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Evaluation of right ventricular dyssynchrony in patients with acute inferior myocardial infarction and its relation with mortality

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Abstract

Purpose: The aim of this study was to evaluate right ventricle (RV) dyssynchrony and its relation with mortality using speckle-tracking echocardiography (STE) in patients with acute inferior myocardial infarction (IMI).

Methods: One hundred and fifty-eight consecutive patients with acute IMI treated with primary percutaneous coronary intervention, and 44 healthy subjects were included. RV myocardial involvement (RVMI) was defined as an elevation >1 mm in V1 or V4R and/or the presence of a culprit lesion at the proximal portion of the first RV marginal branch after reviewing coronary angiography. Patients were followed for 3 years to determine the cardiovascular mortality.

Results: Overall, 70 patients with IMI had RVMI. IMI patients had significantly higher RV peak systolic longitudinal strain dyssynchrony (PLSSD) index, lower peak longitudinal systolic strain (PLSS), longer time to PLSS, and time to PLSS differences compared to healthy controls while the patients with RVMI had significantly worse values compared to patients without RVMI and healthy controls. Twenty-seven patients (17.1%) died within 2 years. RVMI was more prevalent in mortality group, and they had significantly higher RV PSSD index, whereas they had lower RV free wall PLSS and longer time to PLSS differences. Receiver operating characteristics (ROC) analysis revealed that a RV PLSSD index > 65 ms predicted mortality with a sensitivity of 88.9% and specificity of 71.8% in IMI patients.

Conclusions: Intra- and inter-ventricular dyssynhcrony may develop in patients with acute IMI, especially in those with RV involvement, which might have a negative effect on the prognosis of these patients.

KEYWORDS

cardiovascular mortality, inferior myocardial infarction, right ventricular infarction, speckle-tracking echocardiography, ventricular dyssynchrony

1 | INTRODUCTION

The mortality rate is high in patients with myocardial infarction despite current invasive treatment strategies.[1] Acute right ventricle (RV) ischemia leading to RV myocardial infarction is a complication in nearly half of the patients suffering acute inferior myocardial infarction (IMI), resulting in RV dysfunction.[2] Although RV function usually returns to normal in the long term (similar to stunning), the impairment or delay of both RV and left ventricle (LV) functions has been shown to be associated with an increase in all cause of mortality and morbidity.[3,4]

While the assessment of both RV and LV mechanical activation is valuable for determination of prognosis in IMI, the evaluation of RV function can be somewhat more difficult than LV because of the complex RV anatomy.[5] The recent introduction of peak longitudinal systolic strain (PLSS), time to PLSS, and PLSS dyssynchrony (PLSSD) indexes using STE have provided an objective means to quantify intra-ventricular and inter-ventricular dyssynchrony with improved accuracy and greater reproducibility than conventional two-dimensional echocardiography (2DE) and tissue Doppler imaging.[6] A number of studies have used STE to evaluate the LV intraventricular dyssynchrony in patients with acute myocardial infarction.[7,8] However, there is no information concerning the assessment of inter-ventricular dyssynchrony and also RV intraventricular dyssynchrony in patients after acute IMI. The aim of this study was to evaluate RV dyssynchrony and its relation with cardiovascular mortality in patients with acute IMI.

2 | MATERIALS AND METHODS

2.1 | Study population

One hundred and fifty-eight consecutive patients with acute ST segment elevation IMI were included in the study. Acute ST segment elevation IMI was defined as presence of acute ST segment elevation on leads DII, DIII, and AVF with clinical symptoms associated with elevation of cardiac biomarkers. RV myocardial involvement (RVMI) was defined as an elevation >1 mm in V_1 or V_4 R within 12 hours of symptom onset and/or the presence of a culprit lesion at the proximal portion of the right coronary artery before the first RV marginal branch after reviewing coronary angiography films. Patients with poor echogenicity, severe valvular heart disease, congestive heart failure (ejection fraction < 50%), history of coronary artery disease, atrial fibrillation, permanent pacemaker, bundle branch block, and malignancy were excluded. The present study complies with the principles outlined in the Declaration of Helsinki. The study was approved by the local Ethics Committee, and written informed consent was obtained from all patients and healthy subjects.

All patients underwent a complete cardiac examination including patient history and demographic data. All patients were revascularized with primary percutaneous coronary intervention (PCI) within 30 minutes as recommended by the European Society of Cardiology ST elevated myocardial infarction treatment guidelines.[9] Thrombolysis in myocardial infarction (TIMI) flow was evaluated after PCI by a well-trained cardiologist.

Two-dimensional echocardiography (2DE) was performed < 48 hours after admission, and echocardiographic data were analyzed for the assessment of ventricular dyssynchrony with speckle-tracking imaging. Patients were followed prospectively for cardiovascular mortality for 3 years.

2.2 | Echocardiographic evaluation

Two-dimensional echocardiography measurements were performed in accordance with the recommendations of the European Association of Cardiovascular imaging using an ultrasound system (IE33, Philips Medical Systems).[10] Standard echocardiographic views (parasternal long- and short-axis, apical 4- and 2-chamber) were obtained using a 3.5 MHz transducer in all participants.

One independent cardiologist performed STE postprocessing analysis using the QLAB Philips offline software (Philips Healthcare Medical Imaging System performed the STE analysis). They recorded three consecutive cardiac cycles in DICOM format for each view with a frame rate above 50 per second. The region of interest (ROI) was obtained by tracing the RV endocardial borders at the level of the septum and the free wall in a still frame at end-systole.[11] The region of interest was adjusted to cover at least 90% of the myocardial wall thickness. If first tracking is thought to be suboptimal, other retracings were manually or semi-automatically performed. However, after three retracings the nontracking segments were excluded. If more than three of six segments had poor tracking quality, the study was excluded. 3.7% of the segments were excluded from STE analysis due to inadequate endocardial border tracking in our study. Longitudinal strain curves were obtained for 6 RV segments (the basal, mid, and apical segments of the RV free wall and inter-ventricular septum [IVS]), and the longitudinal strain curves of the LV lateral wall were obtained by repeating the same analysis. The extent of myocardial deformation (defined as PLSS) was expressed as a percentage of the longitudinal shortening in systole compared with diastole for each segment of interest. The temporal pattern of RV mechanical contraction was evaluated as the time needed to reach peak strain (time to PLSS) using the beginning of the QRS complex as a reference point. Inter-ventricular synchronicity was defined as the maximum difference in time to PLSS among IVS, LV lateral wall, and the free RV wall (Figure 1).[12] RV peak longitudinal systolic strain dyssynchrony (PLSSD) index, which presented RV synchronicity, was derived from the standard deviation of the times from QRS beginning to PLSS of the six segments.

The STE analysis of randomly selected 20 patients was repeated 1 month later by well-trained two observers in order to assess intra- and inter-observer variability, which was calculated as the average difference between the measurements taken. The intra-observer variability was 6.4%, and inter-observer was 10.6%.

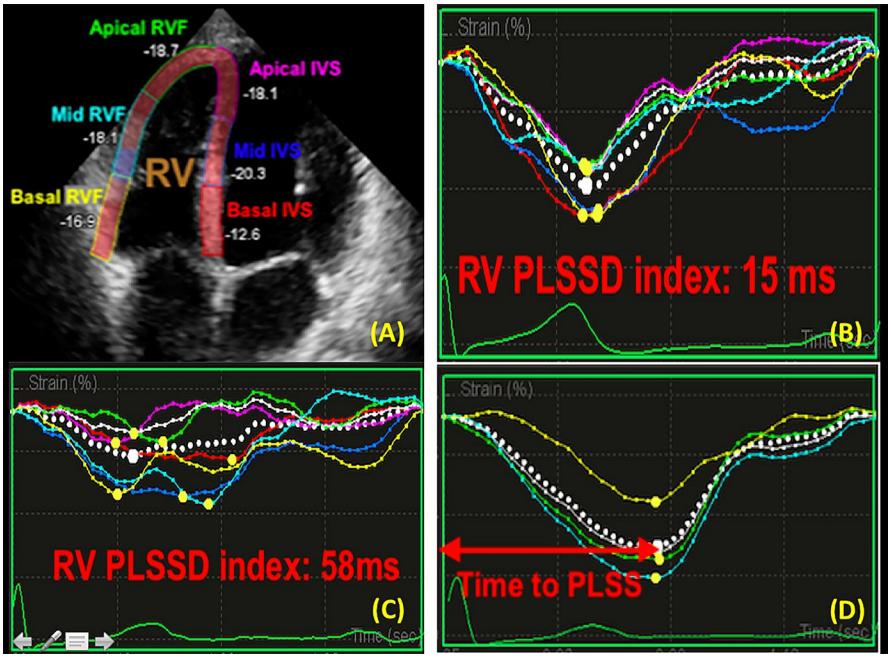

FIGURE 1 A, Speckle-tracking echocardiographic images of right ventricle (RV); B, RV peak systolic strain longitudinal dyssynchrony (RV PLSSD) index of a healthy subject; C, RV PLSSD index for a inferior myocardial infarction patients with RV involvement; E, time to peak longitudinal systolic strain (PLSS) of RV free wall which was measured from the beginning of the QRS complex to the peak systolic strain

TABLE 1 Comparison of baseline characteristics between acute inferior MI patients and healthy controls

	Patients with inferior myocardial infarction (n = 158)	Healthy controls (n = 44)	*P* value
Age (years)	60.8 ± 13.1	61.4 ± 11.8	.34
Men (n, %)	94 (59.4%)	26 (59.0%)	.49
Body mass index (kg/m^2)	27.0 ± 4.4	25.4 ± 4.5	.25
Hypertension (n, %)	54 (34.1%)	14 (31.8%)	.17
Diabetes mellitus (n, %)	48 (30.3%)	13 (29.5%)	.29
Hyperlipidemia (n, %)	32 (20.2%)	10 (22.2%)	.43
Current Smoking (n, %)	73 (46.2%)	21 (47.2%)	.41
Mean arterial blood pressure (mmhg)	88.5 ± 22.1	95.7 ± 20.8	**<.001**

Bold values mean statistically significant.

2.3 | Statistical analysis

All statistical tests were performed with a commercially available software program (SPSS 21.0 for Mac; SPSS, Inc). The variables were investigated using visual (histograms, probability plots) and analytic methods (Kolmogorov–Smirnov/Shapiro–Wilk's test) to determine whether or not they are normally distributed. Categorical variables are presented as numbers and percentages, and continuous data are expressed as mean ± SD. Because of all continuous variables were normally distributed, statistical comparisons of quantitative data were performed by a paired sample *t* test. For the multivariate analysis, the possible factors identified with univariate analyses were further entered into the logistic regression analysis to determine independent predictors of cardiovascular mortality of subjects. Binary logistic regression analysis was used to determine independent predictors of cardiovascular mortality at 3-year follow-up. Hosmer–Lemeshow goodness-of-fit statistics was used to assess model fit. The capacity of time to PLSS difference between RVF-LVL (RV free wall and LV lateral wall) and RV PSSD index values in predicting hospital mortality were analyzed using receiver operating characteristics (ROC) curve analysis. A *P* value of <.05 was considered to show a statistically significant result.

3 | RESULTS

3.1 | Subject characteristics

Overall, 158 patients with acute IMI and 44 healthy subjects were enrolled into the study. The baseline characteristics of IMI patients and healthy subjects are shown in Table 1. The age and sex distributions were similar between groups. Prevalence of cardiovascular risk factors including systemic hypertension, diabetes mellitus, hyperlipidemia, and current smoking were similar between groups. Mean arterial blood pressure was lower in IMI patients at the time of admission.

4 | 2DE and STE-derived measurements

Acute IMI patients were divided into two groups according to the presence of RVMI, with 70 patients (44.3%) in the RVMI group. Comparison of 2DE characteristics and strain analysis between IMI patients and healthy subjects is shown in Table 2. The IMI patients had lower TAPSE and RV S' values and higher RV PLSSD index compared to healthy controls. They had lower PLSS values in RVF and IVS and longer time to PLSS differences between RVF-LVL and RVF-IVS. The IMI patients with RVMI had significantly lower TAPSE and RV S' values and higher RV PLSSD index compared to healthy controls and IMI patients without RVMI. They had significantly lower PLSS values in LVL and IVS compared to healthy controls and significantly lower PLSS values in RVF compared to both healthy controls and IMI patients without RVMI. They also had significantly longer time to PLSS differences between RVF-LVL, RVF-IVS, and LVL-IVS compared to both healthy controls and IMI patients without RVMI.

Twenty-seven patients (17.1%) died due to cardiovascular causes within the following 3 years. The characteristics of these patients are listed in Table 3. The mean age of the mortality group was higher, with more female patients and RV involvement in ECG. The mortality group had significantly higher high sensitivity cardiac troponin T (Trop-hs) level and lower mean blood pressure at admission. They had higher RV PLSSD index and lower RV S', and PLSS in RVF values with significantly longer time to PLSS differences.

Receiver operating characteristics analyses (Figure 2) revealed that RV PLLSD index > 65 predicted mortality with a sensitivity of 88.9%, specificity of 71.8%, positive predictive value 39.3%, and

TABLE 2 Comparison of echocardiographic characteristics between acute inferior MI patients with and without RV involvement in electrocardiogram and healthy subjects

	Patients with IMI (n = 158)	IMI patients with RVMI (n = 70)	IMI patients without RVMI (n = 88)	Healthy controls (n = 44)	*P**	*P***
LV EF (%)	54.6 ± 9.0	53.8 ± 8.1	55.2 ± 9.7	56.2 ± 4.3	.22	.17
RV FAC (%)	38.1 ± 11.4	37.0 ± 11.2	39.0 ± 11.6	40.1 ± 10.2	.34	.25
RV S' (cm/s)	10.8 ± 2.5	9.2 ± 1.8	11.8 ± 2.4	12.7 ± 2.7	**<.001**	**<.001**
TAPSE (mm)	15.4 ± 4.3	12.6 ± 2.9	17.3 ± 3.9	23.2 ± 4.3	**<.001**	**<.001**
RV PSSD index	55.8 ± 21.7	74.0 ± 16.2	31.9 ± 14.9	23.2 ± 15.3	**<.001**	**<.001**[a,b,c]
LV PSSD index	22.8 ± 9.6	21.3 ± 9.1	23.9 ± 10.0	14.2 ± 6.5	.43	.21
Peak longitudinal systolic strain (-%)						
LVL	20.2 ± 5.8	19.1 ± 6.0	21.1 ± 5.7	22.3 ± 4.8	.12	**<.001**[b]
IVS	18.9 ± 5.3	18.3 ± 5.4	19.4 ± 5.2	21.9 ± 4.5	**.04**	**.01**[b]
RVF	15.5 ± 3.8	13.5 ± 3.6	16.9 ± 4.0	25.6 ± 5.9	**<.001**	**<.001**[b]
Time to peak longitudinal systolic strain (ms)						
LVL	315.1 ± 75.7	298.2 ± 82.2	326.9 ± 69.0	254.8 ± 56.2	**<.001**	**<.001**[a,b,c]
IVS	340.8 ± 52.7	354.3 ± 54.5	331.2 ± 49.6	248.5 ± 49.3	**.03**	**<.001**[a,b,c]
RVF	406.9 ± 75.0	450.3 ± 58.1	376.2 ± 70.6	269.4 ± 51.4	**<.001**	**<.001**[a,b,c]
Time to peak longitudinal systolic strain difference (ms)						
RVF-LVLΩ	91.8 ± 101.2	152.1 ± 104.0	49.2 ± 74.5	14.6 ± 24.2	**<.001**	**<.001**[a,b]
RVF-IVS†	66.1 ± 74.2	96.0 ± 76.2	55.0 ± 74.5	20.9 ± 26.3	**<.001**	**<.001**[a,b]
LVL-IVS‡	25.8 ± 66.9	56.0 ± 71.7	4.3 ± 54.2	6.7 ± 18.9	.23	**.04**[a,b]

Abbreviations: EF = ejection fraction; FAC = fractional area change; IMI = inferior myocardial infarction; IVS = inter-ventricular septum; LV = left ventricle; LVL = left ventricular lateral wall; MI = myocardial infraction; PLSSD = peak longitudinal systolic strain dyssynchrony; RV = right ventricle; RVF = right ventricular free wall; RVMI = right ventricular myocardial involvement; S' = systolic velocity; TAPSE = tricuspid annular plane systolic excursion.

[a]Statistical difference was originated from the comparison of patients with and without RVMI.

[b]Statistical difference was originated from the comparison of patients with RVMI and healthy controls.

[c]Statistical difference was originated from the comparison of patients without RVMI and healthy controls.

**P* values derived from statistical comparison between IMI patients and healthy controls.

***P* values derived from statistical comparison among IMI patients with RVMI, IMI patients without RVMI and healthy controls. Post hoc analysis is shown with a, b and c.

ΩIndicates more delay in RV free wall time to peak longitudinal strain compared with LV lateral wall.

†Indicates more delay in RV free wall time to peak longitudinal strain compared with IVS.

‡Indicates more delay in LV lateral wall time to peak longitudinal strain compared with IVS.

Bold values mean statistically significant.

TABLE 3 Comparison of characteristics and echocardiographic parameters of the mortality group

	Mortality (n = 27)	Survived (n = 131)	*P*
Age (years)	69.2 ± 14.1	59.0 ± 12.2	**.003**
Men (n-%)	15 (55.5%)	104 (79.3%)	**.019**
Trop T-hs (μg/L)	6825.4 ± 3317.0	3607.4 ± 2933.3	**<.001**
Mean BP (mmhg)	75.4 ± 23.2	91.1 ± 21.2	**.007**
RVMI	20 (74.0%)	45 (34.3%)	**<.001**
Multivessel coronary disease	9 (33.3%)	45 (34.3%)	.33
TIMI grade 2 or 3 flow	17 (62.9%)	120 (91.6%)	**.03**
RV S' (cm/s)	8.8 ± 1.9	11.1 ± 2.4	**<.001**
RV FAC	37.6 ± 12.0	39.3 ± 11.3	.58
TAPSE (mm)	13.6 ± 5.3	15.8 ± 3.9	.057
RV PLSSD index	79.8 ± 16.2	50.8 ± 19.3	**<.001**
LV PLSSD index	20.2 ± 7.9	23.3 ± 9.8	.34
Peak longitudinal systolic strain (-%)			
LVL	18.4 ± 5.6	20.5 ± 5.7	.25
IVS	17.2 ± 5.0	19.0 ± 5.4	.07
RVF	13.2 ± 4.4	16.0 ± 4.0	**.015**
Time to peak longitudinal systolic strain difference (ms)			
RVF-LVL	185.8 ± 118.0	72.3 ± 86.0	**<.001**
RVF-IVS	120.1 ± 100.5	54.8 ± 62.6	**.019**
LVL-IVS	65.7 ± 56.2	17.4 ± 66.1	**.006**

Abbreviations: BP = blood pressure; FAC = fractional area change; IVS = inter-ventricular septum; LV = left ventricular; LVL = left ventricular lateral wall; PLSSD = peak longitudinal systolic strain dyssynchrony; RV = right ventricular; RVF = right ventricular free wall; RVMI = right ventricular myocardial infarction; RVMI = right ventricular myocardial involvement; S' = systolic velocity; TAPSE = tricuspid annular plane systolic excursion; TIMI = thrombolysis in myocardial infarction; Trop T-hs = high sensitivity cardiac troponin T.

Bold values mean statistically significant.

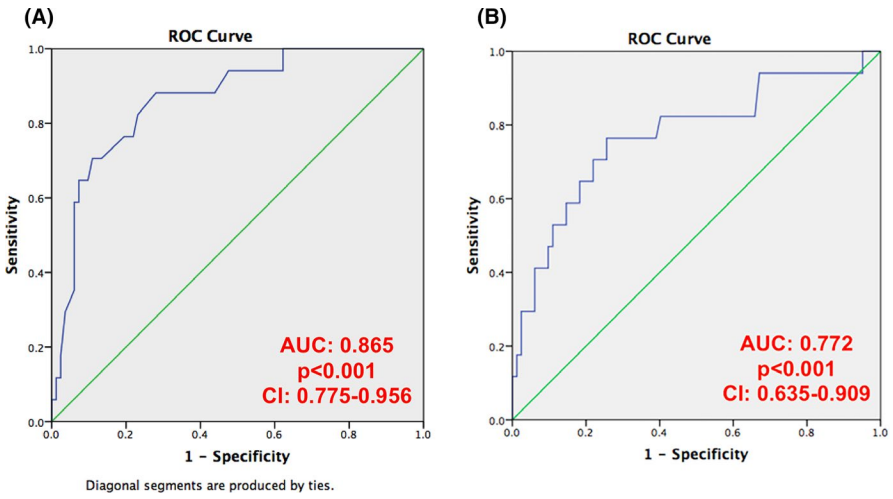

FIGURE 2 A, ROC analysis to predict mortality with in 3-y follow-up. Right ventricle peak longitudinal systolic strain index > 65 ms predicted with 88.2% sensitivity and 72.0% specificity. B, ROC analysis to predict mortality with in 3-y follow-up. Time to peak longitudinal systolic strain difference between RV free wall and LV lateral wall > 110 ms predicted with 76.5% sensitivity and 74.4% specificity

negative predictive value 96.9% (Area under curve: 0.865, $P < .001$) and time to PLSS difference between RVF-LVL > 110 ms predicted mortality with a sensitivity of 77.8%, specificity of 74.1%, positive predictive value 38.2%, and negative predictive value 94.2% (Area under curve: 0.772, $P < .001$).

Binary logistic regression analysis was performed to evaluate the independent predictors of mortality in IMI patients within follow-up. RV PLSSD index > 65 ms, age, and RVMI based on ECG were included in the model. PLSSD index > 65 ms and age were independent predictors of mortality in IMI patients (Table 4).

TABLE 4 Cox multivariate analysis for 3-y follow-up cardiovascular mortality

	P value	Odds ratio	Confidence interval
RV PLSSD index > 65 ms	.009	11.697	1.865–73.368
Age (years)	.041	1.055	1.002–1.110
RVMI	.856	1.178	0.203–6.841

Abbreviations: PLSSD = peak longitudinal systolic strain dyssynchrony; RV = right ventricular; RVMI = right ventricular myocardial involvement.

5 | DISCUSSION

In this study, we demonstrated intra- and inter-ventricular dyssynchrony in patients with IMI compared to healthy controls. In addition, we showed further intra- and inter-ventricular dyssynchrony in IMI patients with RVMI and found intraventricular dyssynchrony shown as RV PLSSD index as an independent predictor for mortality in these patients.

Right ventricle involvement may increase cardiovascular burden in patients with acute IMI. Assali et al showed the increase of major cardiovascular adverse events in acute IMI patients with RV involvement.[13] Furthermore, Hamon et al showed RV dysfunction as a predictor of adverse prognosis in acute myocardial infarction in a meta-analysis of data from 22 relevant studies involving a total of 7,136 patients with acute myocardial infarction at baseline, of whom 1,963 had RVMI determined by ECG (27.5%).[14] Our results are in accordance with these studies. We found that RVMI was more prevalent in mortality group. This may be due to apparent RV intraventricular dyssynchrony in RVMI group. Although there was no study assessing RV intraventricular dyssynchrony in IMI patients, a significant number of clinical studies have investigated LV intraventricular dyssynchrony in patients after acute myocardial infarction. Mollema et al demonstrated significant LV intraventricular dyssynchrony in 18% of patients with acute myocardial infarction treated with PCI.[15] In a subgroup of VALIANT study, the prevalence of LV intraventricular dyssynchrony using velocity vector imaging and its relation with adverse cardiovascular outcomes in patients after high-risk acute myocardial infarction with LV dysfunction was shown.[16] In an acute myocardial infarction study, Antoni et al explored the prevalence of intraventricular dyssynchrony by using radial stain of LV and relation with all cause of mortality and heart failure hospitalization.[4] To best our knowledge, this is the first study demonstrated the intra- and inter-ventricular dyssynchrony using STE and relation with adverse cardiovascular outcomes at long-term follow-up in patients with acute IMI.

The assessment of dyssynchrony can be still challenging. Although cardiac magnetic resonance is a useful imaging method providing morphological and functional information of both ventricles, it is impractical for frequent serial assessment because of high cost and unavailability.[17] STE imaging allows assessment of segmental and global myocardial specific motion. STE overcomes most of the limitations inherent in conventional 2DE and also TDI, given that it is independent of cardiac translation; also, it is angle- and load-independent, thus allowing accurate quantification of myocardial function.[18] As far as recent studies related with inter-ventricular dyssynchrony are concerned, the increase of the time to PLSS noted in our results is in accordance with the reported inter-ventricular dyssynchrony with delayed contraction of the RV free wall in patients with pulmonary hypertension (PHT). Kalogeropoulos et al[19] showed that RV significantly delayed time to PLSS in patients with PHT compared with healthy volunteers using STE.

It is important to emphasize that in this study, RV dyssynchrony was present even with a normal electrocardiographic QRS interval duration. This observation is in agreement with previous data stating that an abnormal electrical conduction is not necessarily needed to produce mechanical intra- or inter-ventricular dyssynchrony, since mechanical dyssynchrony has been identified in the failing myocardium with a normal QRS duration.[20] Therefore, it might appear that not all contributing mechanisms resulting in mechanical dyssynchrony have been identified and are probably complex.

5.1 | Study limitations

We acknowledge the following study limitations. The study was a single-center study, and the sample size was relatively small. We did not test the ROC analysis threshold values in an independent group of patients so the sensitivity and specificity values derived from ROC analysis represent the best-case scenario and future studies with larger sample size are needed to verify our findings. The baseline echocardiographic measurements were performed within 48 hours after primary PCI. The time course of RV function and dyssynchrony before and after the primary PCI was unclear. The preexisting RV systolic dysfunction and dyssynchrony could not be excluded. Because RV systolic function and dyssynchrony can be affected by various conditions, there might be the possibility of the presence of RV systolic dysfunction and dyssynchrony from causes other than acute myocardial infarction. Moreover, validation of RVMI and intra- and inter-ventricular dyssynchrony by CMR and correlation with STE measured PLSS might strengthen our results.

6 | CONCLUSION

Intra- and inter-ventricular dyssynchrony might develop in patients with acute IMI, especially in those with RVMI. Intra- and inter-ventricular dyssynchrony may be an independent predictor for mortality and provide incremental prognostic value over known risk factors for the prediction of long-term outcomes.

CONFLICT OF INTEREST

The authors declare that they have no conflict of interest.

ETHICAL APPROVAL AND CONSENT TO PARTICIPATE

This study approved by the hospital's institutional review board, and informed consent was obtained from each volunteer. We confirm that all authors of this submission have understood approve the journal's licensing policy. Our research involving human participants. Our study was approved by the local Ethics Committee regarding Helsinki Declaration, and written informed consent was obtained from all participants

DATA AVAILABILITY STATEMENT

The data and materials in the current study are available from the corresponding author upon reasonable request.

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