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Original Article

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Changes in the Right Ventricular Diameters and Systolic Function After Successful Percutaneous Coronary Intervention in Patients With First Acute Myocardial Infarction

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Abstract

Background: Right ventricular (RV) diameters and systolic function are strong predictors of outcomes and major adverse cardiovascular events (MACEs) in acute myocardial infarction (AMI). This study evaluated RV parameters via echocardiography in AMI patients and assessed their changes 1 month after discharge.

Methods: A prospective observational study was conducted on 133 consecutive patients with their first AMI. RV diameters and systolic function were evaluated with echocardiography within 24 h after successful percutaneous coronary intervention (PCI) and again 1 month after discharge. MACEs were evaluated during hospitalization and at 1 month post discharge.

Results: Men accounted for 69.92% of the participants, with a mean age of 68 years. Reduced right ventricular free wall longitudinal strain (RVFWSL) and right ventricular four-chamber longitudinal strain (RV4CSL) were observed in 62.4% (mean -18.28±8.77%) and 83.34% (mean -14.78±6.94%) of patients, respectively. Right ventricular longitudinal strain (RVLS) was significantly lower in the ST-elevation myocardial infarction (STEMI) group and Killip III-IV patients. RV basal and mid diameters (RVD1, RVD2) were larger in right coronary artery (RCA) and left main artery (LM) lesions than in left anterior descending artery (LAD) and left circumflex artery (LCx) ones ($P < 0.05$). RVLS correlated significantly with body mass index (BMI), troponin I, and left ventricular ejection fraction (LVEF). After 1 month, RVFWSL and RV4CSL improved significantly, especially in patients without MACEs, Killip III-IV, and single-vessel lesions.

Conclusions: RV diameters varied with the culprit lesion and remained stable after 1 month. RVLS was significantly reduced in AMI, especially in STEMI and Killip III-IV, correlating with LVEF. After 1 month, RVLS improved faster, particularly in patients without MACEs, Killip III-IV, or single-vessel lesions.

Keywords: Right ventricular function; Right ventricular global longitudinal strain; Myocardial infarction; Percutaneous coronary intervention

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Introduction

Acute myocardial infarction (AMI) is the leading cause of death in Asia and the world. According to the World Health Organization, 18.6 million people died of coronary artery disease in 2019. In Asia, the number of cardiovascular disease deaths was 10.8 million annually, accounting for 35% of global causes of death [1]. The burden of disease after myocardial infarction is also significant, with increased risk of heart failure, arrhythmias, reduced quality of life, and decreased life expectancy for patients [2].

AMI not only causes changes in left ventricular function but also alters right ventricular (RV) function due to damage of the culprit coronary artery, which abruptly reduces blood to the right ventricle, the activity of the interventricular septum, and the close interaction between two ventricular chambers through neurohormonal mechanisms [3-5]. The echocardiographic parameters of the right ventricle are valuable in predicting and assessing the severity of cardiovascular diseases, including AMI [6, 7]. However, RV parameters were often neglected in clinical practice due to the complex structure and limitations of measurement techniques. Many echocardiographic indices of RV systolic function confused it, and very few studies could demonstrate the value of each individual index [8]. Morphological changes and reduced RV function, particularly right ventricular four-chamber longitudinal strain (RV4CSL) and right ventricular free wall longitudinal strain (RVFWSL), are common in patients with a first AMI and are associated with major adverse cardiovascular events (MACEs)

during hospitalization and 1 year post-infarction. Early detection of these abnormalities can enhance treatment outcomes for patients [3, 4, 9, 10].

The understanding of RV diameters and RV systolic function changes by right ventricular longitudinal strain (RVLS), including RV4CLS and RVFWLS, and their contributing factors in patients with their first AMI has not been thoroughly researched, especially in Vietnam. Recently, the RVLS that has a high correlation with RV ejection fraction by cardiovascular magnetic resonance (CMR) was recommended by the American Society of Echocardiography (ASE) as one of the valuable indices to evaluate RV systolic function and be able to detect RV dysfunction early [6]. Therefore, the main aim of this study was to evaluate the indices of RV diameters and RV systolic function in patients with first AMI within 24 h after successful percutaneous coronary intervention (PCI), changes 1 month after discharge, and contributing factors to RV dysfunction.

Materials and Methods

Study design and participants

We conducted an observational, single-center study on 133 consecutive first AMI patients admitted and treated in the Cardiovascular Center, Military Hospital 103, Hanoi, Vietnam, from March 2023 to August 2024. The study procedure did not change the effectiveness of treatment for all patients.

Patients were diagnosed with AMI based on the fourth universal definition in 2018 [11] (evidence of elevated cardiac troponin values and at least one of the following criteria: symptoms of myocardial ischemia; new ischemic electrocardiogram (ECG) changes, development of pathological Q waves, evidence of new regional wall motion abnormality or identification of a coronary thrombus by angiography). The 2017 European Society of Cardiology (ESC) defines ST elevation as a rise at the J point in at least two consecutive leads. Specifically, in leads V2 and V3, the criteria are J-point elevation $\geq$ 2.5 mm for males under 40 years old, $\geq$ 2 mm for males aged 40 years or older, and $\geq$ 1.5 mm for females. A J-point elevation of $\geq$ 1 mm is considered significant in all other leads [12].

We excluded patients with chronic pulmonary diseases, acute or chronic pulmonary embolism, primary pulmonary hypertension, congenital heart diseases, moderate to severe valvular heart diseases, arrhythmias (atrial fibrillation or atrioventricular block degree II or III during echocardiographic examination), acute mechanical complications (acute mitral regurgitation, ventricular septum defect, etc.), severe systemic condition (infection, trauma, Child-Pugh B cirrhosis, stage IIIB or higher kidney failure), and patients who did not agree to participate in the study.

Institutional Review Board statement

All human research procedures were conducted in accordance with the ethical standards of the committee responsible for human experimentation (both institutional and national) and the Helsinki Declaration, as revised in October 2013. This study was approved by the Ethical Committee of Military Hospital 103 (No. 2847) on April 26, 2023.

Data collection and measurements

The following demographic, clinical, and laboratory variables were collected for each patient: age, sex, body mass index (BMI), the presence of comorbidities (hypertension, diabetes mellitus), ECG, echocardiography, blood tests, and coronary angiography.

Invasive revascularization strategy in research

Patients diagnosed with AMI received medications and emergency PCI in the culprit artery lesion according to the protocol in our Cardiovascular Center based on recommendations of ESC 2018 on myocardial revascularization [13]. Emergency reperfusion therapy is indicated for all ST-elevation myocardial infarction (STEMI) patients with time from symptom onset < 12 h or patients with time from onset > 12 h in the presence of ongoing symptoms, hemodynamic instability, life-threatening arrhythmias, and routine PCI strategy in patients presenting late without any symptoms. In non-ST-segment elevation myocardial infarction (NSTEMI) patients, urgent PCI is recommended in patients within 2 h with very high risk (hemodynamic instability, cardiogenic shock, ongoing chest pain refractory to medical treatment, life-threatening arrhythmias, cardiac arrest, mechanical complications, acute heart failure or dynamic ST-T wave changes); early PCI (within 24 h) is recommended in patients with high risk (Grace score > 140 points, dynamic ST/T changes, ongoing chest pain); and PCI within 72 h after first presentation in patients with intermediate risk (Grace score $\geq$ 109 and $\leq$ 140; recurrent symptoms, left ventricular ejection fraction (LVEF) $\leq$ 40%, diabetes mellitus or renal insufficiency). PCI was considered successful if the culprit artery had a flow grade 2 or 3 according to the Thrombolysis in Myocardial Infarction (TIMI) flow grading system and residual stenosis < 20% was achieved. We performed staged complete revascularization for patients with indications after 1 month.

Echocardiographic study

Trained cardiologists performed echocardiography examinations within 24 h of successful PCI and 30 days after discharge. Echocardiographic images were obtained by Philips EPIQ 7C (Philips Medical System, Andover, Massachusetts) with a multi-frequency transducer X5-1 (1-5 MHz). The echocardiography protocol was used following the ASE guidelines [14]. Parasternal long-axis and short-axis views, along with apical four-chamber, three-chamber, and two-chamber views, were utilized to assess valvular structural and functional evaluation of the left ventricle (LV). LVEF was measured using the biplane Simpson's method. All studies were performed with simultaneous electrocardiographic monitoring.

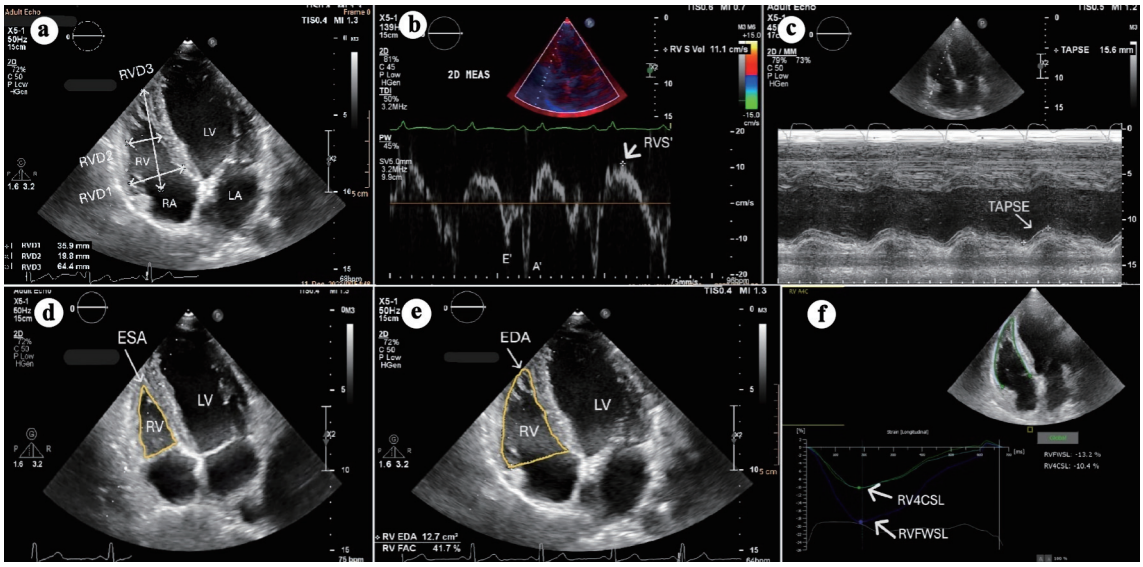

Figure 1. Measurement of the RV conventional parameters and the RV longitudinal strain in the RV-focused apical four-chamber view. (a) RV size. (b) RVS' wave was measured using tissue Doppler imaging. (c) TAPSE measure in M-mode. (d) The end-systolic RV area and (e) end-diastolic RV area were measured for RV-FAC. (f) Automatic RVFWLS and RV4CSL measured by the QLAB AutoStrain Software. RVD1: right ventricular basal diameter; RVD2: right ventricular mid-cavity diameter; RVD3: right ventricular longitudinal linear dimension; RV-FAC: right ventricular fractional area change; TAPSE: tricuspid annular plane systolic excursion; RVS': Doppler tissue imaging-derived tricuspid lateral annular systolic velocity; RVFWSL: right ventricular free wall longitudinal strain; RV4CSL: right ventricular four-chamber longitudinal strain; RV: RV: right ventricle; LV: left ventricle; EDA: end-diastolic area; ESA: end-systolic area.

1) RV size

We measured the RV basal (RVD1), mid-cavity (RVD2), and longitudinal linear dimensions (RVD3) in an RV-focused apical four-chamber view (Fig. 1a). RV wall thickness was measured at end-diastole, below the subcostal view's tricuspid annulus.

2) RV systolic function

We measured conventional RV parameters: tricuspid annular plane systolic excursion (TAPSE), Doppler tissue imaging-derived tricuspid lateral annular systolic velocity (RVS'), and right ventricular fractional area change (RV-FAC) according to ASE guidelines 2015 [14]. The RVS' wave was measured in the apical four-chamber view using Doppler tissue imaging mode (Fig. 1b). TAPSE was measured using M-mode with a cursor placed at the junction of the lateral tricuspid leaflet (Fig. 1c). The apical four-chamber view in two-dimensional (2D) mode was used to measure RV systolic and diastolic areas. RV-FAC was calculated by subtracting the end-systolic area (ESA, Fig. 1d) from the end-diastolic area (EDA, Fig. 1e) and dividing this value by the end-diastolic area.

3) RV 2D-strain analysis

RVFWLS and RV4CSL were obtained using 2D AutoStrain software (AutoStrain, QLAB version 13, Philips Medical Systems, Andover, MA, USA) in an RV-focused four-chamber view at 50 to 70 frames/s. After endocardial border delineation, the software automatically segments the right ventricle into six segments (basal, middle, and apical segments of both the RV free wall and the interventricular septum). It tracks the movement of speckles in the myocardium throughout the cardiac cycle on 2D echocardiographic images [15]. Finally, the software automatically generates RVLS curves of free wall and septum (Fig. 1f). The longitudinal strain was defined as the percentage of myocardial shortening relative to the original length and presented as a negative value, with a larger negative strain value reflecting better shortening. The abnormal ranges, according to ASE (2015), are as follows: TAPSE < 17 mm, FAC < 35%; RVS' < 9.5 cm/s; RVLS > -20% [14, 16].

Long-term follow-up analysis

Patients received medical treatment before and after PCI during hospitalization and after discharge according to ESC guidelines on the management of AMI in patients with NSTE-MI [17] and STEMI [12], but the treatment was individualized for each patient's condition. All 133 patients were monitored for MACEs through phone calls (68 patients) or follow-up visits after 1 month (75 patients). MACEs were documented, including cardiovascular death, readmission for heart failure, or recurrent myocardial infarction. A second echocardiogram was performed on 75 patients who returned for a 1-month follow-up. The study protocol was shown in Figure 2.

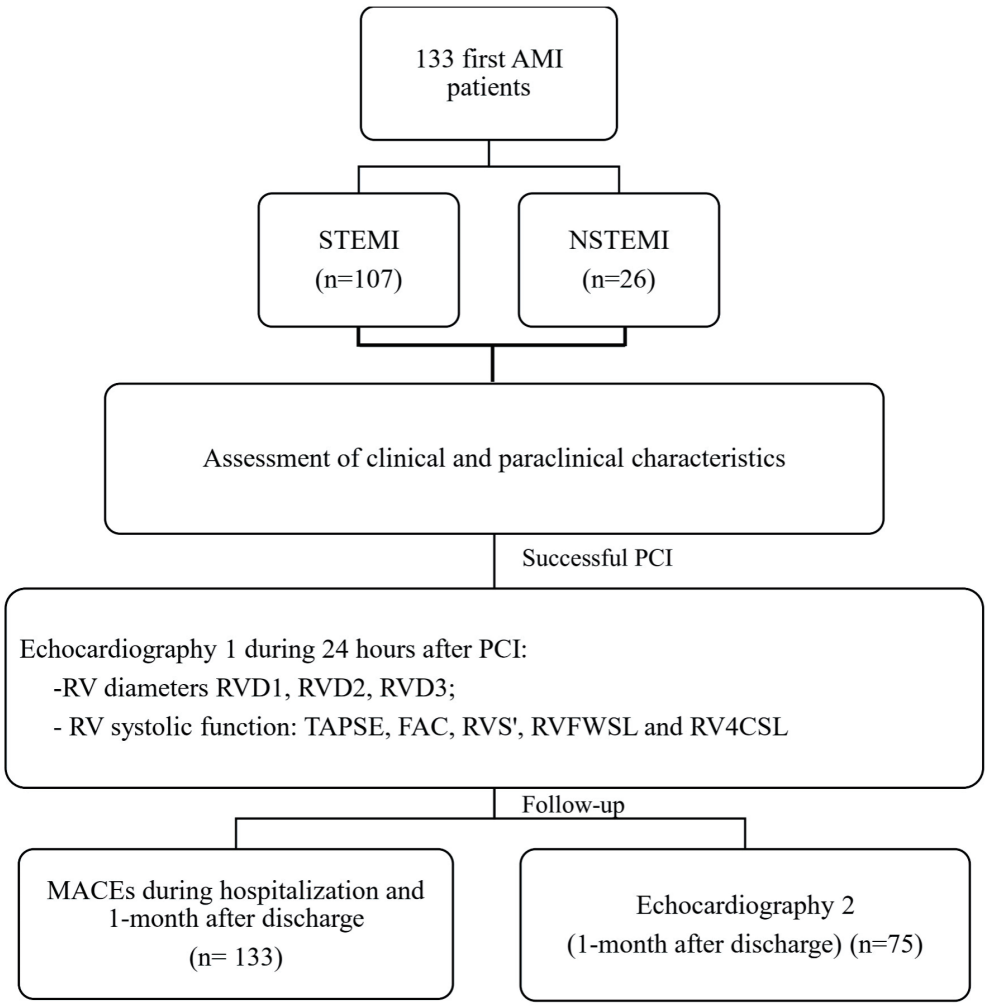

Figure 2. The study protocol. AMI: acute myocardial infarction; FAC: fractional area change; NSTEMI: non-ST-segment elevation myocardial infarction; PCI: percutaneous coronary intervention; RV: right ventricular; RV4CSL: right ventricular four-chamber longitudinal strain; RVD1: right ventricular basal diameter; RVD2: right ventricular mid diameter; RVD3: right ventricular longitudinal linear dimension; RVFWSL: right ventricular free wall longitudinal strain; RVS': Doppler tissue imaging-derived tricuspid lateral annular systolic velocity; STEMI: ST-segment elevation myocardial infarction; TAPSE: tricuspid annular plane systolic excursion.

Statistical analysis

The research data were processed using medical statistical algorithms with SPSS version 22.0 software. Normally distributed data were presented as mean ± standard deviation (SD); non-normally distributed values were presented as quartiles median (IQR). The mean value of normally distributed variables was compared with the threshold value of the reference range using the one-sample *t*-test. The median value of non-normally distributed variables was compared with the threshold value using the Wilcoxon-signed rank test. Two independent continuous variables with a normal distribution were compared using the independent samples *t*-test; two independent continuous variables without a normal distribution were compared using the Mann-Whitney U-test; three or more parameters with a normal distribution were compared using one-way analysis of variance (ANOVA); and for non-parametric multivariate testing without a normal distribution, the Kruskal-Wallis test was applied. Multivariate linear regression analysis was conducted to assess the impact of variables on the continuous variable. Paired variables with a normal distribution were evaluated using the paired sample *t*-test, while paired variables with a non-normal distribution were compared by the McNemar test.

Results

Baseline clinical, angiographic, and conventional echocardiographic characteristics

The study included 133 patients with a first AMI who successfully underwent PCI, with an average age of 68.03 ± 11.81 years, the majority of whom were male (69.92%). Table 1 indicates

Table 1. Baseline Demographic and Clinical Characteristics of the Overall Population

	Study group (n = 133)
Clinical characteristics	
Mean age (years), mean ± SD (Min - Max)	68.03 ± 11.81 (36 - 93)
Male (n, %)	93 (69.92%)
BMI (kg/m^2)	22.26 ± 2.77
Hypertension (n, %)	81 (60.9%)
Diabetes mellitus (n, %)	38 (28.57%)
Chronic renal disease (n, %)	7 (5.3%)
Time of admission (h), median (IQR)	5 (2.75 - 14)
Door to balloon time (h), median (IQR)	4 (2 - 7.5)
Heart rate (bpm), median (IQR)	80 (70 - 89)
Hospital duration (days)	8.89 ± 4.29
Systolic blood pressure (mm Hg), mean ± SD	131.61 ± 21.38
Troponin I (pg/mL), median (IQR)	1,253.40 (189.05 - 4,152.20)
ProBNP (pg/mL), median (IQR)	625.10 (97.87 - 5,659.70)
STEMI (n, %)	107 (80.50)
NSTEMI (n, %)	26 (19.50)
Killips classification (n, %)	
Killips I	75 (56.39)
Killips II	46 (34.58)
Killips III, IV	12 (9.03)
Left ventricular echocardiographic parameters	
LVDd (mm) (mean ± SD)	45.32 ± 6.20
LVDs (mm), median (IQR)	30.00 (26.92 - 34.15)
LVEF biplane (%) (mean ± SD)	47.31 ± 9.41[a]
LVGLS (%) (mean ± SD)	-10.26 ± 3.37[a]
LAVI (mL/m^2), median (IQR)	21.12 (15.47 - 28.47)
Regional wall movement abnormality (n, %)	120 (90.22)
Characteristics of coronary arterial lesions	
Culprit coronary artery (n, %)	
LAD	65 (48.87)
LCx	18 (13.53)
RCA	47 (35.33)
LM	3 (2.27)
Severity of culprit coronary arterial stenosis (n, %)	
100%	71 (53.38)
99%	21 (15.78)
Less than 99%	41 (30.84)
Numbers of coronary arterial lesions (n, %)	
1	56 (42.10)
2	36 (27.06)
≥ 3	41 (30.84)
Drugs (n, %)	
Aspirin	133 (100)

Table 1. Baseline Demographic and Clinical Characteristics of the Overall Population - *(continued)*

	Study group (n = 133)
Clopidogrel	87 (65.41)
Ticagrelor	46 (34.58)
Statin	133 (100)
ACEs	63 (47.36)
Beta blockers	28 (21.05)
Anticoagulation (unfractionated heparin, low-molecular-weight heparin)	133 (100)
MACEs (n, %)	7 (5.26)
Death	2 (1.50) (1 at hospital duration and 1 during follow-up)
Hospitalization due to heart failure	4 (3.00)
Recurrent AMI	1 (0.75)

[a]Significantly less than the normal range according to ASE 2015 (P < 0.05). SD: standard deviation; BMI: body mass index; IQR: interquartile range; ProBNP: pro-B-type natriuretic peptide; STEMI: ST-elevation myocardial infarction; NSTEMI: non-ST-segment elevation myocardial infarction; LVDd: left ventricular diameter in diastole; LVDs: left ventricular diameter in systole; LVEF biplane: left ventricular ejection fraction biplane; LVGLS: left ventricular global longitudinal strain; LAVI: left atrial volume index; LAD: left anterior descending artery; LCx: left circumflex artery; LM: left main artery; RCA: right coronary artery; MACEs: major adverse cardiac events; AMI: acute myocardial infarction; ASE: American Society of Echocardiography; ACEs: angiotensin-converting enzymes inhibitors.

107 patients (80.50%) had STEMI, while 26 patients (19.5%) had NSTEMI. After a 1-month follow-up, seven patients experienced MACEs, including two deaths, four hospitalizations for heart failure, and one readmission due to recurrent myocardial infarction. In the study group, the proportions of patients with the culprit coronary artery being the left anterior descending artery (LAD), left circumflex artery (LCx), and right coronary artery (RCA) were 48.8%, 13.53%, and 35.33%, respectively, with three patients (2.2%) having left main artery (LM) lesions. Among basic echocardiographic parameters, left ventricular diameter in diastole (LVDd), left ventricular diameter in systole (LVDs), and left atrial volume index (LAVI) remained unchanged; however, LVEF biplane and left ventricular global longitudinal strain (LVGLS) showed a significant decrease compared to normal threshold values (P < 0.05) (Table 2).

Baseline RV diameters and systolic function

In the study group, RV diameters remained unchanged, but the thickness of the RV wall was significantly greater than the normal threshold (P = 0.01), with 52.63% showing RV hypertrophy on ultrasound. The parameters TAPSE, FAC, and RVS' showed no changes, but RVFWSL and RV4CSL exhibited a significant decrease compared to the normal range. The percentage of patients with reduced RVFWSL and RV4CSL was 62.4% (mean value -18.28 ± 8.77) and 83.34% (-14.78 ± 6.94), respectively (Table 2). The longitudinal strain of the right ventricle was significantly reduced in the STEMI group compared to the NSTEMI group (P = 0.04). RVD1 and RVD2 in the RCA and LM culprit groups were significantly greater than in the LAD and LCx culprit lesion groups. All

Table 2. Baseline Echocardiographic Characteristics of Right Ventricular Diameters and Systolic Function in the Study Group

Echo data	Study group (n = 133)	Normal range (mean)	P[a]	Numbers of abnormal (n, %)
RVD1 (mm)	27.96 ± 5.58	25 - 41 (33)	1	3 (2.25)
RVD2 (mm)	21.58 ± 4.70	19 - 35 (27)	1	16 (12.03)
RVD3 (mm)	57.76 ± 11,59	59 - 83 (71)	1	0 (0)
RVWT (mm)	5.6 (5.3 - 6.7)[b]	1 - 5 (3)[b]	0.01[b]	70 (52.63%)[b]
TAPSE (mm)	20 (18.4 - 23)	≥ 17	1	15 (11.27)
FAC (%)	45.29 ± 10.19	≥ 35	0.68	20 (15.03)
RVS' (cm/s)	12.4 (10.6 - 13.8)	≥ 9.5	0.91	7 (5.26)
RVFWSL (%)	-18.28 ± 8.77[b]	≤ -20[b]	< 0.001[b]	83 (62.40)[b]
RV4CSL (%)	-14.78 ± 6.94[b]	≤ -20[b]	< 0.001[b]	111 (83.45)[b]

[a]P one-tail when comparing the mean/median value of study variables with the threshold value of reference range according to ASE guidelines 2015. [b]Significant differences from the reference interval threshold value. RVD1: right ventricular basal diameter; RVD2: right ventricular mid-cavity diameter; RVD3: right ventricular longitudinal linear dimension; RVWT: right ventricular wall thickness; TAPSE: tricuspid annular plane systolic excursion; FAC: fractional area change; RVS': Doppler tissue imaging-derived tricuspid lateral annular systolic velocity; RVFWSL: right ventricular free wall longitudinal strain; RV4CSL: right ventricular four-chamber longitudinal strain; ASE: American Society of Echocardiography.

RV systolic function indices decreased in the Killip III and IV groups compared to the Killip I and II groups ($P < 0.05$). However, the size and function parameters of the right ventricle did not change between the subgroups with and without MACEs (Table 3). The linear multivariate regression in Table 4 showed that the variables BMI, troponin I at admission, and LVEF biplane had a significant impact on the values of RVFWSL and RV4CSL, with model values of $R^2 = 0.39$ and 0.32, respectively ($P < 0.05$).

RV diameters and systolic function 1 month after discharge

The results in Tables 5 and 6 indicate that RVD3, RVFWSL, and RV4CSL all showed improvement compared to parameters at baseline after the 1-month follow-up. Notably, the longitudinal strain of the right ventricle improved significantly in the subgroup without MACEs, Killip III, IV, and AMI with one-lesion coronary artery.

Discussion

The echocardiographic examination within 24 h after successful PCI showed no change in RV diameters but increased RV wall thickness in patients with AMI compared to the normal range according to ASE 2015 ($P < 0.05$). In the medical literature of 1985, Gottdiener et al noted the phenomenon of increased RV wall thickness in chronic pressure overload of the left ventricular diseases like hypertension or aortic stenosis [18]. To date, no studies have mentioned this phenomenon, but changes in RV wall thickness and RV remodeling have been shown to be valuable in some other cardiovascular diseases. Sano et al proved that RV wall thickness could predict RV reverse remodeling after treatment in pulmonary hypertension patients and was associated with long-term outcomes [19]. Regarding RV systolic function indices, our results showed that TAPSE, FAC, and RVS' did not change compared to the normal range according to ASE 2015 recommendations; however, RVLS significantly decreased (RVFWSL and RV4CSL > -20%; $P < 0.05$). RVLS allows early detection of RV dysfunction and is proved to be significantly correlated with RV ejection fraction via CMR ($r = -0.797$, $P < 0.001$) compared to other RV systolic function parameters [14, 20]. RVLS has been recently increasingly noticed in clinical practice because it can limit the disadvantages of other commonly used RV systolic function indices such as TAPSE, FAC, and RVS', which are often dependent on cursor location, acoustic window-dependent, and cannot reflect the whole RV function [21]. Since 2018, there has been a consensus between the European Association of Cardiovascular Imaging (EASCI) and the ASE to standardize definitions and techniques for using 2D speckle tracking echocardiography to assess RV deformation. However, there are still limitations in applying RVLS in clinical practice, such as intervendor variability in strain estimates as a result of different algorithms used by different commercial machines and software, as well as a lack of data to provide accurate recommendations on threshold for abnormal values of RVLS [16]. According to the 2015 ASE recommendation, the results from multinational WASE study, and other clinical studies, we applied an abnormal RVLS value of > -20% [16, 21, 22]. The percentage of patients with abnormal RVFWSL and RV4CSL was 62.4% and 83.4%, respectively, while the percentage of patients with RV involvement (RCA culprit lesion) was 35.3%. Thus, not only does RV myocardial infarction cause changes in right heart function (due to the direct damage mechanism of the culprit coronary artery), but changes in RV function were also observed in other myocardial infarction lesions (due to the mechanism of the close interaction between the two ventricular chambers, neurohumoral activity, natriuretic peptides, and the effects of the sympathetic and parasympathetic nervous systems). This may be explained by the anatomical structure of the continuous transverse muscle layer between the left and right heart chambers and the common interventricular septum, which forms a mechanism for a close interactive function between the two ventricular chambers [5]. Recently, many studies have shown that patients with myocardial infarction who have impaired RVLS might experience worse clinical outcomes, poorer prognosis, reduced left ventricular performance, higher risk of arrhythmias and MACEs over time [7, 10, 23]. With the high percentage of patients with reduced RVLS in AMI, there is a need to pay more attention to these parameters to anticipate and improve the quality of diagnosis and treatment of this disease.

In our subgroup study, STEMI patients had a significantly lower RVLS compared to NSTEMI patients ($P < 0.05$). Grenne et al showed the same results. Patients with STEMI due to acute coronary occlusions develop larger infarcts and more impaired left ventricular function than patients with NSTEMI without occlusions through CMR examination [24]. This characteristic may lead to differences in the prognosis of STEMI and NSTEMI patients in the short and long terms. A study on 13,441 AMI patients (8,250 with STEMI, and 5,191 with NSTEMI) by Polonski et al found that after adjustment for the baseline characteristics and treatment strategy, the long-term prognosis was worse in STEMI patients [25]. Although CMR is the gold standard for assessing the morphology and function of heart chambers, it is not always available in healthcare facilities in Vietnam and may not be suitable for evaluating patients in acute phase of AMI. Bedside echocardiography for assessing the RV function in cases provides significant value in clinical practice [5, 26]. The free wall of the right ventricle is primarily supplied by the RCA. In contrast, portions of the interventricular septum are supplied by the LAD or the posterior interventricular artery, which branches from the LCx in cases of RCA dominance. Due to this RV perfusion anatomy, damage to a culprit coronary artery can impact RV function. However, no significant changes were observed in RV systolic function parameters among the culprit artery subgroups. Notably, RVD1 and RVD2 measurements in the RCA and LM culprit groups were larger than those in the LAD and LCx groups ($P < 0.05$). This finding has clinical significance, as detecting abnormal RV wall motion can be challenging, but increased RV diameters are a valuable indicator of RV myocardial infarction. Additionally, we observed a significant decline in all RV systolic function parameters in patients with Killip class III and IV, compared to those in Killip class I and II. Patients with reduced RV function are associated with severe clinical condi-

Table 3. Echocardiographic Evaluation of Right Ventricular Diameters and Systolic Functions in Clinical Subgroups of Study Group at Baseline

Subgroups (n = 133)	RVD1 (mm)	RVD2 (mm)	TAPSE (mm)	FAC (%)	RVFWSL (%)	RV4CSL (%)
Types of myocardial infarction (n = 133)						
STEMI (n = 107)	28.2 ± 5.4	21.6 ± 4.5	20.2 (10.6 - 13.9)	45.2 ± 10.1	-17.5 ± 8.4[a]	-14.3 ± 6.7[a]
NSTEMI (n = 26)	27.2 ± 4.8	20.6 ± 4.5	20.0 (19.1 - 24.8)	46.2 ± 10.5	-22.2 ± 9.7[a]	-17.1 ± 8.9[a]
P	0.78	0.23	0.19	0.66	0.04[a]	0.04[a]
Culprit coronary artery lesion (n = 133)						
LAD (n = 65)	27.4 ± 5.1[a]	20.1 ± 4.1[a]	20.2 (18.0 - 23.6)	46.7 ± 9.6[a]	-17.5 ± 9.7	-12.9 ± 7.3
LCx (n = 18)	26.6 ± 5.2[a]	22.4 ± 5.6[a]	19.1 (18.0 - 21.1)	42.4 ± 8.0[a]	-22.3 ± 7.0	-17.3 ± 5.9
RCA (n = 47)	29.8 ± 5.4[a]	22.7 ± 4.2[a]	20.0 (18.4 - 21.0)	42.1 ± 11.8[a]	-18.3 ± 7.1	-15.6 ± 6.0
LM (n = 3)	31.5 ± 6.2[a]	24.2 ± 4.8[a]	22.4 (18.5 - 28.1)	49.9 ± 11.1[a]	-15.4 ± 2.8	-11.7 ± 3.0
P	0.02[a]	0.02[a]	0.42	0.03[a]	0.56	0.63
Killip classification (n = 133)						
Killip I (n = 75)	28.9 ± 6.0	21.0 ± 4.2	20.4 (18.1 - 22.9)[a]	45.4 ± 9.9[a]	-18.4 (-24.4; -13.0)[a]	-14.5 (-17.7; -10.4)[a]
Killip II (n = 46)	27.06 ± 6.2	21.1 ± 5.2	20.0 (18.5 - 23.8)[a]	47.1 ± 9.7[a]	-18.3 (-23.2; -11.8)[a]	-14.4 (-19.0; -8.9)[a]
Killip III, IV (n = 12)	29.9 ± 5.8	23.1 ± 5.4	17.3 (11.5 - 19.9)[a]	36.7 ± 12.0[a]	-5.0 (-8.5; -2.5)[a]	-8.8 (-14.0; -4.1)[a]
P	0.66	0.06	0.03[a]	< 0.001[a]	0.03[a]	0.04[a]
MACE (n = 133)						
MACE (+) (n = 7)	31.2 ± 3.8	24.1 ± 4.8	21.0 (18.4 - 22.8)	46.1 ± 15.3	-17.3 ± 10.9	-13.1 ± 9.9
MACE (-) (n = 126)	28.1 ± 8.1	21.1 ± 4.7	20.0 (17.7 - 23.0)	45.1 ± 10.0	-17.7 ± 8.1	-14.2 ± 6.5
P	0.11	0.06	0.75	0.81	0.91	0.81

[a]Significant differences. STEMI: ST-elevation myocardial infarction; NSTEMI: non-ST-segment elevation myocardial infarction; LAD: left anterior descending artery; LCx: left circumflex artery; LM: left main artery; RCA: right coronary artery; MACE: major adverse cardiac event; RVD1: right ventricular basal diameter; RVD2: right ventricular mid-cavity diameter; TAPSE: tricuspid annular plane systolic excursion; FAC: fractional area change; RVFWSL: right ventricular free wall longitudinal strain; RV4CSL: right ventricular four-chamber longitudinal strain.

Table 4. Multivariate Linear Regression Analysis of Factors Affecting Right Ventricular Longitudinal Strain at Baseline (N = 133)

Parameters	RVFWSL (R^2 = 0.39) (P < 0.001)		RV4CSL (R^2 = 0.32) (P = 0.01)	
	B	P value	B	P value
Age (years)	0.18	0.05	0.10	0.29
Male	-0.14	0.12	-0.13	0.18
STEMI	0.13	0.14	0.07	0.42
BMI (kg/m^2)	0.27[d]	< 0.001[d]	0.24[d]	0.01[d]
Troponin I (pg/mL)	-0.28[d]	< 0.001[d]	-0.22[d]	0.01[d]
NT-pro-BNP (pg/mL)	0.02	0.79	0.00	0.94
EF biplane (%)	-0.23[d]	0.02[d]	-0.31[d]	< 0.001[d]
LVGLS (%)	0.24[d]	0.01[d]	0.18	0.08
MACE (+)	-0.19[d]	0.04[d]	-0.14	0.16
Culprit coronary artery lesion[a]	0.08	0.37	0.03	0.70
Number of coronary artery lesions	-0.02	0.81	-0.03	0.71
Killips classification[b]	0.15	0.06	0.07	0.42
The severity of the culprit coronary artery lesion[c]	0.05	0.62	0.04	0.64

[a]1: LAD; 2: LCx; 3: RCA; 4: others. [b]1: Killip I; 2: Killip II; 3: Killip III, IV. [c]1: 100%; 2: 99%; 3: less than 99%. [d]Significant differences. BMI: body mass index; NT-pro-BNP: N-terminal pro-B-type natriuretic peptide; STEMI: ST-elevation myocardial infarction; EF: ejection fraction; LVGLS: left ventricular global longitudinal strain; MACE: major adverse cardiac event; RVFWSL: right ventricular free wall longitudinal strain; RV4CSL: right ventricular four-chamber longitudinal strain; LAD: left anterior descending artery; LCx: left circumflex artery; RCA: right coronary artery.

tion during hospitalization and cardiovascular events overtime. Many studies indicated that the cutoff threshold of RV4CSL > -15.5% was significant in predicting composite events over 5 years, the rate of ventricular arrhythmias, or mortality in patients with AMI [23, 27, 28]. Awad et al concluded that FAC < 37.5%, TAPSE < 15.8 mm and RVS' < 9.6 cm/s are independent predictors of MACEs within the first 30 days after STEMI and NSTEMI [29]. Anastasiou et al showed that RV-GLS was independently associated with in-hospital mortality and outperformed conventional RV indices in predicting in-hospital mortality [8]. However, our study did not observe any changes in RV systolic function parameters between the groups with or without MACEs, possibly due to the short follow-up period and the small number of patients with MACEs.

The multivariate linear regression analysis showed that the models were statistically significant, explaining 36% and 32% of the variations in the RVFWSL and RV4CSL indices, respectively. The BMI, troponin I level at admission, and LVEF biplane significantly impacted the RVLS. The results suggest that we should evaluate RV systolic function (especially RVFWSL and RV4CSL) in AMI patients with high levels of troponin I at admission (presenting late from symptom onset), high BMI, and reduced LVEF biplane. Kumar et al also showed the same correlation between RVLS and LVEF (r = 0.642, P < 0.001) [10]. This

Table 5. Comparison of Right Ventricular Diameters and Systolic Function Parameters at Baseline and 1-Month After Discharge

Echo data (n = 75)	Baseline	1-month	P
LVEF biplane (%)	47.3 ± 8.5	48.0 ± 10.0	0.55
LVGLS (%)	-10.1 (-11.5; -8.2)	-10.0 (-12.6; -7.9)	0.57
RVD1 (mm)	27.0 (25.0 - 31.3)	29.6 (25.3 - 30.7)	0.97
RVD2 (mm)	20.6 (17.8 - 24.4)	19.8 (16.5 - 22.0)	0.05
RVD3 (mm)	61.9 ± 9.4[a]	63.8 ± 8.9[a]	< 0.001[a]
TAPSE (mm)	20.7 (18.4 - 22.9)	20.0(18.8 - 23.0)	0.78
FAC (%)	44.5 ± 10.3	45.4 ± 10.4	0.80
RVS' (cm/s)	12.4 (10.7 - 13.6)	12.0 (10.6 - 13.0)	0.10
RVFWSL (%)	-15.7 ± 7.2[a]	-17.8 ± 6.9[a]	0.04[a]
RV4CSL (%)	-12.6 ± 5.6[a]	-14.3 ± 5.2[a]	< 0.001[a]

[a]Significant differences. LVEF biplane: left ventricular ejection fraction biplane; LVGLS: left ventricular global longitudinal strain; RVD1: right ventricular basal diameter; RVD2: right ventricular mid-cavity diameter; RVD3: right ventricular longitudinal linear dimension; RVWT: right ventricular wall thickness; TAPSE: tricuspid annular plane systolic excursion; FAC: fractional area change; RVS': Doppler tissue imaging-derived tricuspid lateral annular systolic velocity; RVFWSL: right ventricular free wall longitudinal strain; RV4CSL: right ventricular four-chamber longitudinal strain.

Table 6. Changes of Right Ventricular Longitudinal Strain at Baseline and 1-Month After Discharge in Clinical Subgroups

Subgroup (total n = 75)	Parameter	Baseline	1-month	P value
MACE				
MACE (+) (n = 6)	RVFWSL (%)	-17.1 ± 0.2	-10.3 ± 6.7	0.38
	RV4CSL (%)	-13.5 ± 0.7	-7.1 ± 6.5	0.36
MACE (-) (n = 69)	RVFWSL (%)	-15.7 ± 7.3[a]	-18.0 ± 6.9[a]	0.01[a]
	RV4CSL (%)	-12.6 ± 5.6[a]	-16.5 ± 5.5[a]	0.02[a]
Killips classification				
Killips I (n = 42)	RVFWSL (%)	-16.4 ± 5.3	-18.0 ± 6.2	0.19
	RV4CSL (%)	-13.2 ± 5.0	-14.6 ± 5.4	0.14
Killips II (n = 27)	RVFWSL (%)	-16.4 ± 7.9	-18.0 ± 8.1	0.42
	RV4CSL (%)	-12.9 ± 6.1	-14.3 ± 5.9	0.36
Killips III, IV (n = 6)	RVFWSL (%)	-7.5 ± 5.2[a]	-15.4 ± 6.5[a]	0.03[a]
	RV4CSL (%)	-7.0 (-11.2; -3.1)[a]	-11.4 (-15.1; -9.3)[a]	0.02[a]
Number of coronary artery lesions				
1 vessel disease (n = 31)	RVFWSL (%)	-14.6 ± 6.9[a]	-18.1 ± 5.3[a]	< 0.001[a]
	RV4CSL (%)	-11.9 ± 5.5[a]	-15.0 ± 4.2[a]	< 0.001[a]
2 vessel disease (n = 16)	RVFWSL (%)	-17.0 ± 7.3	-17.2 ± 7.7	0.94
	RV4CSL (%)	-13.7 ± 5.5	-12.9 ± 5.8	0.91
3 vessel disease (n = 28)	RVFWSL (%)	-16.2 ± 7.4	-17.8 ± 8.2	0.39
	RV4CSL (%)	-12.7 ± 5.8	-13.8 ± 6.8	0.37

[a]Significant differences. MACE: major adverse cardiac event; RVFWSL: right ventricular free wall longitudinal strain; RV4CSL: right ventricular four-chamber longitudinal strain.

result showed a close correlation between the two ventricles in AMI patients. While previously echocardiographic left ventricular indices such as EFBP and LVGLS are valuable in predicting mortality and cardiovascular events in patients with AMI, more studies today are increasingly demonstrating the importance of RV systolic function, particularly RVLS, in predicting outcomes of patients. Lejeune et al showed that even in patients with heart failure with preserved ejection fraction (HFpEF), impaired RVLS provided significant additional prognostic value to identify patients at high risk for adverse events [30].

RVD1 and RVD2 are clinically significant measurements indicating abnormal RV dilation. However, these parameters showed no changes 1 month after discharge. In contrast, RVLS improved significantly after 1 month compared to baseline values ($P < 0.05$), while TAPSE, FAC, RVS', LVEF, and LVGLS remained unchanged.

It is significant that the risk of adverse outcomes is highest within the first 30 days after an infarction or PCI [31, 32], especially stent re-occlusion, which typically occurs within the initial 30 days due to the incomplete endothelization of the stent platform, along with a high risk of arrhythmias, not tolerating to severe heart failure, leading to higher rates of readmissions, recurrent myocardial infarction and mortality in patients with AMI [31]. Roifman et al examined changes in RV function in 31 patients with acute STEMI after successful PCI. RV functions at 3 weeks and 6 weeks differed from the time immediately after the intervention; however, there was no difference between 3 and 6 weeks after PCI. The earliest time to assess changes in RV function after myocardial infarction is 3 weeks [26]. Patients with persistent RV injury had an increased risk of mortality 3 - 4 weeks after PCI [32]. We observed a significant improvement in RVLS in subgroups of patients without MACEs and with one-lesion coronary artery disease ($P < 0.05$). Patients in Killip class III and IV demonstrated a significant improvement in RVLS 1 month after PCI compared to baseline values. This finding highlights that even in AMI patients with severe clinical presentations, such as cardiogenic shock or acute pulmonary edema, PCI can contribute to improved cardiac function and better patient outcomes. However, the strategy of PCI for AMI patients, whether PCI with only one lesion in emergency and staged multivessel or immediate multivessel complete revascularization, remained a topic of debate. According to 2018 ESC guidelines on myocardial revascularization, PCI strategy with only one lesion and staged multivessel revascularization should be indicated to reduce all-cause mortality and severe renal failure instead of immediate complete revascularization, especially in patients with cardiogenic shock [33, 34]. In other cases, it remained controversial in terms of time and stage to perform complete revascularization, which depends largely on clinical status, comorbidities, and angiographic coronary characteristics [33-35]. In our research, to minimize the differences in invasive coronary artery intervention strategies, we included patients with AMI after successfully PCI in the culprit lesion, with short hospital stays (average 8.8 days), and being followed up after 1 month before complete revascularization, provided they had indications. In

such groups, we noticed early improvement in RVLS in subgroups without MACEs, Killip III and IV and especially with one-vessel lesion. In the near future, as recommendations for AMI PCI strategy evolve, with increased numbers of patients having complete revascularization earlier, subgroups with two or more lesions may have improvement in RVLS.

Our study has some clinical implications. RV dysfunction, particularly RV strain, is often associated with severe clinical conditions (types of AMI, left ventricular functions, Killlip classification), events during hospitalization, and cardiovascular events over time.

In patients with AMI, RVLS correlated significantly with LVEF. Those with compromised RVLS faced higher MACEs incidence during short- and long-term follow-up compared to those with preserved RV functions. Early detection of RV dysfunction through strain imaging can guide therapy and improve prognosis [8-10].

Despite our best efforts, this study had some limitations. First, it was a single-center study with a small sample size, which may not adequately represent the entire AMI population. Second, the short follow-up period led to a low incidence of MACEs, limiting our ability to evaluate the prognostic value of RV diameters and RV function parameters in AMI patients. Third, we could not incorporate advanced RV function measurements, such as three-dimensional right ventricular ejection fraction (RVEF) assessed by echocardiography or CMR, due to technical resource constraints at our center. Nonetheless, a larger multicenter study with a more extended follow-up period and more clinical events would clarify our findings and further explore the role of RV diameters and systolic function parameters in clinical outcomes and risk stratification for AMI patients.

Conclusions

At baseline, 52.63% of the 133 first-AMI patients had increased RV wall thickness, and 12.03% had increased RV mid-cavity diameter. RV diameters were largest in the RCA and LM groups, with less change in the LCx and LAD groups. Meanwhile, 62.40% and 83.45% of patients had reduced RVFWSL and RV4CSL, respectively, compared to the normal range (ASE 2015). RVLS was significantly lower in the STEMI group compared to NSTEMI group. RV systolic function was lower in the Killip III-IV subgroup compared to Killip I-II. After 1 month, RVLS improved earlier than LVEF, especially in the subgroup without MACEs, Killip III-IV, and single-vessel disease.

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Conflict of Interest

The authors declare no conflict of interest.

Informed Consent

Informed consent was obtained from all subjects involved in the study

Author Contributions

Conceptualization: TND, TLC. Methodology: TND, TLC. Formal analysis: TAPP. Investigation: TND, TLC, TAPP. Data curation: TND, TAPP. Writing - original draft preparation: TND, TAPP. Writing - review and editing: TND. Visualization: TAPP. Supervision: HNL, TLC.

Data Availability

The data presented in this study are available on request from the corresponding author.

Abbreviations

AMI: acute myocardial infarction; ASE: American Society of Echocardiography; BMI: body mass index; CMR: cardiovascular magnetic resonance; EACVI: European Association of Cardiovascular Imaging; ECG: electrocardiogram; EDA: end-diastolic area; ESA: end-systolic area; ESC: The European Society of Cardiology; FAC: fractional area change; LAD: left anterior descending artery; LAVI: left atrial volume index; LCx: left circumflex artery; LM: left main artery; LV: left ventricle; LVDd: left ventricular diameter in diastole; LVDs: left ventricular diameter in systole; LVEF: left ventricular ejection fraction; LVGLS: left ventricular global longitudinal strain; MACEs: major adverse cardiovascular events; NSTEMI: non-ST-segment elevation myocardial infarction; PCI: percutaneous coronary intervention; RCA: right coronary artery; RV: right ventricular; RV4CSL: right ventricular four-chamber longitudinal strain; RVD1: right ventricular basal diameter; RVD2: right ventricular mid diameter; RVD3: right ventricular longitudinal linear dimension; RVFWSL: right ventricular free wall longitudinal strain; RVLS: right ventricular longitudinal strain (including RVFWSL and RV4CSL); RVS': Doppler tissue imaging-derived tricuspid lateral annular systolic velocity; STEMI: ST-segment elevation myocardial infarction; TAPSE: tricuspid annular plane systolic excursion; TIMI: thrombolysis in myocardial infarction

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