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Effect of reperfusion time on right ventricular remodeling in inferior ST-elevation myocardial infarction patients undergoing primary percutaneous coronary intervention

Jagaddhito Probokusumo[1], Hasanah Mumpuni[1], Pratiwi Noor Purnama Putri Sanggula[1], Hendry Purnasidha Bagaswoto*[1]

[1]Department of Cardiology and Vascular Medicine - Faculty of Medicine, Public Health and Nursing, Gadjah Mada University, Yogyakarta, Indonesia

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***Corresponding author:**
arsyadhiracarissa@gmail.com

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ABSTRACT

Background: Right ventricle (RV) remodeling and dysfunction are frequent in inferior ST-elevation myocardial infarction (STEMI) patients and possibly implicated to poor outcomes.
Objective: The purpose of this study is to investigate the influence of reperfusion time on RV remodeling in patients with inferior STEMI who had primary percutaneous coronary intervention (PPCI).
Methods: The subjects were patients with inferior STEMI who had undergone PPCI and met the inclusion and exclusion criteria. From September 2021 to April 2022, samples were taken in the order in which they arrived at the Dr. Sardjito Hospital in Yogyakarta, Indonesia. To investigate the occurrence of RV remodeling, 2D-transthoracic echocardiography was performed before PPCI as baseline and 3-months after PPCI. Confounding factors were investigated using bivariate and multivariate analysis.
Results: The RV remodeling was observed in 12 (28.6%) of 42 inferior STEMI patients undergoing PPCI. The RV remodeling group had a longer median reperfusion time (798.5 vs 710 minutes, p=0.568). The baseline RV end-systolic area (RVESA) and RV end-diastolic area (RVEDA) had significant correlations with the occurrence of RV remodeling (p=0.046; p=0.008, respectively). The tricuspid annular plane systolic excursion (TAPSE) rose considerably in both groups during the 3-month follow up. There were significant variations in RVEDA and RV basal diameter in the RV remodeling group, but there was a substantial rise in fractional area change (FAC) and RVESA in the non-RV remodeling group.
Conclusion: In patients with inferior STEMI undergoing PPCI, reperfusion time showed no significant effect on RV remodeling.

INTRODUCTION

The CICU (cardiovascular intensive care unit) is a unit in a hospital where patients with cardiovascular problems are treated. The most frequent diagnosis in the CICU was acute coronary syndrome (69.9%). The STEMI was more prevalent than non-ST elevation-acute coronary syndrome (NSTE-ACS) (67.7%).[1,2] This result aligns with the findings of Bohula et al. who discovered that acute coronary syndrome (ACS) (31.8%) and heart failure (18.6%) were the two most frequent diagnoses admitted to CICU.[3] Meanwhile, cardiovascular diseases account for one-third of all deaths in Indonesia. Bagaswoto et al. discovered that cardiogenic shock was the most prevalent cause of death in the CICU.[4] This demonstrates that coronary heart disease is one of the most frequent causes of death in Indonesia.

Inferior STEMI is a frequent form of STEMI that is caused by stenosis or blockage of the right coronary artery (RCA), resulting in RV failure. Remodeling of RV and RV dysfunction are frequent in patients with inferior STEMI and may be related to poor outcomes after inferior acute myocardial

infarction (AMI).[5]

Remodeling is a complex process post-AMI involving biomolecular, cellular, and structural alterations.[6] Cardiomyocytes are the most important cardiac cells involved in the cardiac remodeling process. In contrast, various explanations of cardiac remodeling involve similar molecular, biochemical, and mechanistic pathways. Furthermore, the interstitial tissue, fibroblasts, collagen, coronary vessels, hemodynamic load, and neurohumoral activation influence the cardiac remodeling process.[7] Ventricle remodeling is a compensation process secondary to both intrinsic and extrinsic stimuli. Myocyte hypertrophy and hyperplasia cause RV geometrical changes, whereas apoptosis leads to further myocardial changes and remodeling. Right ventricle remodeling due to STEMI is time dependent. The duration of coronary occlusion influences transmural damage and infarction extent. According to the class I recommendation of the ACS guidelines issued by the European Society of Cardiology (ESC), a PPCI strategy is preferred over fibrinolysis if the estimated time from diagnosis to PCI is <120 min.[8] Reperfusion with an ischemia time of less than 120 minutes provides the greatest benefit in saving the heart muscle. Therefore, it is something that is recommended to reduce the mortality of AMI patients through a strategy of reducing management delays so that it will reduce the total length of ischemic time which is defined as the onset of pain until restoration of coronary blood flow. Measures to reduce ischemic time can be done through pharmacotherapy and PPCI strategy.[9] Hospitals with catheterization rooms are the only ones equipped to do PPCI reperfusion. This facility is currently only found in a very small number of Indonesian hospitals. Reaching the optimal reperfusion time remains difficult in Indonesia because of the distance between the referring healthcare facility and the hospital with catheterization room facilities. The size of the infarction will undoubtedly change with extended reperfusion time. It is therefore crucial for Indonesia to establish a referral system for STEMI care to reduce the amount of time that must pass until reperfusion time is reached.

In recent decades, the examination of left ventricular (LV) functional characteristics has been supplemented by the assessment of RV function, which has grown in popularity and significance. The gradual acceptance and incorporation of RV function into the comprehensive assessment of cardiac function could be partly attributed to challenges in visualizing the full RV, irregularities in the analysis of RV parameters, and inadequate comprehension of the prognostic significance of RV. Important informations about RV have been obtained using a range of characteristics, from M-mode to 2-dimensional (2-D) assessments of RV size and function.[10] RV volume and ejection fraction can only be determined with certain constraints when employing 2D echocardiography. The RV volume and ejection fraction measurements are made possible and repeatable by 3-dimensional echocardiography.[10] Significantly, compared to 2D measurements, 3D data exhibit a greater correlation when verified against cardiac magnetic resonance imaging (CMR). Through the establishment of reference values, the determination of RV volume and EF through 3D echocardiography holds the potential to enhance comprehension of RV function and facilitate investigation into its importance for research outcomes in diverse clinical circumstances.[10]

A crucial component of the clinical assessment of patients with different heart conditions is two-dimensional (2D) echocardiography. Assessing LV and RV function, identifying early post-AMI mechanical problems, and ruling out LV thrombus are all part of routine echocardiography performed on hospitalized AMI patients.[11] The analysis and interpretation of RV function, specifically RV systolic function, receives less attention than the majority of the parameters derived from cardiac ultrasonography examinations, which primarily focus on LV function. The RV was considered more important than LV in the past. The global function of the entire RV is not represented by the RV function parameters found by 2D echocardiography, and it is nearly impossible to accurately estimate the RV's volume and ejection fraction from numerous 2D echocardiography views. Right heart characteristics, however, are crucial in cardiac remodeling.[10] Reconstructing the RV form from numerous 2D image planes is challenging due to the intricate nature of the RV contour. Therefore, to characterize RV function, echocardiographers have employed surrogate metrics. The most commonly reported 2D echocardiographic measures of RV function among them are FAC RV with an apical 4-chamber view and TAPSE.[10] The TAPSE is the

length of time measured at the lateral corner of the tricuspid annulus between the end of diastole and the end of systole. With minimal observer variability, TAPSE has been confirmed to have a good correlation with the RV ejection fraction as determined by radionuclide angiography. The heart's sliding motion within the chest cavity could have an impact on TAPSE readings, as it is angle-dependent. Furthermore load-dependent, TAPSE rises with severe tricuspid regurgitation; nevertheless, TAPSE may seem within normal limits when there is just a mild decrease in RV systolic function.[10]

Right ventricle measurement in 2D echocardiography is based on the RV's enhanced end-systolic area, end-diastolic area, decreased TAPSE, and increased FAC. The benefits of 2D echocardiography include accessibility, speed, reproducibility, and low cost. The right ventricle is an important part of cardiac function. In patients with myocardial infarction exacerbated by cardiogenic shock, RV function is found to be a significant predictor of CICU mortality and related to 28-day mortality.[4,12] In Indonesia, no research investigation incorporating RV remodeling following PPCI in patients with inferior STEMI has ever been conducted. Given the limited diagnostic tools available in Indonesia, RV assessment using echocardiography is the most practical method, faster, less expensive, and more widely available, since the gold standard for assessing ventricular remodeling is MRI CMR is still very limited.[13] Consequently, the aim of the present study was to assess the association between reperfusion time and RV remodeling in patients with inferior STEMI at Dr. Sardjito Hospital Yogyakarta, Indonesia.

METHODS

Research design

This prospective cohort study was undertaken on patients with acute inferior STEMI who came to Dr. Sardjito's Hospital emergency department in Yogyakarta and underwent reperfusion with a PPCI. This study was conducted from September 2021 until July 2022.

Population and sample

Sampling was taken consecutively according to arrival. There was a total of at least 37 subjects required as calculated. The sample size was obtained from the formula for testing the difference in means of two unpaired groups with n= [(Zα + Zβ)S/X1-X2]2, where Zα(5%) and Zβ(20%) were 1.96 and 0.84 respectively; X1 is the mean reperfusion time 212 ± 92 minutes;14 X1-2 is the minimum difference which is considered significant, 20%, namely 42.4 minutes (20%x212); S is the standard deviation with a value of 92.

The inclusion criteria included: aged ≥18 years; diagnosed with inferior STEMI; underwent reperfusion with PPCI; the coronary artery is classified as the thrombolysis in myocardial infarction (TIMI) grade 3 flow post-PPCI; and agreed to participate. Patients with ACS before recent admission; and suspected or diagnosed with coronavirus disease 2019 (COVID-19) were excluded. Patients who died during the 3-month post-PPCI follow-up period or were lost to follow-up were considered drop-outs.

Baseline data collection

Patients with STEMI who were admitted through the Emergency Room (ER) were assessed for suitability to the inclusion criteria. Baseline data included demographic, clinical profiles, time parameters, and baseline echocardiography parameters. Hypertension was stated by systolic blood pressure ≥140 mmHg and/or diastolic ≥90 mmHg, or based on a history of hypertension recorded in the medical record and/or having received anti-hypertensive treatment; Diabetes Mellitus (DM) was declared by patients who have fasting plasma glucose ≥126 mg/dL, or plasma glucose ≥ 200 mg/dl 2 hours after an oral glucose tolerance test with a glucose load of 75 grams, or current plasma glucose ≥200 mg/dL with classic complaints, or hemoglobin A1c ≥ 6.5%, using standardized methods, or based on a history of DM in the medical record and/or having received DM treatment; Current smokers and ex-smokers are included as smoking patients, where current smoker must had a history of smoking in the last 1 month and ex-smoker had a history of quitting smoking for at least 1 month; Hyperuricemia was declared based on the results of a uric acid examination with a value of >5.7 according to the reference value of Dr. Sardjito Hospital; Dyslipidemia was measured based on the main lipid fraction abnormalities, namely increased levels of total cholesterol, low-density lipoprotein (LDL), triglycerides and decreased high density lipoprotein (HDL) levels. We used

the European Atherosclerosis Society (EAS) simple classification to determine dyslipidemia, namely: Hypercholesterolemia (increased LDL lipoprotein, total cholesterol > 240 mg/dL), Hypertriglyceridemia (increased very low-density lipoprotein (VLDL), triglycerides > 200 mg/dL), Mixed dyslipidemia (elevated VLDL+LDL, triglycerides > 200mg/dL + total cholesterol > 240 mg/dL); High-sensitivity troponin I values were obtained from the patient's initial examination in the ER; Culprit vessels are defined as coronary arteries that have total obstruction on STEMI identified through diagnostic angiography at the time of PCI; Multivessel disease, namely lumen stenosis ≥ 70% in ≥ 2 main coronary artery branches or in one main coronary artery branch with 50% stenosis in the left main coronary artery obtained on diagnostic angiography for PCI; Use of ACE inhibitors/angiotensin receptor blockers (ARB)/beta blockers was assessed based on a history taking; The use of inotropic agents is expressed in the form of a history of use or administration of inotropes during hospitalization, both pre and post primary PCI; Use of a temporary pacemaker in the form of a history of use of a temporary pacemaker during hospitalization, both pre and post primary PCI. Reperfusion using PPCI was carried out according to the procedure at Dr. Sardjito Hospital, Yogyakarta. Ischemia time was calculated in minutes. Determination of onset was based on anamnesis on the subject directly or the subject's family if it is not possible to carry out directly on the subject. Determination of the time a patient was diagnosed with STEMI and the time of reperfusion was performed using a digital clock in the resuscitation room and catheter laboratory room at Dr. Sardjito Hospital Yogyakarta.

Echocardiography parameters

The echocardiography was performed before PPCI (pre-PPCI) and 3 months after PPCI in the catheter laboratory and echocardiogram room of the Integrated Heart Center Installation of Dr. Sardjito Hospital, Yogyakarta. Echocardiography was executed using a GE (General Electric, USA) VIVID S6 device and analyzed by two blinded reviewers. The RV end-diastolic area was measured using 2D echocardiography by manually tracing the ventricular endocardium in the apical four-chamber view of the RV focus, excluding the trabeculae during the diastolic phase shown by the ECG waveform, values were expressed in cm^2 units. The RV end-systolic area was measured using 2D echocardiography by manually tracing the ventricular endocardium in the apical four-chamber view of the RV focus, excluding the trabeculae during the systolic phase shown by the ECG waveform, values were expressed in cm^2 units. The RV FAC was calculated using the formula FAC= (RV end-diastolic area - RV end-systolic area/ RV end-diastolic area) x100%; values were expressed in % units. Tricuspid annular plane systolic excursion measurement of the RV regional systolic function index was performed by measuring the excursion of the lateral tricuspid annulus from the end of diastole to the end of systole and only reflects the longitudinal contraction of the RV free wall; values were expressed in mm.

Data analysis

The 2-D echocardiography data was sent to a workstation computer for subsequent online analysis. The analysis was carried out intra-observer and interobserver by 2 expert examiners; a cardiovascular technician and a heart and blood vessel specialist, and an echocardiography consultant, who was blind to the subject's clinical data, who had previously measured the Cohen's kappa (K) value. If the value of $K > 0.5$ with $p < 0.05$ when measuring, then the results are considered the same and can be used as an assessment. Statistical analysis used the Statistical Package for Social Sciences (SPSS) software version 25 for the Windows operating system.

Ethics

This study is a part of the main research titled, "The factors affecting RV remodeling in patients with inferior STEMI". The Medical and Health Research Ethics Committee (MHREC) of the Faculty of Medicine, Public Health, and Nursing, Universitas Gadjah Mada/Sardjito General Hospital Yogyakarta acknowledged this study (ref number: KE/FK/0385/EC/2021) and the Ethics Committee waived the requirement for informed consent.

RESULTS

There were 73 patients with inferior STEMI who underwent PPCI and had no history of prior ACS, with 31 patients who were drop-outs, leaving the total of 42 patients enrolled in the study. The research flow of the research subject can be seen

in Figure 1.

Demographic characteristics of the subjects are provided on Table 1. This study's subjects were grouped into two groups, the remodeling (n=12) and the non-remodeling (n=30). The RV remodeling group had a longer median reperfusion time (798.5 vs 710 minutes); however, it was not statistically significant (p=0.568), indicating that reperfusion time is not associated with RV remodeling in patients with inferior STEMI post PPCI.

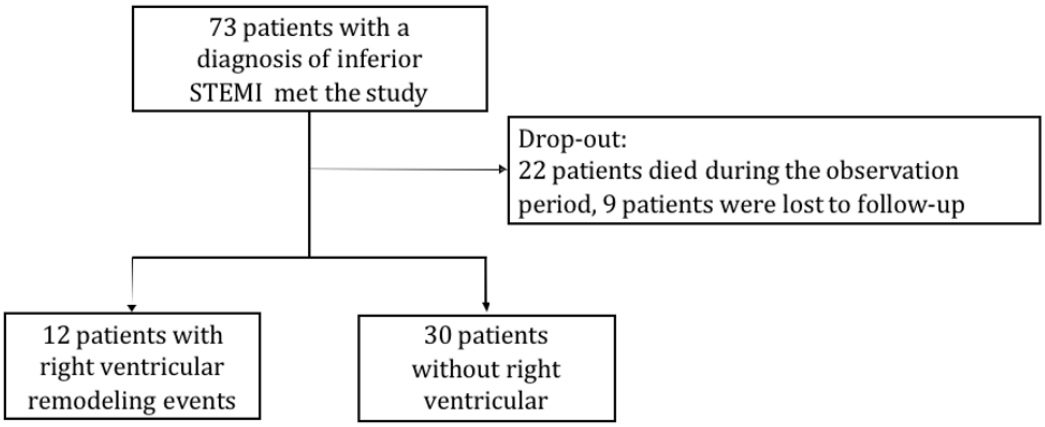

Figure 1. Research subject flow; STEMI: ST-elevation myocardial infarction

Table 1. Baseline characteristics of the participants

Variables	Mean ± SD or median (min-max)	n	%
Age (years)	58.45 ± 8.32		
Gender			
Female		8	19.00
Male		34	81.00
Body Mass Index (kg/m2)	24.7 (22.4 – 26.7)		
Smoking			
Yes		25	59.50
No		17	40.50
Hypertension			
Yes		29	69.00
No		13	31.00
Diabetes mellitus			
Yes		14	33.30
No		28	66.70
Dyslipidemia			
Yes		30	71.40
No		12	28.60
Hyperuricemia			
Yes		16	38.10
No		26	61.90
hs-Trop I (ng/L)	1,151.9 (110 – 28,882.7)		
Diagnosis			
Simple inferior STEMI		11	26.20
Complex inferior STEMI		31	73.80

Variables	Mean ± SD or median (min-max)	n	%
Atrial fibrillation			
Yes		1	2.40
No		41	97.60
Time parameter			
Reperfusion time (minutes)	720 (540 – 1,068)		
Crossing wire time (minutes)	180 (150 – 200)		
Culprit vessel			
Right coronary artery		40	95.20
Left circumflex artery		2	4.80
Multivessel disease			
Yes		31	73.80
No		11	26.20
Baseline echocardiography parameter			
Left ventricle ejection fraction (%)	46.61 ± 10.59		
TAPSE (mm)	16 (13 – 19)		
Fractional area change (%)	34.76 ± 7.75		
RV end-diastolic area (cm^2)	17.44 ± 3.40		
RV end-systolic area (cm^2)	11.42 ± 2.79		
Right ventricle basal diameter (mm)	34.19 ± 5.12		
Double anti platelet therapy			
Yes		42	100.00
No		0	0.00
ACE inhibitors/ARB drugs			
Yes		34	81.00
No		8	19.00
Beta-blocker			
Yes		25	59.50
No		17	40.50
Statin			
Yes		42	100.00
No		0	0.00
Inotropic agent			
Yes		7	16.70
No		35	83.30
Transient pacemaker			
Yes		4	9.50
No		38	90.50
Right ventricle remodeling			
Yes		12	28.60
No		30	71.40

ARB: Angiotensin-II Receptor Blocker; ACE: Angiotensin Converting Enzyme; STEMI: ST-Elevation Myocardial Infarction; SD: standard deviation; TAPSE: Tricuspid Annular Plane Systolic Excursion; RV: Right Ventricle; hs-Trop I: High Sensitivity Troponin I

Bivariate analysis was applied to examine possible confounding factors using Chi-square, Unpaired t-tests, Mann-Whitney, and Fisher's exact tests. Table 2 shows the findings of the bivariate analysis. There were however, no significant associations between age, gender, body mass index (BMI), smoking, comorbidity, culprit vessel, multivessel disease, and the treatment given. In baseline echocardiography, there were no significant differences between the two groups for FAC, tricuspid annular plane systolic excursion (TAPSE), and RV basal diameter, whereas RV end-diastolic area (RVEDA) (p=0.008) and RV end-systolic area (RVESA) (p=0.046) were significantly lower in the RV remodeling group.

There were five echocardiography parameters of RV function, namely TAPSE, RVEDA, RVESA, FAC, and RV basal diameter, that were observed during baseline and 3 months of post PPCI. The results are presented in Figure 1 and analyzed using paired t-tests comparing the two groups, which is presented on Table 3. There was improvement in the 3rd month post PPCI compared to the baseline with a gap of Δ=4 mm (p=0.015) in the RV remodeling group and Δ=4 mm (p=0.00) in the non-remodeling group. The difference was significant in both groups (p<0.05).

The trend of FAC was increasing compared to the baseline in the RV remodeling group, however, it was not significant (Δ=8.85%, p=0.054). Conversely, there was a significant increment of FAC value in the non-remodeling group between the 3 months post PPCI and baseline (Δ=8.66%, p=0.001). The lower RVEDA in the RV remodeling group suggests a RV dilatation, which was significant in the remodeling group but not in the other group. The RVESA, on the other hand, was not significantly decreased in the RV remodeling group. Finally, the RV basal diameter increased significantly in both groups (Table 3).

Table 2. Bivariate analysis of confounding variables of RV remodeling

	RV remodeling		p-value
	Yes (n = 12)	No (n = 30)	
Age	58.0 ± 9.1	58.6 ± 8.1	0.827
Gender			
Male	11 (91.7%)	23 (76.7%)	0.402$
Female	1 (8.3%)	7 (23.3%)	
Smoking	9 (75.0%)	16 (53.3%)	0.300$
Hypertension	8 (66.7%)	21 (70%)	1.000$
Diabetes mellitus	4 (33.3%)	10 (33.3%)	1.000$
Dyslipidemia	8 (66.7%)	22 (73.3%)	0.715$
Culprit vessel			
Right coronary artery	11 (91.7%)	29 (96.7%)	0.495$
Left circumflex artery	1 (8.3%)	1 (3.3%)	
Multivessel disease	8 (66.7%)	23 (76.7%)	0.699$
ACE inhibitors/ARB	8 (66.7%)	26 (86.7%)	0.195$
Beta-blockers	6 (50%)	19 (63.3%)	0.498$
Baseline echocardiography			
TAPSE (mm)	17.5 (15.5-19)	15.5 (12-19)	0.170
Fractional area change (%)	34.9 ± 9.5	34.7 ± 7.1	0.946
RV end-diastolic area (cm^2)	15.3 ± 2.8	18.3 ± 3.3	0.008*
RV end-systolic area (cm^2)	10.1 ± 2.7	11.9 ± 2.7	0.046*
RV basal diameter (mm)	32.9 ± 6.1	34.7 ± 4.7	0.314

ARB: Angiotensin-II Receptor Blocker; ACE: Angiotensin Converting Enzyme; TAPSE: Tricuspid Annular Plane Systolic Excursion; RV: right ventricle; Data were expressed as mean + SD or median (min-max) or n (%);* significance p <0.05; $; Fisher exact test

Table 3. Echocardiography difference before and 3 months after primary PCI

		Before primary PCI	3 months after	Gap	p-value
RV remodeling	TAPSE (mm)	17.5 (13-20)	21 (13-26)	4 (-6-11)	0.015*
	Fractional area change (%)	34.89 ± 9.54	43.74 ± 8.32	8.85 ± 14.2	0.054
	RV end diastolic area (cm^2)	15.28 ± 2.80	21.86 ± 4.82	6.57 ± 3.56	0.001*
	RV end systolic area (cm^2)	10.08 ± 2.70	12.58 ± 4.91	2.50 ± 4.87	0.103
	RV basal diameter (mm)	32.92 ± 6.14	38.57 ± 7.51	5.83 ± 5.67	0.004*
No remodeling	TAPSE (mm)	15.5 (8-21)	20.5 (9-28)	4 (0-13)	0.001*
	Fractional area change (%)	34.71 ± 7.11	43.37 ± 9.11	8.66 ± 11.7	0.001*
	RV end diastolic area (cm^2)	18.30 ± 3.28	17.48 ± 4.14	-0.81 ± 2.76	0.117
	RV end systolic area (cm^2)	11.96 ± 2.68	9.91 ± 2.82	-2.05 ± 2.89	0.001*
	RV basal diameter (mm)	34.70 ± 4.67	37.10 ± 5.01	2.40 ± 6.75	0.061

TAPSE: Tricuspid Annular Plane Systolic Excursion; RV: Right ventricle; PCI: percutaneous coronary intervention; Data were expressed as mean + SD or median (min-max), *significant p<0.05 compared to before primary PCI.

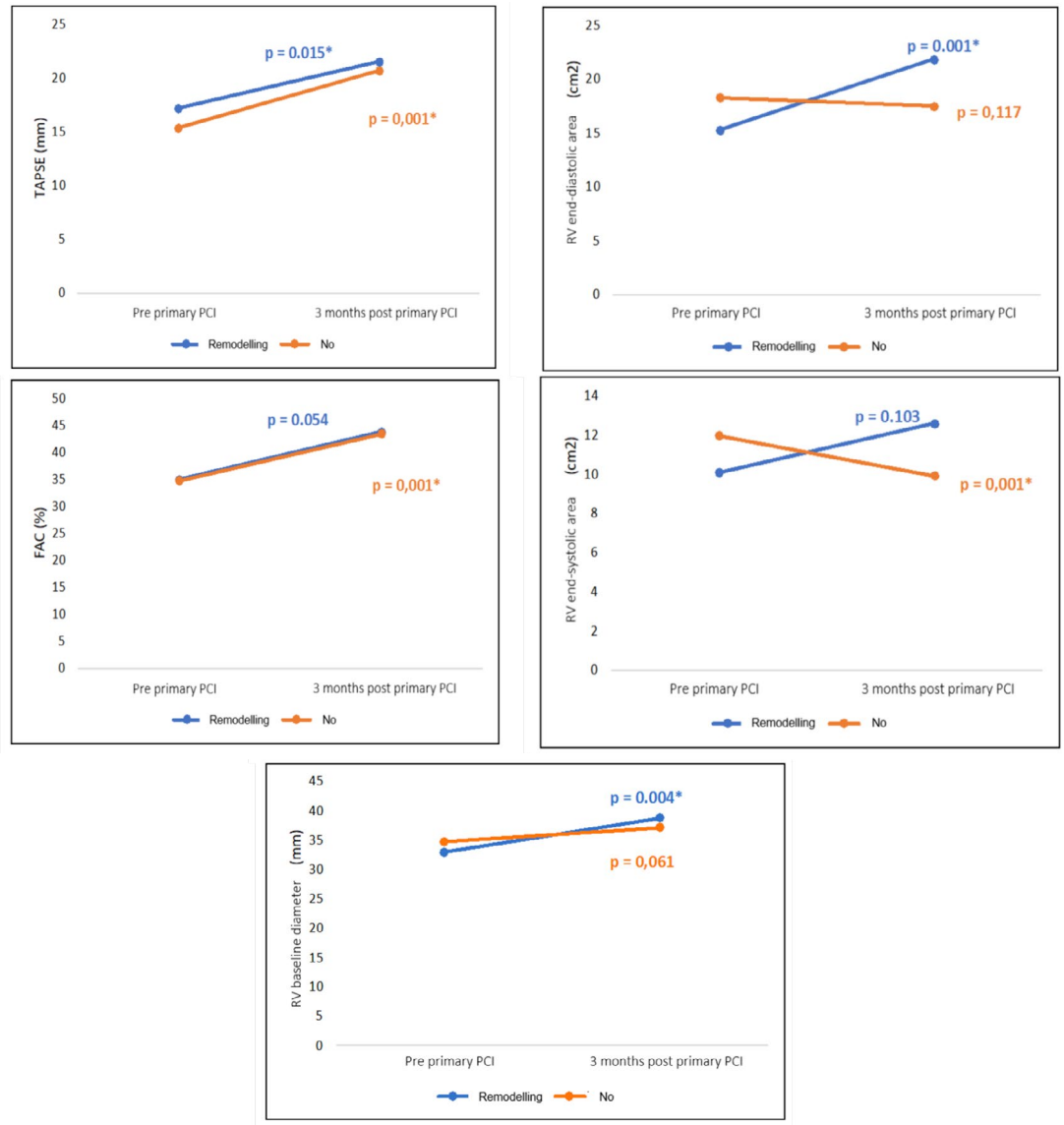

Figure 2. Echocardiographic parameters at baseline (pre Primary PCI) and 3 months after primary percutaneous coronary intervention

DISCUSSION

The patients with inferior STEMI in this study are predominantly male (81%) with the median age of 58.45 ± 8.32 years old. This finding is similar to previous studies where a recent study in 2019 showed a mean age of 61.3 ± 13.5 years.[15] Moreover, epidemiology shows that ACS is 3-4 times more common in males <60 years old, while for females, it is more common in the >75 years old age group.[11] The most frequent conventional risk factor (71.4%) is dyslipidemia, followed by hypertension, smoking, and diabetes mellitus. A lipid research clinics-coronary primary prevention trial found that patients with lower levels of total cholesterol and low-density lipoprotein (LDL) experienced a significant decrease in the incidence of coronary heart disease (CHD). The Framingham heart study found increased risk of CHD in pre-hypertensive and diabetic patients of adjusted age and ethnicity.[16] All of those patients received dual antiplatelet therapy (DAPT) or statin post-PPCI. The European Society of Cardiology (ESC) year 2017 recommended beta-blockers in STEMI patients with heart failure and/or left ventricle ejection fraction ≤40% and should be considered in those who have no contraindications.[12]

The mean reperfusion time (720 minutes) and crossing wire time (180 minutes) were below ESC standard (≤90 minutes for wire crossing).[12] The culprit vessel was mostly (95.2%) originated from the right coronary artery (RCA), and the rest were left circumflex artery (LCx). Previous studies found that the most common culprit vessel was found in the left anterior descending (LAD), followed by RCA and LCx.[17] Considering the sample population were patients with inferior STEMI, no culprit vessel originated from the LAD.

There was a decrease in Left Ventricular Ejection Fraction (LVEF) with the mean of 46.61 ± 10.59% and similar findings have been reported in multiple studies. There also was an abnormal median of TAPSE value, which was 16 (13-19) mm, and is aligned with the findings of research conducted by Park et al. with a TAPSE of 16 ± 4 mm. However, it was lower compared to Chimed et al. with a normal mean of TAPSE. There were some discrepancies of mean FAC and RVESA compared to other studies. The RV remodeling involves an increase of RVEDA ≥20%. Remodeling in the early AMI is mainly caused by fibrotic tissues, infarct extension, and elevated filling pressure.

The study found no evident correlation between both reperfusion time and remodeling of RV, where both groups showed a non-ideal reperfusion time based on the literature, even after age-matching analysis. Gorter et al. failed to find any decrease of right ventricular ejection fraction (RVEF) using cardiac magnetic resonance (CMR) at 4th month post-infarction, possibly due to a short reperfusion time, which was 156 minutes.[18] The longer the reperfusion time, the greater the infarct area, so the risk of heart failure will also increase.

Bivariate analysis in this study did not reveal any significant correlations between age and gender with both RV remodeling and/or dysfunction, in accordance with previous studies.[17] Some studies found no significant correlations regarding hypertension, dyslipidemia, and hyperuricemia. However, other research has found that those with diabetes and who smoke have a much-increased prevalence of RV remodeling.[17]

There were no significant correlations found between the culprit vessels for both RCA and LCx with RV remodeling. On the contrary, Chimed et al. found that RCA is more uncommon to be a culprit vessel in the RV remodeling group.[17] There was no correlation between treatment and RV remodeling since both groups received DAPT or statin, in accordance with ESC guidelines. The usage of ACE inhibitors/ARB and beta-blocker in RV remodeling was higher, however, it was not correlated with the incidence of remodeling. Galves et al. found that beta-blockers significantly improved RV systolic remodeling, especially when given early.[19] Numerous studies have shown that renin-angiotensin-aldosterone system (RAAS) inhibitors can restrict infarct extension and prevent remodeling. However, a meta-analysis found a non-significant association between ACE inhibitors/ARB and RV end-systolic and end-diastolic volume.[20]

The RV remodeling group showed lower areas of end-systolic and end-diastolic, and the remodeling group had higher baseline TAPSE, while Hoogslag et al. found the opposite. There appeared to be no significant relationship between initial TAPSE value and the incidence of RV remodeling. We found a significant increment of TAPSE in the 3rd month post PPCI in both groups. A faster RV recovery post-STEMI is associated with the RV resistance to ischemia, which results from a better supply and demand connection, smaller

mass, and lower afterload than LV. Moreover, extensive collateral is common and perfusion occurs in both diastole and systole. Different from TAPSE, the RVEDA and RVESA were progressively increased in the remodeling group, suggesting the extension of RV. This finding is supported by several studies with different follow-up times.[17] The FAC variable was only significant at the 3rd month evaluation post PPCI compared to baseline in the non-remodeling group. Previous study also found a significantly increased FAC between pre- and 2 months post PPCI in RCA occlusion.[21] Unfortunately, some literatures did not use the FAC parameter and RV basal diameter in their study and mostly used right atrial area and RV free wall longitudinal strain.

There are several limitations in our study. First, the median reperfusion time (720 minutes) is not ideal compared to the standard guideline (≤120 minutes). Second, other factors affecting RV remodeling have not been analyzed. Third, it is difficult to determine the exact onset of arterial occlusion since we depended only on the history from the family and/or patient.

CONCLUSION

It is concluded that reperfusion time was not significantly associated with the incidence of RV remodeling in inferior STEMI post PPCI. The RV remodeling group, on the other hand, had a longer period of reperfusion time. The incidence rate of RV remodeling in inferior STEMI post primary PPCI was 28.6%. The results of this study indicate that reperfusion time is not a factor that needs to be considered in the incidence of right ventricular remodeling in inferior STEMI patients undergoing PPCI. However, the results of this study cannot be used as a reference that achieving a fast reperfusion time is not necessary in cases of Inferior STEMI. Further research is needed to find out what factors influence the incidence of right ventricular remodeling in post-STEMI patients undergoing PPCI.

CONFLICT OF INTEREST

There are no conflicts of interest.

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AUTHOR CONTRIBUTION

JP: Conceptualization, Writing- Original draft preparation, Methodology, Software, Editing and Visualization. HM: Validation, Supervision, Reviewing. HPB: Methodology, Supervision, Writing-reviewing, Data Curation. PNPPS: Writing-reviewing, Editing.

LIST OF ABBREVIATIONS

ACS: acute coronary syndrome; ACE: angiotensin converting enzyme; AMI: acute myocardial infarction; ARB: angiotensin receptor blocker; BMI: body mass index; CHD: coronary heart disease; CICU: cardiovascular intensive care unit; CMR: cardiac magnetic resonance imaging; DAPT: dual antiplatelet therapy; ESC: European society of cardiology; FAC: fractional area change; LAD: left anterior descending; LCx: left circumflex artery; LDL: low density lipoprotein; LV: left ventricle; LVEF: left ventricular ejection fraction; NSTE-ACS: non ST-elevation acute coronary syndrome; PPCI: primary percutaneous coronary intervention; RAAS: renin-angiotensin-aldosterone system; RCA: right coronary artery; RVEF: right ventricular ejection fraction; RV: right ventricle; RVESA: RV-end systolic area; RVEDA: RV-end diastolic area; STEMI: ST-elevation myocardial infarction; TAPSE: tricuspid annular plane systolic excursion; TIMI: thrombolysis in myocardial infarction.

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