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Right ventricular longitudinal strain predicts acute kidney injury and short-term prognosis in patients with right ventricular myocardial infarction

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Abstract

Right ventricular (RV) systolic dysfunction due to acute myocardial infarction is associated with serious complications in the short-term. Acute kidney injury (AKI) is a frequent and unrecognized complication. This study aimed to assess whether RV longitudinal strain predicts AKI and short-term prognosis in patients with RV infarction. Prospective cohort of patients with RV infarction. RV function was evaluated with global and free wall right ventricular longitudinal strain (GRVLS and FWRVLS), tricuspid annular plane systolic excursion, and tricuspid S′ wave. The primary endpoint was AKI defined as an increase $\geq 50\%$ in serum creatinine and/or a decrease $\geq 25\%$ in glomerular filtration rate during follow-up at 7 days. The secondary endpoint was death from any cause at 30 days. We included 101 patients with RV infarction (male 67%, age 66 ± 11 years). During follow-up at 7 days, 40% of patients developed AKI. At 30 days, 8% of patients died. At univariate analysis, FWRVLS was significantly associated with AKI (Hazard ratio [HR] 1.11, 95% confidence interval [CI] 1.03–1.20, $p = 0.006$). At multivariate analysis, only age, temporary pacemaker implant, and FWRVLS remained as independent predictors of AKI (HR 1.05, 95% CI 1.02–1.08, $p = 0.002$; HR 2.12, 95% CI 1.11–4.07, $p = 0.023$; HR 1.10, 95% CI 1.02–1.19, $p = 0.018$, respectively). At 30 days, patients with FWRVLS $\geq -15.5\%$ showed a lower survival rate than those with lower strain (84 ± 6 vs. $97 \pm 2\%$, $p = 0.021$). In patients with RV infarction, FWRVLS was an independent predictor of AKI and was associated with higher mortality in the short-term.

Keywords Ventricular dysfunction · Right · Myocardial infarction · Acute kidney Injury · Echocardiography

Introduction

Patients with right ventricular (RV) infarction have a poor prognosis, even if the left ventricular ejection fraction (LVEF) is preserved [1]. Acute kidney injury (AKI) is a frequent and unrecognized complication.

Patients with RV dysfunction have a high risk of AKI for two reasons [2]. First, they have raised central venous pressure, and this can lead to a lower renal perfusion pressure [3]. Second, the low cardiac output is associated with higher systemic vascular resistance, and this contributes to lower renal perfusion [2]. AKI is associated with high mortality in the short-term [2, 4].

Echocardiography is a useful tool for evaluating RV function and can be performed at the bedside; however, there is little information to determine its prognostic utility in patients with RV infarction. Tricuspid annular plane systolic excursion (TAPSE), S′ tricuspid wave, the fractional area change (FAC), and Tei index are the parameters currently recommended by current guidelines for RV evaluation; right ventricular longitudinal strain (RVLS) also seems to be a promising parameter [5].

Because RVLS evaluates longitudinal myocardial tissue, which accounts for 80% of RV stroke volume [6], we hypothesized that RVLS might give prognostic information about short-term events, such as AKI and mortality. The

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study aimed to evaluate the association between RVLS and AKI and mortality in patients with RV infarction.

Materials and methods

A prospective cohort design of patients with ST-elevation acute myocardial infarction (MI) and RV involvement was used. Cases were included from 2015 to 2016 at a third-level Cardiology Hospital. Patients must fulfill a third universal definition of MI, plus ST-segment elevation of at least 0.1 mV in the V4R lead [7]. Patients with more than 24 h of ischemic time, LVEF < 40%, chronic kidney disease in substitutive therapy, and valvular disease equal to or more than moderate degree were excluded. According to our institutional protocol, patients with ischemia-to-reperfusion time ≤ 12 h were taken to percutaneous coronary intervention (PCI) if the door-to-balloon time was expected to be ≤ 90 min; otherwise, thrombolysis was administered, and then patients were taken to PCI either as pharmacoinvasive or rescue strategy. Patients with ischemia to reperfusion time higher than 12 h were not taken to coronariography. Failed reperfusion was defined as coronariography showing thrombolysis in myocardial infarction (TIMI) flow ≤ 1, or if the patient was not taken to coronariography and there were no electrocardiographic signs of reperfusion.

We did comprehensive echocardiograms at the bedside during the first 24 h after admission. Echocardiograms were acquired after PCI in all patients. All echocardiography images were digitally stored. Echocardiographic variables: TAPSE, tricuspid S′ velocity, FAC, Tei index, cardiac output and pulmonary vascular resistances were measured, according to the American Society of Echocardiography [5]. For RVLS, we looked for the best acoustic window to get images with the best quality, while keeping a frame rate of at least 50 frames per second, along with electrocardiogram synchronization. We got apical four-chamber views of sufficient quality by placing the transducer medially and one intercostal space either higher or lower. In all cases, parameters were measured by the average of three consecutive beats. RVLS was analyzed offline with QLAB version 10.5 (Philips Healthcare, Andover, MA). RVLS analyses consisted of a semi-automative process in which three points were selected: tricuspid annular plane with the free wall, the interventricular septum, and RV apex. When necessary, the RV endocardial border was delimited with special emphasis, avoiding strong pericardium speckles. Visual inspection for strain curves was done for appropriate tracking of the myocardium. We defined GRVLS as the average of seven segments analyzed, and FWRVLS as the average of three free wall segments (Fig. 1).

Regarding right atrial pressure, in this study observational study, placement of a central venous catheter for right atrial
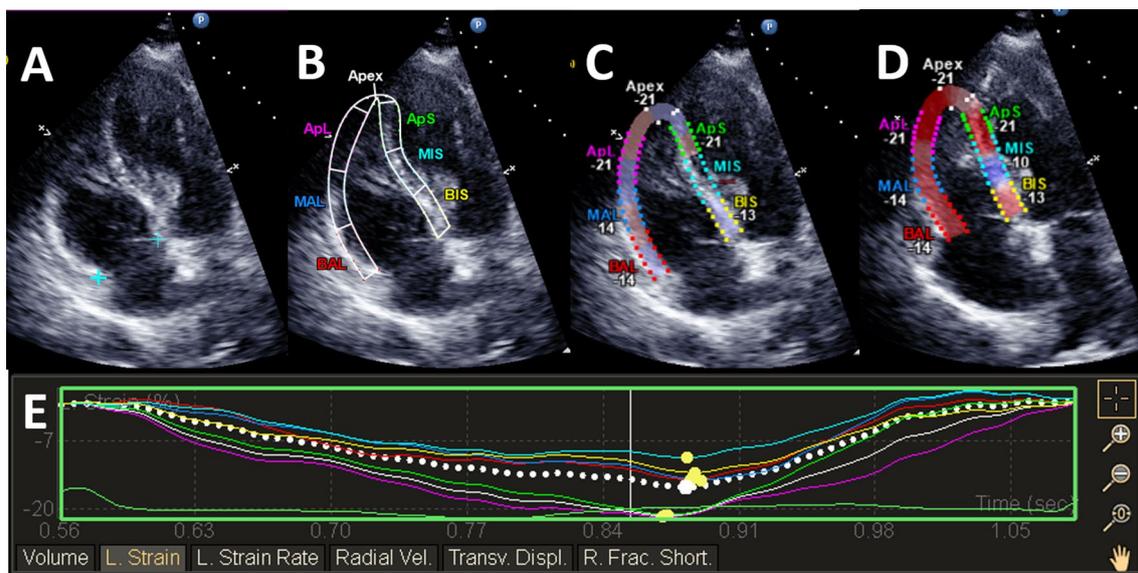

Fig. 1 Representative right ventricular longitudinal strain images. Global right ventricular longitudinal strain (GRVLS) was measured by selecting three points. **a** Selection of union of tricuspid valve with interventricular septum and union of tricuspid valve with right ventricular free wall, also, selection of right ventricular apex was done. **b** Software automically detects the right ventricle. **c** and **d** End-diastole and end-systole images respectively. Software shows longitudinal strain of seven segments. **e** Inspection of curves from the seven segments showing adequate tracking. We defined GRVLS as the average of the seven segments analyzed, three from de interventricular septum, one from the apex and three from the right ventricular free wall. In this case GRVLS was −15.14%. We defined free wall right ventricular longitudinal strain (FWRVLS) as the average of the three segments from the free wall. In this case was −16.3%

pressure measurement was not performed in every patient. In patients in whom the treating physician decided to place a central venous catheter, we collected the average blood pressure in mmHg, as previously described [8].

We compared echocardiographic variables with invasive parameters of the right ventricular function in 45 patients in whom central venous pressure was measured at the same time that the echocardiographic examination was done.

Demographic and clinical data were collected through patient interviews (or relatives), and by reviewing medical records. Blood samples were extracted from patients when arriving at the emergency department and then every 24 h for creatinine measurements. Investigators who were unaware of the echocardiography determined AKI and death. Creatinine measurements and clinical evaluations were done every day to achieve follow-up up to day 7 after the MI, then another clinical evaluation up to hospital discharge, and finally a phone call on day 30. We defined AKI as an increase $\geq 50\%$ in serum creatinine and/or a decrease $\geq 25\%$ in glomerular filtration rate during follow-up at 7 days, which is in line with international guidelines [9]. To determine mortality, we followed patients for 30 days after infarction.

Because we analyzed survival data, we estimated time to the event as follows: time to AKI was calculated by subtracting the date the patient fulfilled the criteria of AKI minus the date of the infarction; patients who did not suffer AKI at 7 days were censored. Time to death was calculated similarly; patients who survived at least 30 days were censored.

Informed consent was obtained from each patient, and the study protocol conforms to the ethical guidelines of the 1975 Declaration of Helsinki as reflected in a priori approval by the Cardiology Hospital Ethical Committee.

We assessed reproducibility by selecting ten random echocardiographic studies that were recorded as raw data for intra- and inter-observer variability. Observers were blinded to previous information; for intra-observer variability, the same observer evaluated raw echocardiographic data at least 1 month after the first evaluation.

The sample size was estimated according to data from Testani et al., who showed that patients with RV dysfunction have three times the risk of AKI compared to patients without RV dysfunction [10]. We estimated that with a hazard ratio (HR) of 3.0 and a standard deviation of 0.5, we would need 35 events of AKI to reach 90% of power to show a higher risk of AKI in patients with right ventricular dysfunction. In the study of Tandon et al., the probability of AKI after RV MI was 50%, so we determined that we would need to include at least 70 patients [11].

Statistical analysis

Descriptive analyses are reported as the mean and standard deviation or with median and interquartile range. Absolute frequencies and percentages are given for qualitative variables. Groups were compared using the Student's t-test for independent groups or χ^2 test. Pearson's correlation coefficient was used for the association of continuous variables. Receiver operating characteristic (ROC) curves were used to determine the diagnostic accuracy of echocardiographic variables to predict AKI. Cox proportional hazard models were used for prediction of AKI. HR and 95% CI are reported. We fit a multivariable Cox proportional hazard model with stepwise backward-selection; entry criteria was association at univariate analysis with a p-value < 0.25; the exit criteria was a p-value > 0.05. Kaplan–Meier AKI-free survival rate and overall survival rate were constructed and tested with log-rank p. We evaluated reproducibility with intra-class correlation coefficient and Bland–Altman analysis. A two-tail p-value ≤ 0.05 was considered significant. We used Stata 12 (StataCorp LP, College Station, TX) and SPSS version 22 (IBM, Chicago, IL) for statistical analysis.

Results

From 2016 to 2017, 1095 patients were admitted with ST-segment elevation acute MI. Of this group, 460 patients had inferior MI, and 106 patients fulfilled RV infarction criteria; five patients were excluded (three had LVEF less than 40%, and two had end-stage chronic kidney disease). One-hundred-one patients were included (male 67%, aged 66 ± 11 years) in the study. Complete echocardiographic data were obtained for all included patients. No patients were lost in follow-up. Baseline characteristics and echocardiographic variables are summarized in Tables 1 and 2, respectively.

The median ischemic time (from the onset of symptoms to reperfusion) was 6 h. Forty-three patients received thrombolysis as the primary reperfusion strategy; 22 were taken to rescue PCI and 15 to a pharmaco-invasive strategy; the remaining six patients were not taken to PCI because of long ischemic time. Fifty-three patients were taken to primary PCI. Overall, PCI was done in 89% of patients; the remaining were not candidates for PCI because of long ischemic time. Among patients taken to PCI, 77% achieved reperfusion (TIMI flow 3 or a change from TIMI 0 to TIMI 2 flow). Of the entire cohort, the percentage of patients with a non-reperfused infarction was 30%.

FWRVLS and TAPSE were the RV echocardiographic parameters with the strongest associations with central venous pressure obtained invasively (R = 0.50 and − 0.55, respectively; both $p < 0.001$). See Table S1 of the Supplementary Appendix.

The ROC curve revealed that TAPSE and FWRVLS had the highest area under the curve for predicting AKI (0.70, 95% CI 0.59–0.81 and 0.68, 95% CI 0.57–0.79; respectively, both $p = 0.001$). See Table 3.

Table 1 Baseline characteristics of patients

Variable	Total population (n = 101)
Age (years)	66 ± 11
Male	68 (67%)
Current/ex-smoker	68 (67%)
Diabetes	53 (52%)
Hypertension	68 (67%)
Dyslipidemia	37 (37%)
Coronary artery disease	14 (14%)
Myocardial infarction	12 (12%)
Aspirin	11 (11%)
P2Y12 Inhibitors	5 (5%)
Angiotensin converting enzyme inhibitor or angiotensin receptor blocker	56 (55%)
Beta blockers	18 (18%)
Statins	23 (23%)
Systolic blood pressure (mmHg)	123 ± 29
Heart rate (bpm)	76 ± 17
Hemoglobin (g/dl)	14.2 (13.4–15.1)
Creatinine (mg/dl)	1.0 (0.85–1.4)
Glomerular filtration rate (ml/min/1.73 m^2)	71 (46–89)
Treatment	
Ischemic time (h)	6 (3–10)
Aspirin	101 (100%)
P2Y12 inhibitors	96 (95%)
Fractioned-heparin	100 (99%)
Statins	100 (99%)
Abciximab	16 (16%)
Diuretics	29 (29%)
Angiotensin converting enzyme inhibitor or angiotensin receptor blocker	21 (21%)
Thrombolysis	43 (43%)
PCI	90 (89%)
Primary PCI	53/90 (59%)
Rescue PCI	22/90 (24%)
Pharmaco-invasive strategy	15/90 (17%)
Failed PCI	20 (22%)
Dyes (ml)	150 (110–185)
Number of diseased vessels	
1	29/90 (32%)
2	35/90 (39%)
3	26/90 (29%)
Initial TIMI flow	
0	51/90 (57%)
1	8/90 (9%)
2	11/90 (12%)
3	20/90 (22%)
Final TIMI flow	
0	13/90 (14%)
1	8/90 (9%)
2	6/90 (7%)

Table 1 (continued)

Variable	Total population (n = 101)
3	63/90 (70%)
Complications at follow-up	
AKI	40 (40%)
Temporary pacemaker implant	33 (33%)
Shock	45 (45%)
Atrial fibrillation	12 (12%)
Death	8 (8%)

Data are n (%); mean ± standard deviation or median (quartile 1–quartile 3)

AKI acute kidney injury, *PCI* percutaneous coronary intervention, *TIMI* thrombolysis in myocardial infarction

Table 2 Echocardiographic variables

Variable	Total population (n = 101)
LVEF (%)	53 ± 8
Cardiac index (l/min/m^2)	2.6 ± 0.6
Mass (g/m^2)	88 ± 21
Left atrial volume (ml/m^2)	25 (21–29)
E wave (cm/seg)	78 (64–91)
A wave (cm/seg)	81 (63–96)
E/E′	10.7 (8.8–12.5)
Pulmonary artery systolic pressure (mmHg)	29 (26–36)
Inferior vena cava diameter (mm)	20 (18–23)
Right atrial area (cm^2)	14.9 (13–17)
Right ventricular dilatation	32 (32%)
Tei index	0.54 (0.41–0.57)
FAC (%)	35 (28–42)
TAPSE (mm)	18 (15–20)
Tricuspid S′ (cm/seg)	10.7 (9.1–12.4)
GRVLS (%)	− 16.3 (− 19.2–13.4)
FWRVLS (%)	− 17.1 (− 19.5–12.7)

Data are mean ± standard deviation or median (quartile 1–quartile 3)

FAC fractional area change, *FWRVLS* free wall right ventricular longitudinal strain, *GRVLS* global right ventricular longitudinal strain, *LVEF* left ventricular ejection fraction, *TAPSE* tricuspid plane annular systolic excursion

Different RV echocardiographic parameters showed different proportions of RV dysfunction: 24% of patients had TAPSE values > 15 mm; 33% of patients had S′ tricuspid wave less than 9.5 cm/s; 42% of patients had FAC less than 35%. Since there is no universal cut-off value for RVLS, the ROC curve was done showing an optimal cut-off value of − 15% for predicting AKI. We chose a cut-off value of − 15.5% as it corresponds to three standard deviations under the mean, reported by the American Society of Echocardiography [5], and to maintain uniformity with previous studies

Table 3 Results of receiver-operating characteristic curves comparing different echocardiographic parameters for their accuracy to predict acute kidney injury

Parameter	AUC	95% CI	p	Cutoff	Sensitivity	Specificity
TAPSE	0.70	0.59–0.81	< 0.001	15 mm	43	88
GRVLS	0.65	0.54–0.76	0.008	− 15.5%	51	70
FWRVLS	0.68	0.57–0.79	0.001	− 15.5%	58	75
Tricuspid S′	0.67	0.55–0.78	0.004	9.5 cm/seg	50	79
FAC	0.61	0.48–0.72	0.077	35%	58	59
Tei index	0.67	0.56–0.78	0.002	0.55	58	20

AUC area under the curve, *FAC* fractional area change, *FWRVLS* free wall right ventricular longitudinal strain, *GRVLS* global right ventricular longitudinal strain, *TAPSE* tricuspid plane annular systolic excursion

[12]; 41% of patients had GRVLS > − 15.5%, while 38% of patients had FWRVLS > − 15.5%. With regards to reperfusion, patients with failed reperfusion showed a trend towards higher FWRVLS compared to patients with successful reperfusion (− 15.4 ± 5.0 vs. − 17.1 ± 3.9, $p = 0.077$).

The AKI incidence was 40% at 7 days. In our univariate analysis, older age, longer ischemic time, temporary pacemaker implant, and most RV echocardiographic parameters showed significant associations with AKI. However, after multivariate analysis, only age (HR 1.05, 95% CI 1.02–1.08, $p = 0.002$), temporary pacemaker implant (HR 2.13, 95% CI 1.11–4.07, $p = 0.023$), and FWRVLS (HR 1.10, 95% CI 1.02–1.19, $p = 0.018$) remained as independent predictors of AKI (Table 4).

The net reclassification index was 54% (95% CI 15–94, $p = 0.008$) when adding FWRVLS to the full model that included age and temporary pacemaker implant, suggesting that FWRVLS improved the risk stratification of the full model. The integrated discrimination index was 7.6% (95% CI 2.7–12.6, $p = 0.003$); the IDI can be viewed as a difference between improvement in average sensitivity and any potential increase in average "one minus specificity" [13].

When FWRVLS was included as a dichotomous variable in the same multivariable analysis (cutoff − 15.5%), it remained as an independent predictor (HR 2.46, 95% CI 1.30–4.70, $p = 0.006$).

Patients with higher FWRVLS (> − 15.5%) had a lower AKI-free survival rate compared to patients with lower FWRVLS (72 ± 6% vs. 38 ± 8% $p < 0.001$, respectively) (Fig. 2).

Mortality incidence was 8% at 30 days. Patients with FWRVLS > − 15.5% had a lower survival rate compared to patients with lower FWRVLS (84 ± 6% vs. 97 ± 2%, $p = 0.021$, respectively) (Fig. 2).

Echocardiographic variables showed good intra- and inter-observer reproducibility: intraclass correlation coefficient for FWRVLS was 0.95 (95% CI 0.83–0.99, $p < 0.001$) and 0.92 (95% CI 0.68–0.98, $p < 0.001$) for intra-observer and inter-observer, respectively. Bland–Altman comparison showed a mean difference of 0.44 and − 2.41 for intra and inter-observer, respectively (Table 5).

Discussion

In this study, FWRVLS was an independent predictor of AKI and patients with higher FWRVLS had a lower survival rate at 30 days. FWRVLS was strongly associated with both central venous pressure and RV myocardial contractile performance, which are markers of preload and RV cardiac output respectively. Thus, this echocardiographic parameter accurately identified patients with a high risk of developing AKI, a complication that occurs due to high central venous pressure and low renal perfusion. In the same way, a severely reduced FWRVLS was a marker of higher short-term risk of mortality; this might be explained by the high incidence of complications that these patients suffer as a consequence of this poor hemodynamic profile.

As previously described, patients with right ventricular dysfunction have a higher risk of AKI due to raised central venous pressure and low cardiac output. Mielniczuk et al. found that patients with higher mean right atrial pressure had a higher incidence of worsening renal function, and worsening renal function was an important marker of in-hospital death [3]. Because FWRVLS showed a high correlation with central venous pressure, this might explain why it was so sensitive to predict AKI and death. In fact, the association between FWRVLS and central venous pressure has already been reported in these patients [14]. Also, RVLS evaluates longitudinal myocardial tissue, which accounts for 80% of RV stroke volume [6], and longitudinal strain is associated with myocardial contractile performance and worse RV systolic dysfunction in patients with RV MI affecting the proximal right coronary artery [15, 16]. In the same way, RVLS has been shown to be a predictor of adverse cardiovascular outcomes in patients with reduced and preserved LVEF in the long term [12].

In the study of Tandon et al., lower RV longitudinal function (evaluated with TAPSE) was significantly associated with AKI development during a 3-day follow-up [11]. We found similar results with FWRVLS. Because other predictors of AKI have been described [17–19], we tested whether FWRVLS improved the risk stratification of the

Table 4 Univariate and multivariate analysis for AKI prediction

	Univariate		Multivariate*	
	HR (95% CI)	p value	HR (95% CI)	p value
Age (years)	1.05 (1.02–1.08)	0.002	1.05 (1.02–1.08)	0.002
Male	1.17 (0.60–2.31)	0.646	–	–
Current/ex-smoker	0.90 (0.47–1.72)	0.750	–	–
Diabetes	0.98 (0.53–1.83)	0.952	–	–
Hypertension	1.16 (0.59–2.28)	0.667	–	–
Dyslipidemia	0.69 (0.35–1.36)	0.287	–	–
Myocardial infarction	1.06 (0.42–2.71)	0.901	–	–
Systolic blood pressure (mmHg)	0.99 (0.98–1.01)	0.738	–	–
Ischemic time (h)	1.02 (1.01–1.03)	0.024	–	–
No. of diseased vessels				
1	Reference			
2	0.69 (0.32–1.46)	0.326	–	–
3	0.66 (0.29–1.54)	0.339		
Dyes (ml)	1.00 (0.99–1.01)	0.195	–	–
Percutaneous coronary intervention	1.11 (0.39–3.11)	0.845	–	–
Failed reperfusion	1.49 (0.79–2.84)	0.219	–	–
Diuretics	1.56 (0.82–2.96)	0.174	–	–
Angiotensin converting enzyme inhibitor or angiotensin receptor blocker	1.12 (0.53–2.35)	0.767	–	–
Temporary pacemaker	2.70 (1.45–5.04)	0.002	2.13 (1.11–4.07)	0.023
LVEF (%)	0.96 (0.92–0.99)	0.023	–	–
E/E′	1.05 (0.95–1.16)	0.300	–	–
Tei index	4.16 (1.58–10.9)	0.004	–	–
FAC (%)	0.99 (0.96–1.01)	0.228	–	–
TAPSE (mm)	0.89 (0.83–0.95)	0.001	–	–
Tricuspid S′ (cm/seg)	0.86 (0.77–0.96)	0.010	–	–
GRVLS (%)	1.10 (1.02–1.19)	0.021	–	–
FWRVLS (%)	1.11 (1.03–1.20)	0.006	1.10 (1.02–1.19)	0.018

AKI acute kidney injury, *CI* confidence interval, *FAC* fractional area change, *FWRVLS* free wall right ventricular longitudinal strain, *GRVLS* global right ventricular longitudinal strain, *HR* hazard ratio, *LVEF* left ventricular ejection fraction, *TAPSE* tricuspid annular plane systolic excursion

*The full model included the following variables: age, ischemic time, dyes, failed reperfusion, temporary pacemaker implant, LVEF, Tei index, FAC, TAPSE, FWRVLS and GRVLS; only variables with a *p* value < 0.05 were retained

base model. As described above, FWRVLS added prognostic information.

In clinical practice, TAPSE is the most frequent measure used to assess RV function given its simplicity and reproducibility. A study that compared several RV systolic function parameters with RV ejection fraction by cardiac magnetic resonance showed RVLS as having the best correlation [20]. This supports our results about the high correlation of FWRVLS with hemodynamic parameters, such as central venous pressure and the usefulness of FWRVLS in the prediction of AKI and death.

It is unusual to perform cardiac magnetic resonance imaging in a patient who has just suffered ST-elevation acute MI. Given that RVLS shows a good correlation with RV ejection fraction, and it detects regional myocardial dysfunction in

patients with acute MI [21], it might explain its sensitivity in predicting AKI and death in the cohort we studied. In fact, a very interesting study showed that in patients with RV MI, a persistent impairment in RV strain is not identified by cardiac magnetic resonance-derived ejection fraction or conventional echocardiographic parameters [22]. Also, an RV longitudinal strain higher than − 15.8% was very sensitive and specific for RV scar prediction [23].

LVEF is a well-known predictor of poor prognosis in patients with ST-elevation MI [24]. However, in this study, patients had preserved or mid-range LVEF. This might explain why LVEF was not associated with AKI after multivariate analysis, whereas RV systolic function did show an association. In fact, an association between RV systolic dysfunction and long-term prognosis in patients
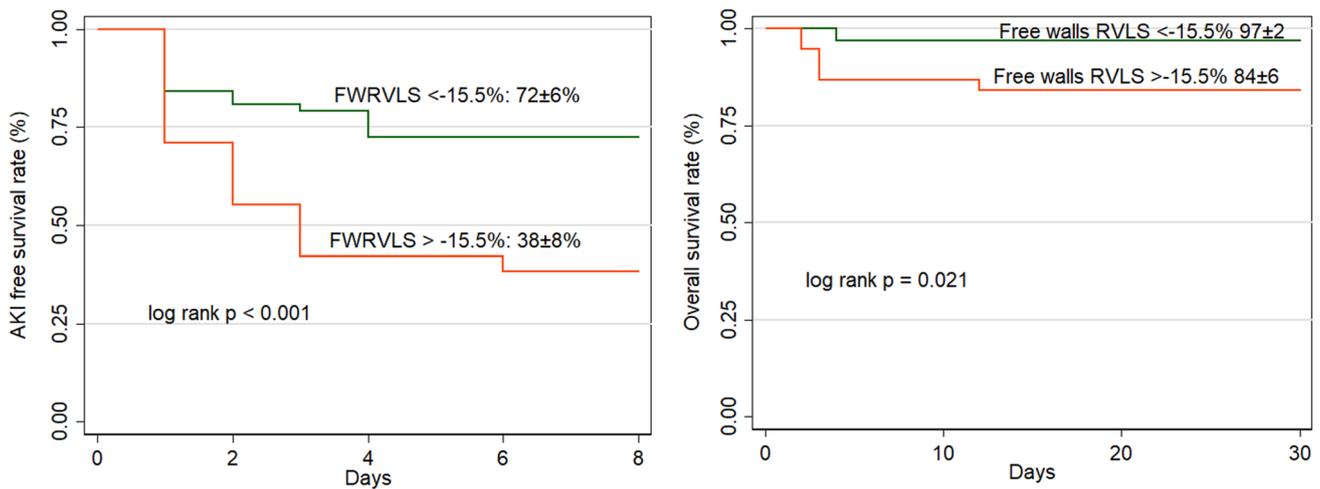
Fig. 2 AKI-free survival curve and overall survival curve by Kaplan–Meier analysis. Left: patients with higher free wall right ventricular longitudinal strain (FWRVLS > − 15.5%) showed lower acute kidney injury (AKI) free survival rate ($38 \pm 8\%$ vs. $72 \pm 6\%$, $p < 0.001$).

Right: patients with higher free wall right ventricular longitudinal strain (FWRVLS > − 15.5%) showed lower overall survival rate ($84 \pm 6\%$ vs. 97 ± 2, $p = 0.021$)

Table 5 Intraclass correlation coefficient and results of Bland–Altman comparison

A	Intraobserver			Interobserver		
	ICC	95% CI	*p* value	ICC	95% CI	*p* value
FWRVLS	0.952	0.834–0.986	< 0.001	0.919	0.675–0.980	< 0.001
GRVLS	0.950	0.799–0.988	< 0.001	0.954	0.815–0.989	< 0.001
TAPSE	0.974	0.910–0.993	< 0.001	0.960	0.847–0.990	< 0.001
S′	0.995	0.984–0.999	< 0.001	0.995	0.983–0.999	< 0.001
B	Mean difference	95% CI		Mean difference	95% CI	
FWRVLS	0.439	− 0.925–1.912	–	− 2.413	− 3.774–1.052	–
GRVLS	0.860	− 0.491–2.211	–	− 2.23	− 3.466–0.994	–
TAPSE	− 0.060	− 1.405–1.285	–	0.940	− 0.605–2.485	–
S′	− 0.230	− 0.622–0.162	–	0.460	− 0.035–0.885	–

A: intraclass correlation coefficient, B: mean difference in Bland–Altman comparison

FWRVLS free wall right ventricular longitudinal strain, *GRVLS* global right ventricular longitudinal strain, *TAPSE* tricuspid plane annular systolic excursion

with myocardial infarction and preserved ejection fraction has been reported [25].

Park et al. found that RVLS at a cut-off value of − 15.5% was an independent predictor of a worse prognosis in patients with inferior MI [12]. As described above, in our study, the same cut-off value was also an independent predictor of AKI; patients with an FWRVLS ≥ − 15.5% had 2.5-times the risk of AKI compared to patients with lower FWRVLS.

In a recent cohort of patients with inferior MI, Kanar et al. reported that RVLS was an independent predictor of early mortality with a cut-off of − 14% [26]. Hence, our

results are consistent with previous reports supporting the utility of RV systolic evaluation.

Interestingly, in our study, dyes did not show an association with AKI. Although there are many possible explanations, we suspect that the main reason for this finding is that most patients received the same amounts of dyes. Likewise, failed reperfusion was not significantly associated with AKI in our multivariate analysis; a possible explanation for this is that although patients with failed reperfusion had a worse RV systolic function, only patients with severe RV systolic dysfunction have a sufficient hemodynamic impact to cause AKI. On the other hand, the right ventricular pacing did

show an independent association with AKI; as previously reported, the lack of atrioventricular synchrony in patients with RV MI can reduce cardiac output and lead to deleterious effects in these patients [27].

The cohort of our study had a high incidence of complications. This might be because of the high incidence of right ventricular dysfunction; in this study, 38% of patients had low FWRVLS (higher than − 15.5%), while in a recent cohort of MI patients the proportion of patients with low FWRVLS was only 11% [28]. These data help to understand the poor prognosis of patients with RV infarction and the need for identifying patients with a higher risk for developing serious complications in the short-term. FWRVLS was independently associated with a worse prognosis, and thus, this echocardiographic parameter might help in terms of identifying very sick patients.

This is a single-center observational study and is, therefore, susceptible to bias. However, prospective follow-up helped to control for potential confounding variables, such as LVEF and chronic kidney disease. We acknowledge the lack of hemodynamic parameters in all patients, which predict renal outcomes in RV MI patients [8]. Given that the study was done in a reference center, patients with an ischemic time greater than 24 h could not be included. Also, very sick patients might die before being transferred and were, therefore, not included. Nevertheless, many patients were in a critical condition and were still included in the study. So we could include all patients, we performed a thorough echocardiogram examination, although this might not be possible in the current clinical practice (as it takes an extended time to get sufficient high-quality images). RVLS was analyzed assuming that the left ventricle algorithm could be appropriate to the right ventricle; although this analysis could have some limitations, it has been used in other studies [29]. AKI diagnosis was made by serum creatinine changes, although there might be patients who suffered some degree of kidney damage without having elevated creatinine values.

Conclusion

FWRVLS was measured in all patients with high reproducibility and was predictive for AKI, even after multivariate analysis. Patients with higher FWRVLS showed lower overall survival rate at 30 days.

Compliance with ethical standards

Conflict of interest We report that we have no relationships to disclose, relevant to the contents of this paper.

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