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ORIGINAL PAPER

The association of right ventricular dysfunction with in-hospital and 1-year outcomes in anterior myocardial infarction

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Abstract

In anterior ST-segment elevation myocardial infarction (STEMI), attention paid mainly to the left ventricle. The predictive significance of right ventricular (RV) dysfunction in patients with anterior STEMI has been frequently neglected. In this study, we evaluated the prognostic effect of RV dysfunction on in-hospital and long-term outcomes in patients with first anterior STEMI. A total of 350 patients without known coronary artery disease with first anterior STEMI and treated with primary percutaneous coronary intervention were prospectively enrolled in this study. In-hospital and long-term outcomes were compared between two groups of with or without RV dysfunction. In-hospital mortality was significantly higher in the RV dysfunction group (26.7% vs. 1.6%, $P < 0.001$). The RV dysfunction group also had a higher incidence of cardiogenic shock, recurrent myocardial infarction, target lesion revascularization and stent thrombosis. The 1-year overall survival in patients with and without RV dysfunction was 62.2% and 95.0% respectively. After multivariable analysis, RV dysfunction remained as an independent predictor for in-hospital and long-term mortality. RV dysfunction is an independent predictor of cardiogenic shock, recurrent myocardial infarction, and, in-hospital and long-term mortality in anterior STEMI. Therefore, attention should be paid to the function of right ventricle as in the left ventricle after anterior STEMI.

Keywords Myocardial infarction · Right ventricle · Echocardiography · Mortality

Introduction

Coronary artery disease (CAD) is the most common cause of death in the world, with more than seven million casualties every year. One in every six men and one in every seven women die from acute myocardial infarction (AMI) in Europe [1]. Despite its decline in incidence, ST-segment elevation myocardial infarction (STEMI) is still an important issue in cardiovascular medicine [2]. The prognostic value of right ventricular (RV) function has been disregarded in the past, but recently RV dysfunction was identified as an important prognostic factor for mortality, atrioventricular blocks, arrhythmias, cardiogenic shock and mechanical complications patients with AMI [3–7]. However, it is unclear whether RV dysfunction after anterior AMI also portends poor prognosis or not [7]. Some novel studies have drawn attention to the cardiogenic shock associated with RV dysfunction and have provided new information about the management and outcomes. RV dysfunction has been shown in up to 50% of patients with acute inferoposterior STEMI, and in up to 10% of patients with anterior STEMI. Mechanism of depression in RV functions in inferior wall infarction was found to be similar with the left ventricular (LV) function depression in anterior wall infarction [3, 4, 6–9]. Nonetheless, to date, the prognostic value of RV dysfunction after AMI remains unclear especially in anterior STEMI. There is no study evaluating the outcomes of patients with isolated anterior STEMI with this regard. We aimed to examine the prognostic effect of RV dysfunction in predicting in-hospital and long-term mortality in patients present with first anterior STEMI in a prospective fashion.

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Methods

Patient selection and data sources

This is a single center prospective study. Patients who were diagnosed with first anterior STEMI (n = 356) and underwent primary percutaneous coronary intervention (PPCI) were enrolled to the study in a consecutive way. The patients were evaluated in a high volume tertiary heart center [> 2500 percutaneous coronary intervention (PCI) per year]. The duration of study was 13 months, from May 2015 to May 2016.

All the patients were examined by a trained cardiologist. Their previous health issues and clinical and demographic properties were recorded according to the first examination. The statements of the patients' first-degree relatives were recorded when it was not possible to gain information from the patients. Treatment and laboratory data were obtained from the hospital's medical database system. The electrocardiography, angiography and echocardiography of the patients were analyzed and recorded while patients were in hospital. Mortality information was obtained from the national death notification records and/or by follow-up interviews (directly or by telephone). After the intervention, the patients allowed to coronary care unit for observation. We followed European Society of Cardiology Guidelines during in-hospital and long-term medical management of the patients [10].

Patients with previous CAD diagnosis and with evidence of chronic inflammatory disorders and patients who received antineoplastic and glucocorticoid drugs were excluded from the study. A total of four patients were excluded from the study due to lack of data after hospitalization and two patients were excluded because of not giving the informed consent. A total of 350 patients without known CAD admitted to emergency department with anterior STEMI and treated with PPCI were enrolled to the study. In-hospital and long-term outcomes were compared between the groups with or without RV failure (Fig. 1).

Ethical compliance

Ethical approval was obtained from the Ethics and Research Committee of our hospital. All participants gave written informed consent prior to enrollment in the study. The research was conducted in accordance with the principles of the Declaration of Helsinki.

Definitions

STEMI was defined to have a new ST segment elevation of > 0.1 mV (1 mm) in two contiguous leads or definite or probable a new left brunch bundle block with symptoms suggesting myocardial infarction. The primary end points of this study were the incidence of in-hospital and long-term all-cause of mortality. Acute kidney injury is defined as an increase in serum creatinine level of ≥ 0.3 mg/dL or a relative increase in serum creatinine level of ≥ 50% [11, 12]. Transthoracic echocardiography (TTE) was performed with an already settled Vivid 7 system (GE Vingmed Ultrasound AS, Horten, Norway) in emergency room before the interventions but, did not cause any delay for them. TTE

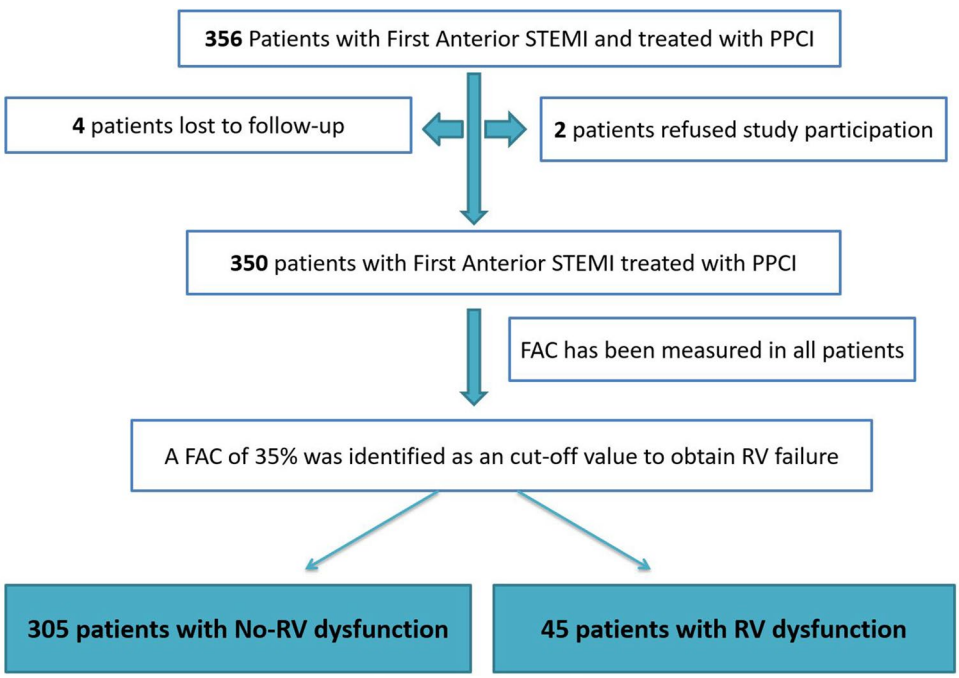

Fig. 1 Study flow chart. *STEMI* ST-segment elevation myocardial infarction, *PPCI* primary percutaneous coronary intervention, *RV* right ventricular, *FAC* fractional area change

was performed while the patients were prepared for the interventions. RV function was assessed quantitatively, as the percent change in the cavity area from end diastole to end systole by echocardiographic analysis. End diastole was identified with the onset of the R wave on the simultaneously recorded electrocardiogram. End systole was identified as the smallest RV cavity size just before tricuspid valve opening. The RV free wall was traced from the base to apex, and the RV areas were calculated from the average of three measurements. RV fractional area change (FAC) was calculated by using the following formula: (end-diastolic area − end-systolic area)/end-diastolic area. Two-dimensional FAC < 35% defined as RV dysfunction according to American Society of Echocardiography Guideline [13].

Reproducibility

The inter-observer variability was assessed by another experienced physician who traced the RV in 50 randomly selected patients; the correlation coefficient between the two FAC measured was 0.85 (coefficient of repeatability = 8.1%, by the Bland and Altman method). The intra-observer reproducibility of FAC measurement was assessed by the primary reader performing two sets of FAC measurements in 50 randomly selected patients, in a blinded fashion. The correlation coefficient (r) between the two assessments was 0.92 (coefficient of repeatability = 6.2%, by the Bland and Altman method).

Table 1 Baseline characteristics of patients with and without right ventricular dysfunction after first anterior myocardial infarction

	No RV dysfunction (n = 305)	RV dysfunction (n = 45)	P value
Age	57.5 ± 13.3	58.0 ± 10.2	0.054
Male gender	247 (81.0)	41 (91.1)	0.097
Body mass index	28.0 ± 4.6	26.7 ± 3.1	0.071
History			
Hypertension	114 (37.4)	14 (31.1)	0.415
Diabetes mellitus	65 (21.3)	9 (20.0)	0.841
Hyperlipidemia	28 (9.2)	2 (4.4)	0.289
Current smoking status	185 (60.7)	21 (46.7)	0.075
Chronic kidney disease	14 (4.6)	2 (4.7)	0.993
Family history of coronary artery disease	147 (48.2)	31 (68.9)	0.010
Chronic lung disease	22 (7.2)	4 (8.9)	0.689
At admission			
Systolic blood pressure, mmHg	134 ± 30	115 ± 21	< 0.001
Pre-infarction angina pectoris	125 (41.0)	23 (51.1)	0.199
Killip class ≥ 3	22 (7.2)	2 (4.4)	0.493
Chest pain period, h	3.6 ± 3.1	3.2 ± 3.3	0.418
Door-to-balloon time, m	29.5 ± 18.3	37.0 ± 22.5	0.310
Creatinine, mg/dL	0.81 ± 0.21	0.79 ± 0.17	0.430
Estimated glomerular filtration rate, mL/m	73.8 ± 23.5	78.3 ± 18.0	0.154
Hematocrit, %	40.5 ± 5.4	39.6 ± 4.3	0.352
GRACE score	102 ± 32	114 ± 26	0.003
TIMI score	3.2 ± 2.1	4.1 ± 2.5	0.006
Medication			
Aspirin	298 (97.7)	45 (100.0)	0.305
PY212 receptor inhibitors	303 (99.3)	45 (100.0)	0.586
Tirofiban	99 (32.5)	19 (42.2)	0.196
Beta blockers	279 (91.5)	43 (95.6)	0.346
Statins	262 (85.9)	40 (88.9)	0.587
Diuretics	52 (17.0)	10 (22.2)	0.211
Angiotensin converting enzyme inhibitor or angiotensin receptor blockers	249 (81.6)	39 (86.7)	0.410

Continuous variables are presented as mean ± SD

Nominal variables presented as frequency (%)

Power analysis

The study must have recruited 15 individuals for each group to have 80% power with 5% type 1 error level to detect a minimum clinically significant difference of FAC when the average expected value in the control group is 46.7 with a standard deviation of 5.9. The power of the study increased to 98% with selection of 305 controls and 45 RV-dysfunction patients with 5% type 1 error level.

Statistical analysis

Categorical variables were expressed as number and percentages and Pearson's Chi square or Fisher's exact tests were used to evaluate the differences. Continuous variables with skewed and normal distribution were expressed as mean ± SD. Continuous variables with normal distributions compared using independent sample *T* test. Continuous variables with skewed distributions were compared using the Mann–Whitney *U* test. The median survival times of two groups were compared using the Kaplan–Meier survival method. Differences between the groups were analyzed by the log-rank test. Hierarchical logistic regression and Cox proportional regression model were used for multivariable analysis. The odds ratio (OR) indicate the relative risk of in-hospital mortality of RV dysfunction group and the hazard ratio (HR) indicate the relative risk of 1-year mortality of RV dysfunction group. In multivariable models, confounders in multivariate analysis as predictors of in-hospital and long-term mortality (some significant history, electrocardiographic, echocardiographic and angiographic confounders) were considered. Confounders were obtained according to the current literature and previous studies and experts' opinions [2, 4–7, 17]. A two-tailed p value of <0.05 was

Table 2 Electrocardiographic and echocardiographic parameters of patients

	No RV dysfunction (n = 305)	RV dysfunction (n = 45)	P value
Electrocardiographic parameters			
Normal sinus rhythm	291 (95.4)	43 (95.6)	0.965
Resting heart rate > 100 beats/min	94 (30.8)	13 (28.9)	0.793
Significant Q waves	12 (3.9)	5 (11.1)	0.037
Left bundle branch block	2 (2.0)	5 (11.1)	0.001
Right bundle branch block	8 (2.6)	3 (6.7)	0.147
Prolonged QRS > 120 ms	20 (6.6)	6 (13.3)	0.106
Prolonged corrected QT > 440 ms	66 (21.6)	12 (26.7)	0.449
Echocardiographic parameters			
Left ventricular ejection fraction, %	44 ± 7	39 ± 6.3	< 0.001
Tricuspid annular plane systolic excursion	2.37 ± 0.46	1.78 ± 0.38	< 0.001
Right ventricle S′ velocity, cm/s	14.1 ± 3.0	11.0 ± 2.8	< 0.001
Right ventricle diastolic area, cm^2	16.5 ± 3.3	17.3 ± 3.3	0.110
Right ventricle systolic area, cm^2	8.8 ± 2.1	11.8 ± 2.5	< 0.001
Fractional area change, %	46.7 ± 5.9	31.8 ± 3.0	< 0.001
Pulmonary artery systolic pressure, mmHg	20.8 ± 10.8	24.6 ± 13.9	0.033
Left ventricle diastolic diameter, cm	4.7 ± 0.5	5.0 ± 0.5	0.002
Left ventricle systolic diameter, cm	3.5 ± 0.5	3.5 ± 0.8	0.444
Interventricular septal thickness, mm	9.9 ± 1.7	10.0 ± 2.0	0.604
Posterior wall thickness, mm	9.8 ± 1.6	10.1 ± 2.0	0.384
Mitral valve E velocity, cm/s	68.0 ± 20.4	72.2 ± 20.3	0.198
Mitral valve e′ velocity, cm/s	9.7 ± 3.4	10.2 ± 3.8	0.126
Mitral valve E/e′ ratio	7.2 ± 2.6	7.1 ± 2.8	0.882
Mitral regurgitation vena contracta, mm	1.4 ± 0.23	1.6 ± 0.28	0.103
Tricuspid regurgitation vena contracta, mm	1.7 ± 0.13	2.0 ± 0.12	0.063
Right ventricle basal diameter, mm	3.2 ± 0.5	3.2 ± 0.4	0.903
Right ventricle mid-diameter, mm	2.4 ± 0.4	2.4 ± 0.4	0.478
Right ventricle length, mm	7.1 ± 0.9	6.8 ± 0.8	0.061
Right ventricular Tei index	0.36 ± 0.16	0.53 ± 0.17	< 0.001

Continuous variables are presented as mean ± SD

Nominal variables presented as frequency (%)

considered as statistically significant, and 95% CIs were presented for all odds ratios and hazard ratios. Analyses were performed using Statistical Package for Social Sciences software, version 20.0 (SPSS; IBM, Armonk, New York, USA).

Results

Patients' baseline characteristics were listed in Table 1. Patients with RV dysfunction had lower systolic blood pressure and higher GRACE and TIMI risk scores compared to the other group of patients, who had no RV dysfunction. Table 2 shows electrocardiographic and echocardiographic parameters of patients. The RV dysfunction group had significantly lower LV ejection fraction (LVEF), tricuspid annular plane systolic excursion (TAPSE), RV S′ velocity and RV Tei index compared to the other group. Spearman correlation analysis revealed a significant correlation between the FAC and the TAPSE (r = 0.881; P < 0.001) and between the FAC and the RV S′ velocity (r = 0.813; P < 0.001). Whereas the RV dysfunction group had slightly higher pulmonary artery systolic pressure, there were not any differences regarding Doppler filling pattern of LV and mitral and tricuspid valvular insufficiency. The RV dysfunction group had a significantly higher number of patients with left bundle branch block (LBBB), RV Tei-index, and LV diastolic diameter and lower LVEF compared to the other group. Table 3 shows angiographic analysis of patients. RV dysfunction group also had significantly higher osteal and lower mid-distal stenosis of left anterior descending artery (LAD). Additionally, the RV dysfunction group had higher SYNTAX score.

Table 3 Angiographic analysis of patients

	No RV dysfunction (n = 305)	RV dysfunction (n = 45)	P value
Culprit segment of left anterior descending artery			
Osteal portion	8 (2.6)	11 (24.4)	< 0.001
Proximal portion	179 (58.7)	25 (55.6)	0.691
Mid-distal portion	114 (37.4)	2 (4.4)	< 0.001
Vessel stenosis > 70%			
Left main coronary artery	6 (2.0)	3 (6.7)	0.063
Right coronary artery	66 (21.6)	9 (20.0)	0.802
Left circumflex artery	64 (21.0)	6 (13.3)	0.231
SYNTAX score	18.8 ± 5.2	22.4 ± 7.3	0.004
Stent type			
Drug eluting stent	211 (69.2)	33 (73.3)	0.571
Bare metal stent	94 (30.8)	12 (26.7)	0.571
Manual thrombectomy	13 (4.3)	4 (8.9)	0.178
TIMI flow grade before intervention			
TIMI 0–I	259 (84.9)	37 (82.2)	0.640
TIMI II–III	46 (15.1)	8 (17.8)	0.640
TIMI flow grade after intervention			
TIMI 0–II	0 (0.0)	0 (0.0)	1.000
TIMI II–III	305 (100.0)	45 (100.0)	1.000

Nominal variables presented as frequency (%)

In-hospital outcomes

Table 4 shows in-hospital and 1-year clinical outcomes of the study population. In-hospital mortality was significantly higher in the RV dysfunction group (26.7% vs. 1.6%, P < 0.001). These patients also had a higher incidence of cardiogenic shock, recurrent myocardial infarction, target lesion revascularization and stent thrombosis. Age, smoking, hypertension, diabetes mellitus, hyperlipidemia, heart rate > 100 bmp, prolonged QRS, prolonged corrected QT, osteal stenosis of LAD, LVEF, LBBB, right bundle branch block (RBBB), LV diastolic diameter and RV dysfunction were found to be univariate predictors of in-hospital mortality in hierarchical logistic regression analysis. Age, heart rate > 100 bpm, osteal stenosis of LAD, LVEF, LBBB, RBBB and RV dysfunction [OR 6.32, 95% confidence interval (CI) 2.16–18.40] were found to be independently associated with in-hospital mortality in multivariate hierarchical logistic regression analysis (Table 5).

Long-term outcomes

The 1-year overall survival for patients with and without RV dysfunction was 62.2% and 95.0% respectively (Fig. 2). Age, smoking, hypertension, diabetes mellitus, hyperlipidemia, renal failure, heart rate > 100 bmp, osteal stenosis of LAD, LVEF, LBBB, LV diastolic diameter and RV failure were found to be the univariate predictors of 1-year mortality in Cox regression analysis. Age, hypertension, renal failure, osteal stenosis of LAD, LVEF, LBBB and RV failure [HR 4.21, 95%

Table 4 In-hospital and long-term outcomes of patients

	No RV dysfunction (n = 305)	RV dysfunction (n = 45)	P value
In-hospital course			
Cardiogenic shock	5 (1.6)	12 (26.7)	< 0.001
Acute kidney injury	13 (4.3)	3 (6.7)	0.471
Recurrent myocardial infarction	4 (1.3)	5 (11.1)	< 0.001
Target lesion revascularization	8 (2.6)	5 (11.1)	< 0.001
Stent thrombosis	3 (1.0)	3 (6.7)	0.006
Mortality	27 (8.9)	17 (37.8)	< 0.001
Out-hospital course			
All-cause mortality	10 (3.3)	5 (17.9)	0.001

Nominal variables presented as frequency (%)

Table 5 Univariate analysis and multivariate model for in-hospital mortality

Univariate analysis	P value	Multivariate analysis	P value	OR (95% CI)
Age	< 0.001	Age	< 0.001	1.14 (1.07–1.30)
Sex	0.611			
Body mass index	0.500			
Hypertension	0.012			
Diabetes mellitus	0.027			
Hyperlipidemia	0.083			
Smoking	0.001			
Renal failure	0.108			
Heart rate > 100 bpm	0.003	Heart rate > 100 bpm	0.032	3.18 (1.87–21.14)
Non-sinus rhythm	0.136			
Prolonged QRS > 120 msn	0.013			
Prolonged corrected QT > 440 msn	0.034			
Family history of CAD	0.208			
Osteal stenosis of LAD	< 0.001	Osteal stenosis of LAD	0.027	2.51 (1.54–6.13)
Proximal stenosis of LAD	0.131			
> 70% stenosis of RCA	0.164			
> 70% stenosis of LCx	0.597			
Left ventricular ejection fraction	< 0.001	Left ventricular ejection fraction	< 0.001	0.85 (0.80–0.91)
Right ventricular failure	< 0.001	Right ventricular failure	< 0.001	6.32 (2.16–18.40)
Left bundle branch block	< 0.001	Left bundle branch block	0.044	8.71 (1.07–48.14)
Right bundle branch block	0.150			
Left ventricular diastolic diameter	0.098			

All clinically relevant parameters were included in the model

OR Odds ratio, *CI* confidence interval, *CAD* coronary artery disease, *LAD* left anterior descending artery, *LCx* left circumflex artery, *RCA* right coronary artery

CI 2.08–11.85] were found to be independently associated with 1-year mortality in multivariate Cox logistic regression analysis (Table 6).

Discussion

Although the clinical significance of RV dysfunction has been thoroughly discussed; in STEMI, attention has primarily been given to the left ventricle [14]. Previous studies evaluating RV dysfunction after STEMI mainly examined patients with acute RCA occlusion, although the blood flow of the anterior wall of the RV is provided by LAD. Thus, acute LAD occlusion may cause RV infarction in different stages [7, 15–17]. Several other mechanisms have been postulated to explain the poor outcomes in patients with AMI and RV dysfunction which include (i) ventricular interdependence, (ii) arrhythmogenic environment of RV and (iii) LV underfilling because of decrease in RV stroke volume; however, they have not been confirmed by prospective studies with large patient population [2, 4–7, 18]. Thus, the current study was designed to evaluate the prognostic effect of RV dysfunction on clinical outcomes in patients with first anterior STEMI. We observed that RV dysfunction remained an independent risk factor for in-hospital and long-term mortality in a relatively large patient population.

The location, shape and unique contractile functions of right ventricle make the RV systolic assessment challenging [2]. Because of these methodological limitations, FAC has been commonly considered as an efficacious way to determine RV dysfunction [2, 5, 13]. Several previous studies examined the association of RV dysfunction with AMI. Azevedo et al. showed the effectiveness of RV FAC measuring in assessment of RV dysfunction after anterior STEMI. Moreover, they revealed that FAC can predict RV failure 6-months after AMI, although they did not evaluate the clinical outcomes of these patients [2]. The VALIANT Echo study is another prospective multi-center study demonstrating that RV FAC is a useful way of predicting mortality in AMI [18]. In a comprehensive analysis, Zornoff et al. revealed that RV dysfunction is an independent predictor of death in AMI; however, they included all types of AMI and did not exclude the patients who presented with previous AMI which could significantly affect RV functions and outcomes [6]. Unlike this study, we enrolled a larger patient population with first anterior STEMI to

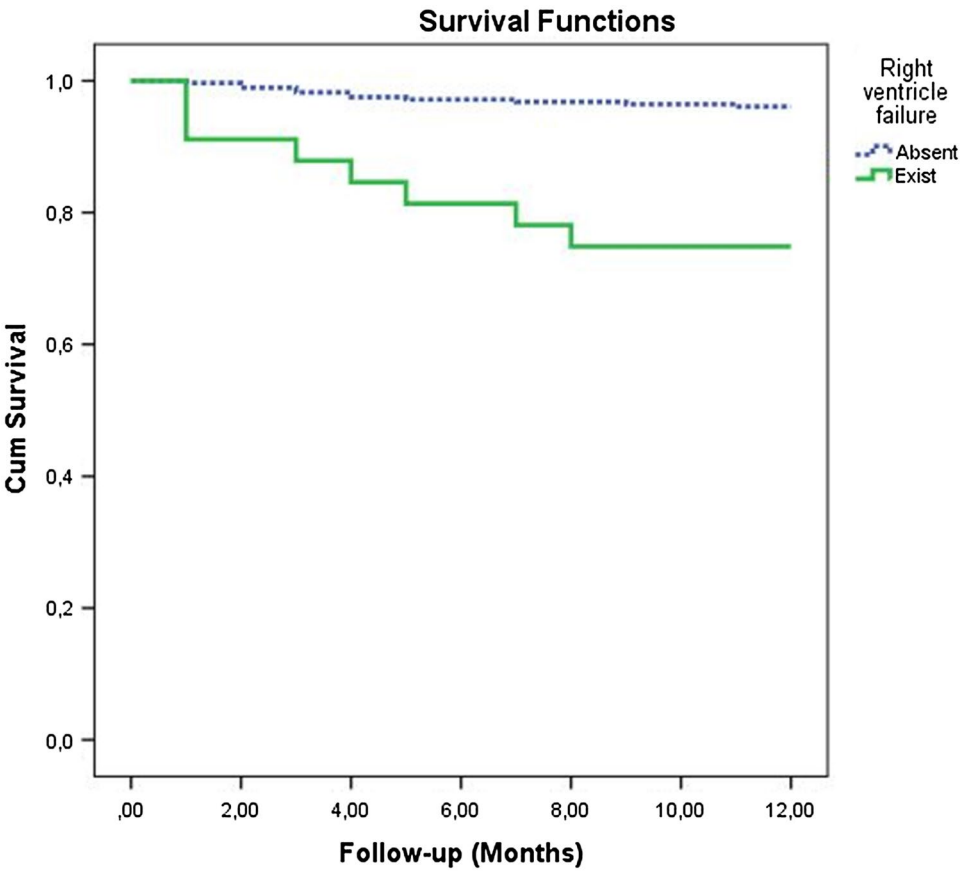

Fig. 2 Kaplan–Meier survival curve for overall survival in patients with first anterior ST segment elevation myocardial infarction (n = 350) stratified by existence of right ventricular dysfunction

better reveal the prognostic value of RV dysfunction in isolated LAD occlusion.

In a cardiac magnetic resonance imaging (MRI) study, Bodi et al. reported that approximately 1/3 of the RV mass was at risk in anterior STEMI; however, 94% of the area at risk was restored, and the final infarct size was only 2% of the RV. The infarct size remained small owing to successful revascularization of LAD. Bonanad et al. reported that in anterior STEMI, microvascular obstruction frequently occurs in the RV [19]. In addition, Abbate et al. revealed significant myocardial apoptosis of RV in patients with LV AMI [20]. Another MRI study, which is conducted by Jensen et al. showed that there was remarkable myocardial edema in the RV of patients with anterior STEMI [21, 22]. These studies demonstrate that RV dysfunction after anterior STEMI does not originate solely from RV infarction but may also arise from LV failure and ventricular interdependence [8, 23]. The results of the current study confirmed this hypothesis. In our study, the RV dysfunction group had significantly lower LVEF, higher LBBB, and higher LAD osteal stenosis. The severity and proximity of LAD stenosis might have determined LV and concurrent RV failure. However, in our study, the RV dysfunction group had slightly higher pulmonary artery systolic pressure. There was no difference with respect to the Doppler filling pattern of LV and mitral and tricuspid valvular insufficiency between the groups. Although higher systolic pressure in the right ventricle was not associated with ventricular filling patterns or valvular regurgitations, it might have contributed to RV dysfunction.

In anterior STEMI, new onset LBBB indicates proximal or osteal LAD occlusion. These situations may explain the higher prolonged QRS > 120 msn in duration, higher incidence of LAD osteal stenosis and lower LVEF in RV dysfunction group. Although the co-existence of LV and RV failure might have caused higher mortality, RV dysfunction remained an independent risk factor for both in-hospital and long-term mortality after multivariable analysis.

RV dysfunction has gained much attention after echocardiographic improvements in right-chambers' quantification. Some previous studies evaluated the prognostic effect of RV dysfunction via different modalities on different patient populations [2, 4–7, 17]. In anterior STEMI, the focus has commonly been on LV functions and PPCI. Although the above-mentioned studies have drawn attention to the RV functions, there has been no comprehensive evaluation of the impact of RV dysfunction in anterior STEMI. Although accumulated evidence suggests that FAC could be a reliable marker of RV dysfunction and could provide an important prognostic value, we observed that TAPSE and RV S′ velocity had a significant correlation with the FAC in these patients. Hence,

Table 6 Univariate analysis and multivariate model for 1-year mortality

Univariate analysis	P value	Multivariate analysis	P value	OR (95% CI)
Age	< 0.001	Age	< 0.001	1.11 (1.04–1.28)
Sex	0.478			
Body mass index	0.216			
Hypertension	0.004	Hypertension	0.014	2.21 (1.42–5.13)
Diabetes mellitus	0.018			
Hyperlipidemia	0.069			
Smoking	0.062			
Renal failure	0.007	Renal failure	0.018	2.34 (1.47–6.89)
Heart rate > 100 bpm	0.018			
Non-sinus rhythm	0.379			
Prolonged QRS > 120 msn	0.107			
Prolonged corrected QT > 440 msn	0.536			
Family history of CAD	0.315			
Osteal stenosis of LAD	< 0.001	Osteal stenosis of LAD	0.031	2.44 (1.47–7.62)
Proximal stenosis of LAD	0.234			
> 70% stenosis of RCA	0.129			
> 70% stenosis of LCx	0.438			
Left ventricular ejection fraction	< 0.001	Left ventricular ejection fraction	< 0.001	0.81 (0.76–0.94)
Right ventricular failure	< 0.001	Right ventricular failure	< 0.001	4.21 (2.08–11.85)
Left bundle branch block	< 0.001	Left bundle branch block	0.029	3.16 (1.42–18.14)
Right bundle branch block	0.247			
Left ventricular diastolic diameter	0.103			

All clinically relevant parameters were included in the model

OR Odds ratio, *CI* confidence interval, *CAD* coronary artery disease, *LAD* left anterior descending artery, *LCx* left circumflex artery, *RCA* right coronary artery

TAPSE and RV S′ velocity may provide rapid and accurate measurements of RV systolic functions in these patients. Therefore, we propose that RV functions should be evaluated in patients with anterior STEMI as in inferior STEMI. Echocardiography is a reliable modality to examine the RV functions (especially FAC) and may provide an additional prognostic value in patients presented with anterior STEMI.

Limitations

Several limitations of this study should be noted before interpreting results of this study. First and the most important limitation of the current study was the complexity of RV geometry when assessing the RV. The method used to quantify RV function was not a true volumetric based method. Three-dimensional echocardiographic data are especially valuable for obtaining RV quantification. Second, ST-elevation in V4R lead was not provided for RVMI diagnosis. Third, the conal branch of RCA was not recorded and evaluated. Fourth, the current study was conducted in one-center in a one-geographical area. Besides, a big part of the enrolled patients were men. Thus, this study has not sufficient value by the respect of generalization. Hence, some big-volume and multi-center studies are needed to confirm our findings. Finally, although RV dysfunction at the time of patient enrollment was an important predictor of outcomes in these patients; we cannot exactly show the predictive role of RV function at different periods after MI.

Conclusion

Evaluation of RV dysfunction after anterior STEMI is not so common as in LV dysfunction and this is the first study evaluating the effect of RV dysfunction on in-hospital and long-term outcomes in patients with first anterior STEMI. In this study, we observed that both in-hospital and 1-year outcomes were affected in patients with anterior STEMI and RV dysfunction. Thus, special care should be given for the function of right ventricle in anterior STEMI.

Compliance with ethical standards

Conflict of interest All authors declare that they have no conflict of interest.

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