible, as they do not depend on ninety-degree probe angling for an ideal signal.[9] In our study, the reproducibility of measurements of ventricular function was high, which is in line with previous analyses on test–retest variability of echocardiographic assessments of RV and LV function.[32] RVGLS and FWS have also demonstrated a higher degree of correlation with RVEF on cardiac magnetic resonance imaging as the gold standard for RV functional assessment.[9] In contrast to TAPSE, speckle tracking also enables the

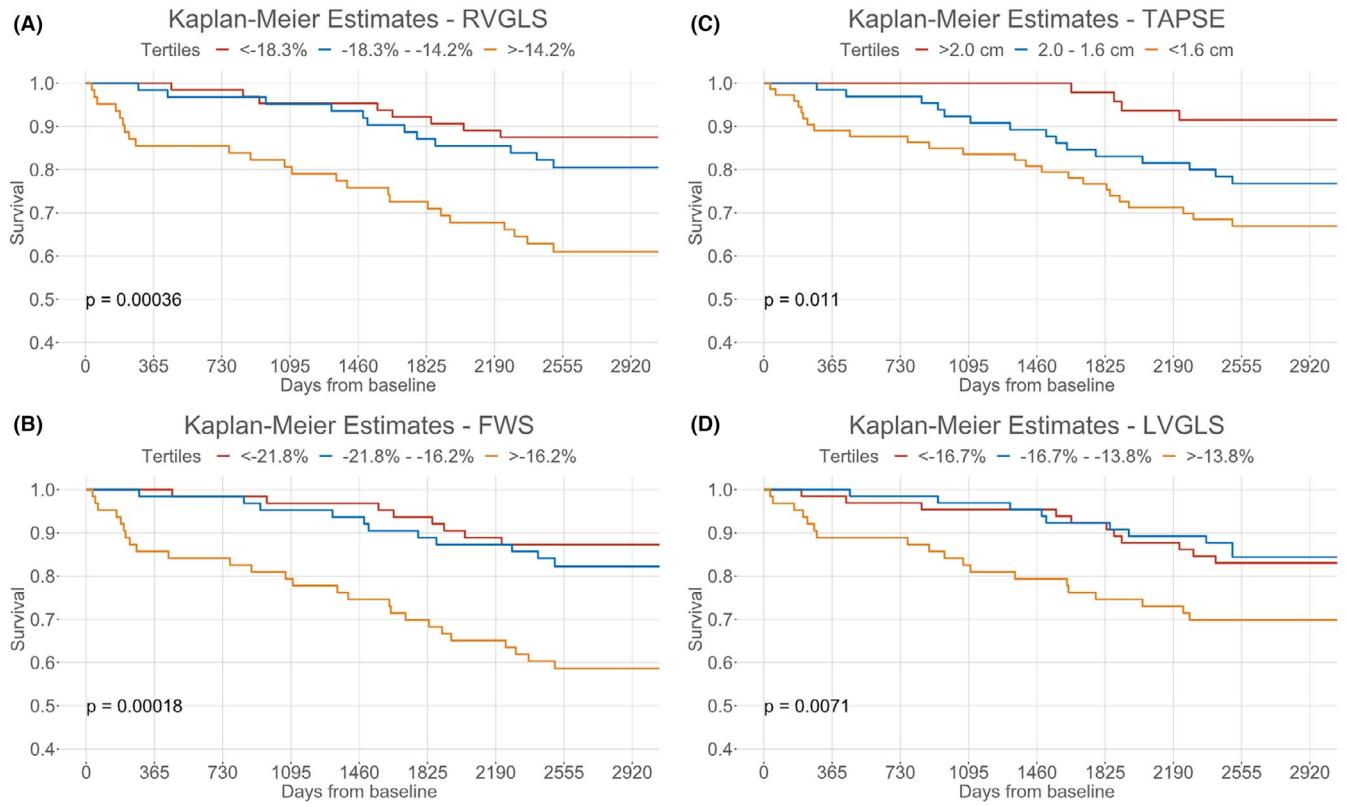

FIGURE 4 Kaplan–Meier estimates for survival stratified by tertiles of parameters of ventricular function; (A) RVGLS, right ventricular global longitudinal strain; (B) FWS, free wall strain; (C) TAPSE, tricuspid annular plane systolic excursion; (D) LVGLS, left ventricular global longitudinal strain.

assessment of regional dysfunction, as deformation can reliably be measured on a segmental level. This is another advantage over conventional methods for regional function assessment, such as Tissue Doppler Imaging, which measures velocities relative to the transducer and can therefore not reliably distinguish between healthy actively contracting segments and dysfunctional segments with only passive motion due to adjacent segment tethering.[8] In the present study, we also evaluated the prognostic value of segmental RV strain measures. Out of the 6 assessed segments, only the basal free wall segment and the septal mid-cavity segment independently predicted all-cause mortality in our analysis, whereas all other segments did not.

4.1 | RV dysfunction and prognosis

RV dysfunction is known to be linked to significant hemodynamic and electrical complications[33] which might explain the poorer prognosis of inferior STEMI patients with RV involvement. Cardiac output may be compromised by multiple mechanisms, including systolic RV dysfunction, tricuspid regurgitation, ventricular interdependence, as well as brady- and tachyarrhythmia. If the coronary occlusion is very proximal, usually in the RCA,[34] not only the RV branches but also the right atrial branches might be involved, causing also right atrial ischemia and consequently additional compromise of RV filling.[33,35] RV dysfunction also results in a decline of LV stroke volume due to a diminished transpulmonary blood delivery and reduced LV preload.[35] Furthermore, the ischemic RV myocardium eventually undergoes adverse remodelling, resulting in RV stiffness and diastolic dysfunction.[35] Accordingly, it is unsurprising that the prognosis of patients with inferior STEMI and a comparable left-sided infarction territory is worse in patients with RV involvement as compared to those without RV involvement.[36] On the other hand, it is known that impaired RV function is more common in patients with extended left-sided inferior STEMIs, as culprit lesions in the rather proximal part of the RCA are not only associated with a higher risk of a direct myocardial involvement of the RV but also with more left-sided myocardium at risk than distal lesions.[29,37] Indeed, utilizing cardiac magnetic resonance imaging in patients with inferior STEMI, Juul et al. have provided evidence that RV infarction was associated not only with worse RV function but also with a larger LV infarction size.[38] These patients also presented with more than double the Troponin T levels in serum.[38] This is in line with the

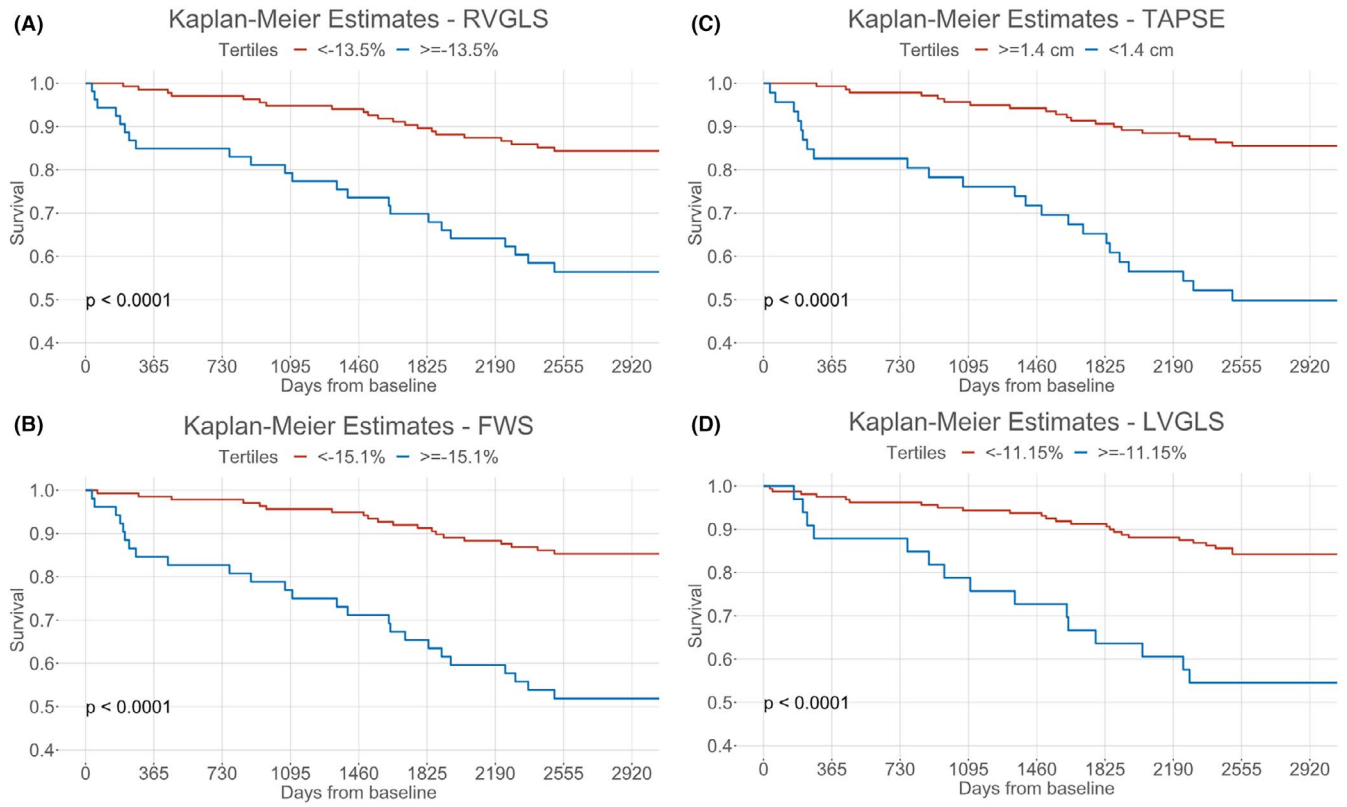

FIGURE 5 Kaplan–Meier estimates for survival stratified by cut-off values for parameters of ventricular function; Cut-offs according to Youden index. (A) RVGLS, right ventricular global longitudinal strain; (B) FWS, free wall strain; (C) TAPSE, tricuspid annular plane systolic excursion; (D) LVGLS, left ventricular global longitudinal strain.

TABLE 4 Linear regression analysis demonstrating the association between parameters of ventricular function and peak Troponin T levels.

	Coef.	95% CI	*p*-Value	Coef.	95% CI	*p*-Value
Variable	**Crude**			**Adjusted for LVGLS**		
TAPSE	−.909	−1.722 to −.096	.029	−.464	−1.332 to .403	.292
RVGLS	.172	.082 to .261	<.001	.122	.023 to .221	.016
FWS	.111	.046 to .176	<.001	.078	.007 to .149	.031
LVGLS	.297	.182 to .42	<.001			

Abbreviations: CI, confidence interval; FW, free wall; FWS, free wall strain; IVS, interventricular septum; LVGLS, Left ventricular global longitudinal strain; RVGLS, right ventricular global longitudinal strain, TAPSE, tricuspid annular plane systolic excursion.

present study, where RV dysfunction was related to higher peak Troponin T and creatine kinase levels, as well as poorer LVGLS, which suggests more extensive myocardial damage. The larger left-sided infarction size observed in the patients with RV dysfunction might also contribute to the poor prognosis of these patients. Importantly, RV dysfunction after inferior STEMI is not always caused by direct myocardial involvement of the RV due to ischemia, as RV dysfunction might also occur secondary to extensive LV myocardial damage. The subsequent decline in LV function may hamper RV function due to an increase in left-sided filling pressures and RV afterload, impaired contraction patterns caused by dyssynchronization, and also a diminished RV preload. Hence, global RV function during the early recovery period of inferior STEMI might be determined by several overlapping and sometimes also reversible phenomena and might reflect the summarized function of neighbouring necrotic, recovering and/or compensating hyper-functional areas, as well as pressure-overloaded areas. As illustrated by the presented Kaplan–Meier curves, patients with only moderately depressed RV function in the 2nd tertile still had a fairly good long-term outcome as compared to the 3rd tertile. This might reflect the potential for recovery of RV function in this group.

4.2 | Clinical implications

The present findings emphasize the importance of assessing parameters of RV (dys-)function in patients with inferior STEMI. Easily accessible parameters such as FWS, RVGLS and TAPSE might improve risk stratification and could help to identify vulnerable patients with poor prognosis. Although currently no specific treatment recommendations exist for the subset of STEMI patients with reduced RV function, these patients might benefit from a closer follow-up and intensified medical therapy in the postinfarction period in terms of individualized patient care. The addition of RV dysfunction to existing scoring systems in myocardial infarction may also refine their predictive utility.[39,40] With the emergence of artificial intelligence in health care, more complex prediction models utilizing data from several modalities, including echocardiography, could help to implement truly personalized risk assessment and therapy.[41]

4.3 | Limitations

The present study is a retrospective, single-centre study. Our patient cohort may not be representative of all patients with inferior STEMI due to differences in population genetics and epidemiology of coronary anatomy and would ideally be validated in an external patient cohort. As patients with echocardiographic imaging quality insufficient for strain analysis had to be excluded from the study, a bias with regard to patient selection and image acquisition cannot be fully excluded. Furthermore, ventricular function was assessed only at a single point in time during the first 3 days after myocardial infarction. Repeated strain measurements during follow-up might have helped to identify patients with only temporarily depressed RV function or with further deteriorating function and might have improved risk stratification. Finally, end-diastole was defined by the peak of the R wave on ECG, which is the standard setting of the GE EchoPAC AFI software. A definition of end-diastole by tricuspid valve closure could have been superior to the peak of the R wave to truly depict the mechanical output of the RV, especially in certain pathologies such as bundle branch block.

5 | CONCLUSION

RV dysfunction assessed by RV strain measurements and TAPSE independently predicts long-term mortality in inferior STEMI patients and is associated with biochemical parameters of myocardial damage. The easily applicable parameters of RV strain and TAPSE might help to improve risk stratification and to identify high-risk patients who need special attention.

AUTHOR CONTRIBUTIONS

Conceptualization: B.R., A.K.; Data curation: C.J., D.F., S.E., E.S., B.R.; Investigation: M.P., A.K., B.R.; Methodology: B.R., A.K.; Supervision: B.R.; Writing – original draft: M.P., A.K., C.J.; Writing – review & editing: M.P., A.K., C.H., A.N., I.L., T.B., B.R.

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CONFLICT OF INTEREST STATEMENT

None.

DATA AVAILABILITY STATEMENT

Data available upon reasonable request to the corresponding author.

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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