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Multivessel percutaneous coronary intervention in patients with acute myocardial infarction and severe renal dysfunction

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KEYWORDS

- multiple vessel disease
- non-STEMI
- renal insufficiency
- STEMI

Abstract

Aims: The aim of this study was to compare the outcomes between multivessel and infarct-related artery (IRA)-only percutaneous coronary intervention (PCI) in patients with acute myocardial infarction (AMI), multivessel disease (MVD), and severe renal dysfunction (RD) using the nationwide AMI registry.

Methods and results: Among 13,104 patients, 537 diagnosed with AMI and MVD who had severe RD at presentation (estimated glomerular filtration rate [GFR] <30 mL/min/1.73 m^2, mean: 19.1±7.5 mL/min/1.73 m^2) and underwent PCI during index hospitalisation were selected. The patients were classified according to treatment strategy, i.e., multivessel PCI (49.0%) or IRA-only PCI. The primary endpoint was major adverse cardiac events (MACE), a composite of all-cause death, myocardial reinfarction, re-hospitalisation for heart failure, and any repeat revascularisation at one year. The safety outcome was the worsening of renal function (WRF), defined as a 30% reduction in estimated GFR from baseline to 12-month follow-up. The adjusted MACE risks were similar in groups after Cox regression (41.8% vs 39.8%, hazard ratio [HR] 1.008 [0.743-1.367]) and propensity score-matching analysis (HR 0.974 [0.651-1.377]). Multivessel PCI showed a significant tendency of higher rates of WRF (24.8% vs 11.1%, adjusted odds ratio 2.134 [0.976-4.668]).

Conclusions: Multivessel PCI was associated with similar outcomes compared to IRA-only PCI in patients with AMI, MVD, and severe RD.

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Abbreviations

ACC/AHA	American College of Cardiology/American Heart Association
AMI	acute myocardial infarction
CI	confidence interval
GFR	glomerular filtration rate
HR	hazard ratio
IRA	infarct-related artery
KAMIR-NIH	Korean Acute Myocardial Infarction Registry-National Institutes of Health
MACE	major adverse cardiac events
MVD	multivessel disease
PCI	percutaneous coronary intervention
RD	renal dysfunction
STE	ST-segment elevation
WRF	worsening of renal function

Introduction

In contemporary practice, among patients with ST-segment elevation acute myocardial infarction (STE-AMI) in need of primary percutaneous coronary intervention (PCI), between 40% and 65% have concurrent multivessel disease (MVD), which has been associated with worse clinical outcomes[1]. Although the findings suggesting a beneficial effect of multivessel PCI in recently published randomised trials have led to changes in major cardiology societies' guidelines, now supporting multivessel intervention (Class IIa, level of evidence A)[2], patients with severe renal dysfunction (RD) were excluded in the majority of the trials reported to date[3-6]. These patients with severe RD exhibit highly complex and different pathogenic processes of coronary arterial luminal narrowing compared with individuals with normal renal function; some treatments have no definite clinical benefit, similar to statins in individuals with end-stage RD[7]. Thus, whether multivessel PCI should be conducted during index hospitalisation in patients with AMI, MVD, and severe RD remains controversial.

Therefore, we aimed to compare the clinical outcomes between multivessel PCI and infarct-related artery (IRA)-only PCI in patients with AMI who had MVD accompanied by severe RD using a large-scale, nationwide, multicentre, dedicated registry for AMI.

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Methods

STUDY PROTOCOL AND POPULATION SELECTION

The study population was derived from the nationwide, multicentre, prospective Korean Acute Myocardial Infarction Registry-National Institutes of Health (KAMIR-NIH). The KAMIR-NIH is a dedicated prospective, web-based observational cohort study that consecutively enrolled patients diagnosed with AMI who were eligible for primary PCI at 20 tertiary university hospitals from November 2011 to December 2015 without any exclusion criteria[8]. The protocol of the KAMIR-NIH was approved by the ethics committee at each participating centre and all patients provided written informed consent upon enrolment. Among 13,104 patients who were enrolled in the KAMIR-NIH, those with AMI and MVD who also presented with severe RD and underwent PCI were selected. Patients were excluded from the analysis if they underwent thrombolysis before PCI, presented in cardiogenic shock, had single-vessel disease, or were lost to follow-up before one year. Finally, 537 patients were selected for this analysis. These patients were then classified according to treatment strategy (i.e., multivessel PCI or IRA-only PCI) **(Figure 1)**. Patients who underwent non-IRA PCI at the time of primary PCI or within index hospitalisation were included in the multivessel PCI group.

DEFINITIONS

The specific definition of AMI is presented in **Supplementary Appendix 1**. The glomerular filtration rate (GFR) was used to determine renal function. Under steady-state conditions, the GFR can be estimated from the serum creatinine using the Cockcroft-Gault formula. Severe RD was defined as an estimated GFR of <30 (mL/min/1.73 m^2)[9]. MVD was defined as ≥50% diameter stenosis in at least one major non-IRA or in the left main coronary artery, as in previous trials[10]. PCI was considered successful if the final residual stenosis was <30% with Thrombolysis In Myocardial Infarction (TIMI) grade 3 flow.

PATIENT MANAGEMENT, DATA COLLECTION, AND FOLLOW-UP

Patient management was performed in accordance with the current standard AMI guidelines. The patients who underwent PCI received 300 mg aspirin and 600 mg clopidogrel, 180 mg ticagrelor, or 60 mg prasugrel before PCI. Unfractionated heparin (50 to 70 U/kg) was administered before or during PCI to maintain an activated clotting time of 250 to 300 seconds. Unless there was an acknowledged reason for discontinuing dual antiplatelet therapy, all patients were recommended to take 100 to 300 mg aspirin indefinitely in addition to 75 mg clopidogrel or other potent antiplatelet agents, such as 10 mg prasugrel once daily or 90 mg ticagrelor twice daily, for ≥1 year. The choice of the prescribed $P2Y_{12}$ inhibitor was left to the operator's discretion according to the guidelines and the patients' bleeding risk. Medications, including renin-angiotensin-aldosterone system blockers, beta-blockers, and statins, were also recommended in accordance with the practice guidelines. All data were collected by independent clinical research coordinators using a web-based case report form in the internet-based Clinical Research and Trial (iCReaT) management system, a data management system established by the Centers for Disease Control and Prevention, Ministry of Health and Welfare, Republic of Korea (iCReaT Study No. C110016). Clinical events that occurred during hospitalisation and within the one-year follow-up were examined.

STUDY ENDPOINTS

The primary study endpoint was major adverse cardiac events (MACE) within 12 months, a composite of all-cause death, myocardial reinfarction, re-hospitalisation for heart failure, and any

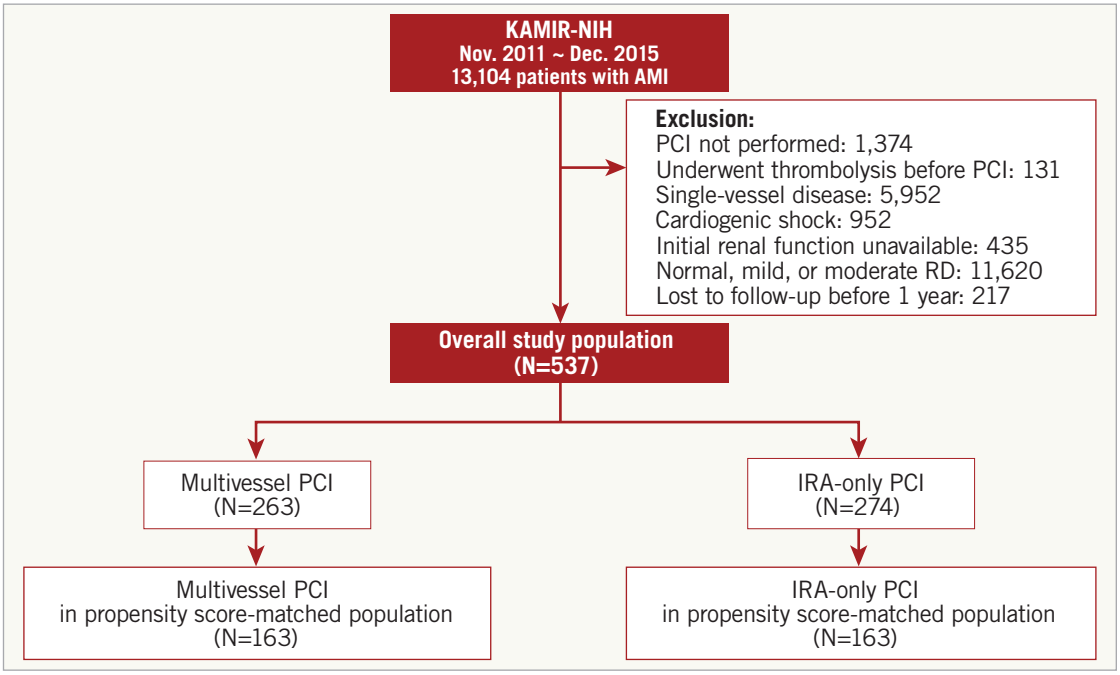

Figure 1. *Study flow. AMI: acute myocardial infarction; IRA: infarct-related artery; KAMIR-NIH: Korean Acute Myocardial Infarction Registry-National Institutes of Health; PCI: percutaneous coronary intervention; RD: renal dysfunction*

repeat revascularisation at one year. The secondary endpoints were the individual components of MACE, non-IRA repeat revascularisation, and definite or probable stent thrombosis at one year. All clinical outcomes were defined in accordance with the Academic Research Consortium[11]. In addition, the safety outcome was the worsening of renal function (WRF), defined as a 30% reduction in estimated GFR from baseline to 12-month post-AMI follow-up.

STATISTICAL ANALYSIS

Cox proportional hazard regression in a propensity score-matched population was performed. A multivariable logistic regression model was used to generate the propensity scores, which indicate the probability of the patients being treated with the multivessel PCI strategy. All available covariates were included in this model, following the recommendations for analysis using the propensity score exactly[12]. For the propensity score matching, a 1:1 matching process without replacements was performed using a greedy algorithm with a calliper width of 0.2 standard deviations, yielding 163 patients in the multivessel PCI group matched with 163 controls in the IRA-only PCI group. Balance between the two groups after propensity score matching was assessed by calculating the percent standardised mean differences in the covariates used in the propensity score generation. The percent standardised mean differences after propensity score matching were within ±10% across all matched covariates, demonstrating successful balance achievement between the groups **(Supplementary Table 1)**. An extended description of the statistical analysis is presented in **Supplementary Appendix 1**.

Results

Of the total 13,104 patients who were enrolled in the KAMIR-NIH, 7,152 subjects had multivessel disease. Among them, 537 subjects were selected for the study: 263 (49.0%) underwent multivessel PCI, and 274 underwent IRA-only PCI. The baseline clinical characteristics, including medications at discharge, are summarised in **Supplementary Table 2**. The multivessel PCI group had a higher body mass index and a lower prevalence of previous cerebrovascular disease. The proportion of patients with non-STE-AMI at presentation was higher; the left ventricular ejection fraction (%) on echocardiography was lower in the multivessel PCI group than in the IRA-only PCI group. The baseline estimated GFR was 19.1±7.5 mL/min/1.73 m^2; there was no difference in the baseline estimated GFR between the groups. Also, the groups did not differ in terms of the levels of evidence-based medications taken to treat AMI (antiplatelets, beta-blockers, renin-angiotensin-aldosterone system blockers, and statins).

Supplementary Table 3 shows the angiographic and procedural features of all patients. The left main artery as the culprit vessel was more prevalent in the multivessel PCI group. Among the 263 patients in the multivessel PCI group, 205 (77.9%) underwent immediate non-IRA PCI, and 58 (22.1%) underwent staged non-IRA PCI during the same hospitalisation. The patients of the multivessel PCI group underwent less thrombus aspiration and received fewer stents. Based on the angiographic assessment findings, 56.7% of the multivessel PCI cases were classified as complete revascularisation without residual stenosis, and the remaining 43.3% were classified as incomplete revascularisation.

We found no significant between-group differences in the incidence of in-hospital complications **(Table 1)**, although the multivessel PCI group patients received more haemodynamic support.

The primary endpoint was achieved in a total of 219 (40.8%) patients at a median follow-up of 362 days (interquartile range, 286-383 days). A comparison of the clinical outcomes between the multivessel PCI and IRA-only PCI groups is presented in **Table 2**. The incidence of MACE and all-cause death was comparable between the groups **(Figure 2)**. Further, myocardial reinfarction, all-cause death or myocardial reinfarction, re-hospitalisation for heart failure, any repeat revascularisation, non-IRA repeat revascularisation, and definite or probable stent thrombosis did not differ significantly between the multivessel PCI group and the IRA-only PCI group **(Figure 3)**.

Multivariable Cox regression analysis **(Table 2)** revealed that the MACE were similar regardless of the initial PCI strategies. The adjusted risks for all-cause death, myocardial reinfarction, all-cause death or myocardial reinfarction, re-hospitalisation for heart failure, any repeat revascularisation, non-IRA repeat revascularisation, and definite or probable stent thrombosis were also similar in the two groups, irrespective of the initial PCI strategy.

After 1:1 propensity score matching, 163 patients were generated in each group. The C-statistic value for the propensity score model was 0.789 (95% confidence interval [CI]: 0.751-0.827), indicating good discrimination. There were no significant differences in the baseline clinical, angiographic, or procedural characteristics of the propensity score-matched cohort **(Supplementary Table 1)**. A total of 124 (38.0%) MACE occurred during follow-up in the matched population **(Supplementary Table 4)**. Sensitivity analyses using propensity score-matching adjustment consistently showed that the risks of all clinical outcomes were comparable between the multivessel PCI and IRA-only PCI groups **(Table 2)**.

Figure 4 presents the prognostic impact of multivessel PCI among the various subgroups. In the subgroup analysis, the similar risk of MACE between the multivessel PCI group and IRA-only PCI group was consistent across all subgroups without significant interaction p-values (elderly patients aged >70 years, women, patients with diabetes mellitus, patients with STE-AMI, patients with left ventricular ejection fractions of <40%, left main or left anterior descending artery as the culprit vessel, procedure for complex lesions [American College of Cardiology/American Heart Association (ACC/AHA) lesion classification type B2/C], and patients with three-vessel disease). Next, we examined whether the two different types of AMI, STE-AMI versus non-STE-AMI, were independently affected according to PCI strategy regarding clinical outcomes **(Supplementary Table 5, Supplementary Table 6)**.

The multivariable Cox proportional hazards models identified the independent predictors of MACE and all-cause death **(Supplementary Table 7)**. Multivessel PCI was not independently

Table 1. Comparison of in-hospital complication or clinical outcomes according to treatment strategy in the overall population.

	Overall population (n=537)	Multivessel PCI (n=263)	IRA-only PCI (n=274)	*p*-value
Haemodynamic support				
Intra-aortic balloon pump	45 (8.4)	25 (9.5)	20 (7.3)	0.356
Extracorporeal membrane oxygenation	9 (1.7)	7 (2.7)	2 (0.7)	0.100
In-hospital outcomes				
Cardiogenic shock	106 (19.7)	53 (20.2)	53 (19.3)	0.814
New heart failure	65 (12.1)	34 (12.9)	31 (11.3)	0.567
Major bleeding	10 (1.9)	7 (2.7)	3 (1.1)	0.214
Acute kidney injury	23 (4.3)	13 (4.9)	10 (3.6)	0.459

IRA: infarct-related artery; PCI: percutaneous coronary intervention

Table 2. Comparison of 1-year clinical outcomes according to treatment strategy.

	Overall (n=537)	Multivessel PCI (n=263)	IRA-only PCI (n=274)	Unadjusted in overall population		Adjusted in overall population		PS-matched population	
				HR (95% CI)	*p*-value	HR (95% CI)	*p*-value	HR (95% CI)	*p*-value
All-cause death	148 (27.6)	71 (27.0)	77 (28.1)	0.954 (0.691-1.317)	0.773	0.826 (0.510-1.338)	0.437	0.785 (0.498-1.238)	0.297
Reinfarction	27 (5.0)	12 (4.6)	15 (5.5)	0.830 (0.388-1.773)	0.630	0.989 (0.354-2.761)	0.983	0.544 (0.179-1.652)	0.283
All-cause death or reinfarction	165 (30.7)	78 (29.7)	87 (31.8)	0.922 (0.680-1.252)	0.605	0.719 (0.465-1.112)	0.138	0.760 (0.491-1.176)	0.218
Re-hospitalisation for heart failure	43 (8.0)	25 (9.5)	18 (6.6)	1.472 (0.803-2.698)	0.211	1.120 (0.573-2.187)	0.741	1.415 (0.706-2.834)	0.328
Any repeat revascularisation	38 (7.1)	18 (6.8)	20 (7.3)	0.938 (0.496-1.773)	0.844	0.810 (0.427-1.536)	0.518	0.845 (0.368-1.940)	0.691
Non-IRA repeat revascularisation	19 (3.5)	10 (3.8)	9 (3.3)	1.159 (0.471-2.852)	0.748	1.084 (0.440-2.670)	0.861	1.351 (0.458-3.983)	0.586
Definite or probable ST	4 (0.7)	3 (1.1)	1 (0.4)	3.150 (0.326-30.476)	0.322	2.701 (0.271-26.898)	0.397	2.012 (0.181-22.414)	0.570
MACE	219 (40.8)	110 (41.8)	109 (39.8)	1.039 (0.797-1.354)	0.780	1.008 (0.743-1.367)	0.961	0.947 (0.651-1.377)	0.774

CI: confidence interval; HR: hazard ratio; IRA: infarct-related artery; MACE: major adverse cardiac events (a composite of all-cause death, reinfarction, re-hospitalisation for heart failure, and any repeat revascularisation); PCI: percutaneous coronary intervention; PS: propensity score; ST: stent thrombosis

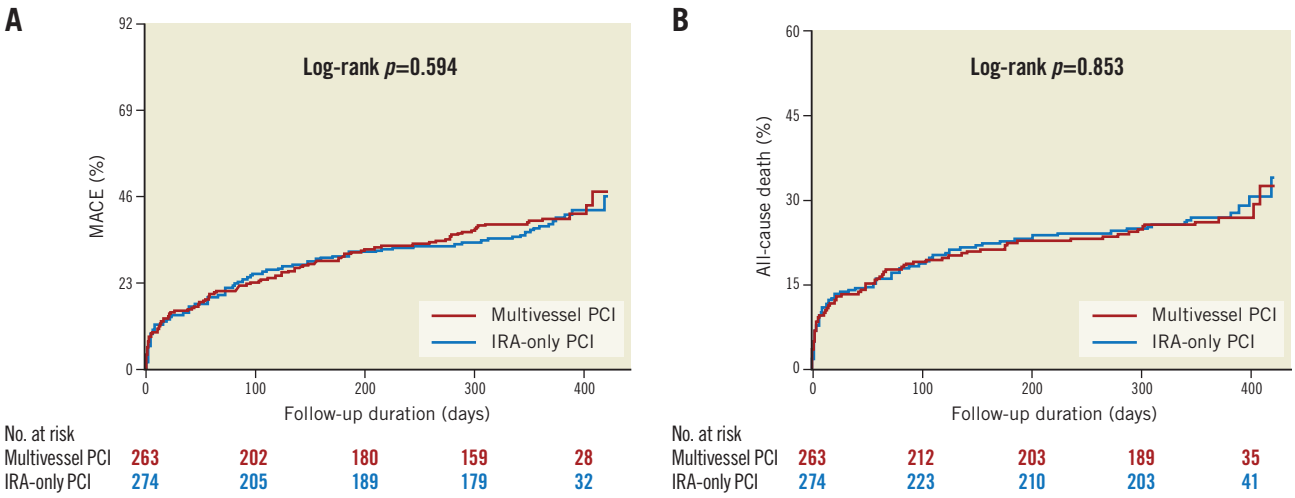

Figure 2. *Cumulative incidence of MACE and all-cause death. Kaplan-Meier curves with cumulative hazards of (A) MACE and (B) all-cause death compared according to the PCI strategy. IRA: infarct-related artery; MACE: major adverse cardiac events; PCI: percutaneous coronary intervention*

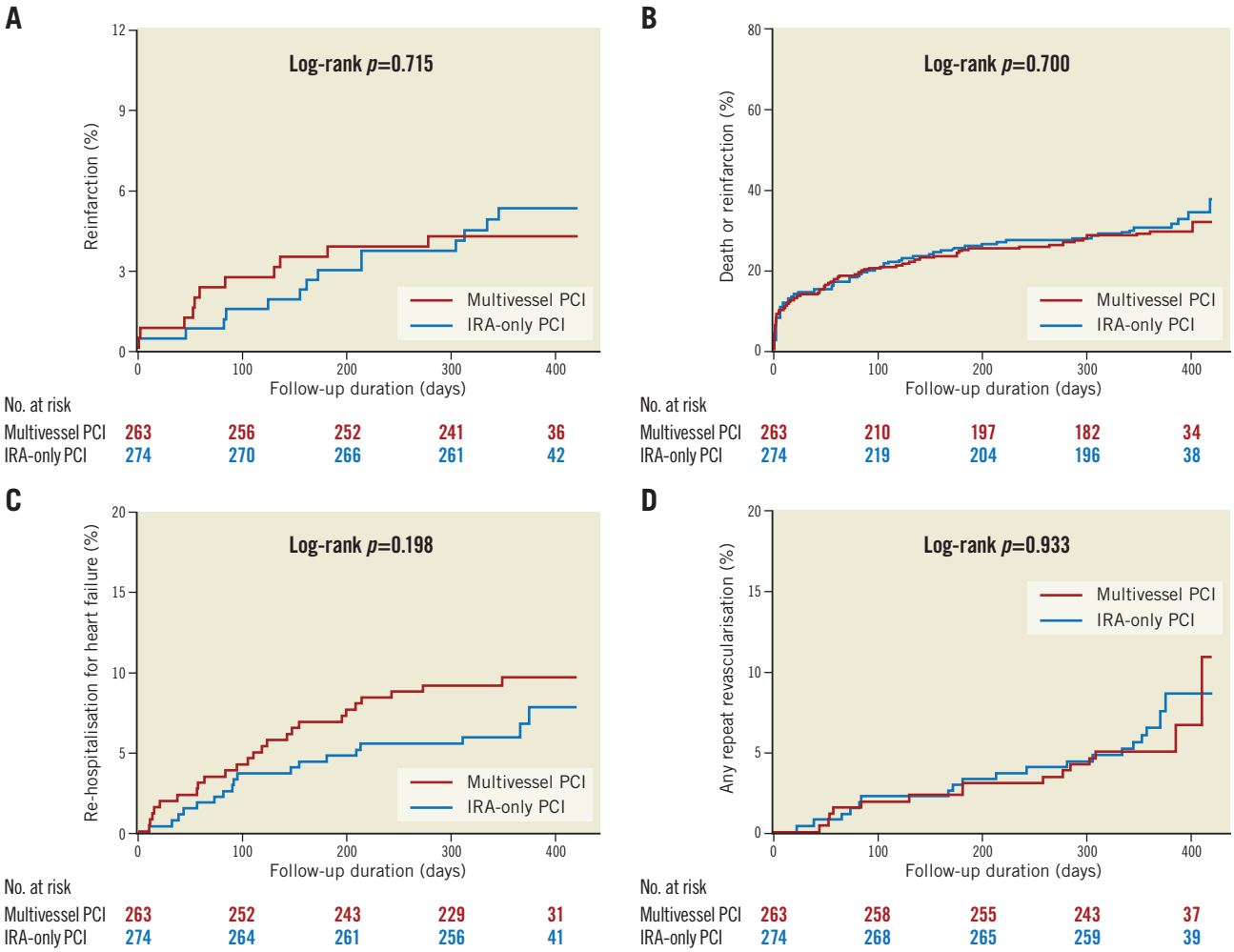

Figure 3. *Cumulative incidence of individual clinical outcomes. Cumulative incidence of individual clinical outcomes including components of the composite outcomes (A) reinfarction, (B) all-cause death or reinfarction, (C) re-hospitalisation for heart failure, and (D) any repeat revascularisation. IRA: infarct-related artery; MACE: major adverse cardiac events; PCI: percutaneous coronary intervention*

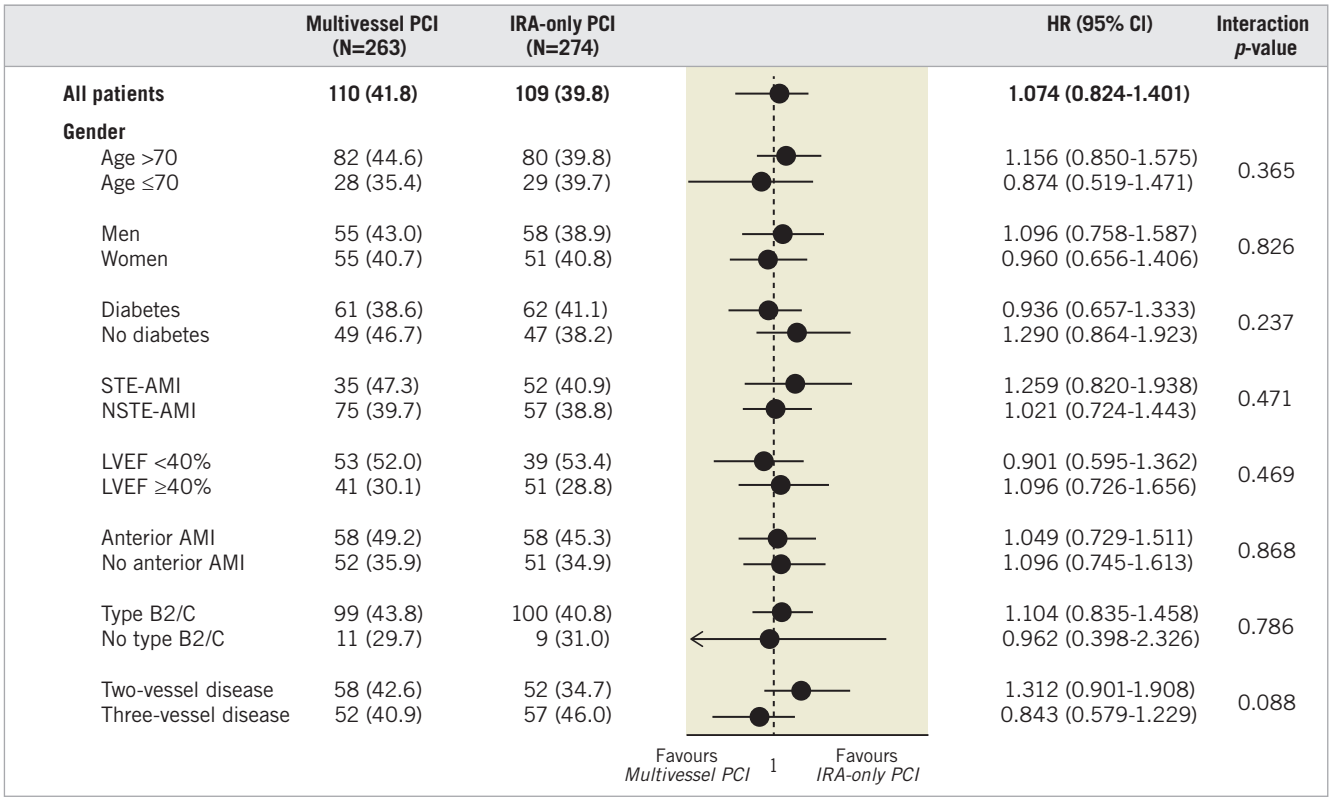

Figure 4. *Exploratory subgroup analysis for MACE. AMI: acute myocardial infarction; CI: confidence interval; HR: hazard ratio; IRA: infarct-related artery; LVEF: left ventricular ejection fraction; MACE: major adverse cardiac events; NSTE-AMI: non-ST-segment elevation acute myocardial infarction; PCI: percutaneous coronary intervention; STE-AMI: ST-segment elevation acute myocardial infarction*

associated with a decreased risk of MACE (adjusted hazard ratio [HR] 1.008, 95% CI: 0.743-1.367; p=0.961) and all-cause death (adjusted HR 0.826, 95% CI: 0.510-1.338; p=0.437) at one year.

Among the 537 patients analysed in the study, 213 had complete estimated GFR data sets at both baseline and the 12-month follow-up. The mean estimated GFR increased by 4.47 mg/dL (95% CI: 2.55-6.40; p<0.001). As regards the safety outcome, the overall incidence of WRF was 17.8% (38/213); the multivessel PCI group showed a significant tendency of having higher event rates of WRF compared with the IRA-only PCI group, even after adjustment for age, body mass index, diabetes, left ventricular ejection fraction, acute kidney injury during index hospitalisation, and type of treatment (multivessel PCI versus IRA-only) (24.8% vs 11.1%, adjusted odds ratio [OR] 2.134, 95% CI: 0.976-4.668; p=0.058).

Discussion

In the present study, we analysed 537 patients with AMI who had MVD and severe RD and underwent PCI in order to evaluate the differential prognostic impact between multivessel PCI and IRA-only PCI. The main findings of this study were as follows: there was no difference of outcomes between multivessel and IRA-only PCI among the population with severe RD at a median follow-up of 12 months. In addition, this finding was consistent even in the multiple sensitivity analyses, including the Cox proportional hazards regression in the propensity score-matched cohort, and even across the various exploratory subgroup analyses.

Based on the results of recent randomised controlled trials[3-6], the Task Force on Myocardial Revascularisation of the European Society of Cardiology issued guidelines stating that one-stage multivessel PCI during STE-AMI without cardiogenic shock should be considered in patients in the presence of multiple, critical stenoses or highly unstable lesions[2]. However, evidence-based studies on the benefits of multivessel PCI in patients with AMI and severe RD are lacking because randomised trials often exclude those with severe RD. Therefore, the findings from these randomised clinical trials cannot be applied to patients with severe RD. As the guideline recommendations already vary according to the presence or absence of cardiogenic shock[2,10,13], further individualisation of the treatment strategy may require adaptation in certain clinical circumstances, especially in patients with high-risk features, such as severe RD.

Theoretically, treatment of the non-IRA may not only lower the risk of further repeat revascularisation, but also improve the clinical outcomes by assisting the recovery of myocardial perfusion and ventricular function. However, our study was not able to determine the better treatment strategy between multivessel PCI and IRA-only PCI for patients with severe RD accompanied by AMI and MVD. The lack of difference between the two treatment strategies in this study might be due to several reasons. Firstly, the higher

dose of contrast medium in the multivessel PCI group may have led to acute left ventricular volume overload with a negative effect on myocardial function and recovery. Secondly, the prolonged duration of the multivessel PCI procedure may be especially hazardous at a time when the patient is haemodynamically compromised, leading to a potentially higher incidence of bleeding and inflammation. Thirdly, additional myocardial damage may have been induced by PCI in stable lesions. Finally, we speculate that the lack of difference between the multivessel and IRA-only PCI might be related to the significantly higher dose of contrast material that was used in the multivessel PCI group and the consequent decline in renal function. Although estimated GFR data at the 12-month follow-up were available only in ~40% of the patients, the overall incidence of WRF tended to be higher in the multivessel PCI group. This may be explained by the potential risk of contrast-induced nephropathy after a greater contrast load in those undergoing multivessel PCI. The CULPRIT-SHOCK trial also showed that, among patients with AMI and cardiogenic shock, the rate of renal replacement therapy tended to be lower in the culprit lesion-only PCI group than in the multivessel PCI group[13]. Because of these hazards, it is possible that the additional benefits of the multivessel intervention at the time of index hospitalisation in these patients might be offset.

Considering the higher incidence of multiple comorbidities and the sequentially higher death rate in the population with RD, however, a high proportion of patients might die earlier before the potential benefit of multivessel PCI is realised. Therefore, further evaluation using biomarkers of renal function, inflammation, and myocardial damage from the central core laboratory, as well as detailed angiographic analyses, could be performed to elucidate the potential underlying mechanisms for these clinical outcomes. Compared to the general population, patients with RD are susceptible to an increased risk of periprocedural complications and are also more likely to have diffuse coronary artery disease that may be challenging for revascularisation. These facts may cause clinicians to consider a conservative management approach instead of revascularisation in patients with RD. Previous data suggest that aggressive therapy is underutilised in patients undergoing dialysis who experience AMI[14].

Study limitations

The present study provides novel insight because it considered exclusively patients with AMI and severe RD using the outcomes of revascularisation performed by a variety of cardiologists in major PCI centres around Korea. However, our results should be viewed in the context of several important limitations. Firstly, although we used various analytical methods, including propensity score matching, to address possible confounding factors, this study has all the limitations of a registry, and residual confounding cannot be excluded. In addition, these data did not capture physician rationale for the selection between multivessel and IRA-only PCI, specifically the multivessel intervention in patients with STE-AMI in an era when multivessel PCI for STE-AMI was assigned a class III recommendation by the ACC/AHA guidelines during the KAMIR-NIH study period. Thus, we cannot draw definitive conclusions. The findings of this study should be considered as hypothesis-generating and warrant prospective evaluation in adequately powered and randomised controlled trials. Secondly, patients who underwent a planned staged revascularisation after discharge could not be identified from the database and, in this study, such patients would be classified into the IRA-only PCI group followed by a repeat revascularisation event. Thirdly, we assessed the lesion severity of the non-IRA patients using angiographic assessment alone. As shown in the DANAMI-3—PRIMULTI and COMPARE-ACUTE trials, nearly one half of visually significant non-IRA lesions were physiologically insignificant, with fractional flow reserve values of >0.80[5,6]. Also, we do not have data on the SYNTAX score or the severity of the non-IRA diameter stenosis, which would be important for the clinical outcomes following complete revascularisation, as shown in the previous study[15]. Fourth, clinical events were not centrally adjudicated in this registry. Fifth, procedure-related risk factors, such as procedure time, total radiation dose, and amount of contrast dye used, were not evaluated. If data regarding the elapsed time and dose of contrast media used during PCI were available, we might be able to understand the relationship between these parameters and the incidence of complications better; however, this information was unavailable. Finally, definition of the type of lesion (culprit versus non-culprit) did not follow a protocol, and it is therefore likely that this decision varied according to how the interventional cardiologist interpreted the angiographic results at the time of coronary angiography. Given how difficult it can be to identify the culprit artery in patients with non-STE-AMI and MVD, it cannot be ruled out that incorrect identification may have influenced the results.

Conclusions

In the present study, there was no difference in the clinical outcomes between multivessel and IRA-only PCI among the population with AMI and severe RD at a median follow-up of 12 months. Rather, multivessel PCI might be related to an increased incidence of renal complications during follow-up. Additional data may be required. Until the results are available, interventional cardiologists have to make their decisions based on the findings of their clinical evaluation of the patients, and taking into account clinical characteristics, disease severity, and lesion complexity.

Impact on daily practice

In the present study, multivessel PCI at the time of primary PCI or within the index hospitalisation showed no difference in clinical outcomes compared with IRA-only revascularisation in patients with AMI and severe RD. Additional data may be required. Until the results are available, interventional cardiologists have to make their decision based on the findings of their clinical evaluation of the patients, and taking into account clinical characteristics, disease severity, and lesion complexity.

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Conflict of interest statement

The authors have no conflicts of interest to declare.

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Supplementary data

Supplementary Appendix 1. Methods.

Supplementary Table 1. Percent standardised differences of variables among the unadjusted and propensity score-matched cohort, and baseline characteristics of the propensity score-matched cohort.

Supplementary Table 2. Baseline clinical characteristics.

Supplementary Table 3. Baseline angiographic and procedural characteristics.

Supplementary Table 4. Sensitivity analysis of baseline adjustment using propensity score matching for 1-year clinical outcomes according to treatment strategy.

Supplementary Table 5. Comparison of 1-year clinical outcomes according to treatment strategy in patients with STE-AMI.

Supplementary Table 6. Comparison of 1-year clinical outcomes according to treatment strategy in patients with non-STE-AMI.

Supplementary Table 7. Independent predictors of MACE or all-cause death.

The supplementary data are published online at: https://eurointervention.pcronline.com/doi/10.4244/EIJ-D-19-00034

Supplementary data

Supplementary Appendix 1. Methods

Definition of acute myocardial infarction (AMI)

1. *ST-segment elevation AMI (STE-AMI).*

Elevation of ST-segment more than 0.1 mV in 2 or more contiguous ECG leads or new left bundle branch block with elevated biomarkers of myocardial necrosis.

2. *Non-ST-segment elevation AMI (Non-STE-AMI).*

Elevated biomarkers of myocardial necrosis (cardiac troponin or creatine kinase-MB isoform >x 1 URL) with one of the following:-

1) Transient ST-segment elevation or depression, or T-wave changes consistent with myocardial ischaemia.
2) Identification of a culprit lesion on coronary angiography.

Statistical analysis

Categorical variables are presented as numbers and relative frequencies (percentages) and are compared using the chi-squared test. Continuous variables are expressed as means±standard deviations or medians (interquartile ranges) according to whether they were normally distributed and were compared using the independent samples t-test or Mann-Whitney test, as appropriate. Cumulative event rates were calculated on the basis of the Kaplan-Meier time-to-event analysis, and the clinical outcomes between the multivessel PCI and IRA-only PCI groups were compared using the log-rank test.

Since differences in the baseline characteristics could significantly affect the outcomes, sensitivity analyses were performed to adjust for confounders as much as possible. First, a multivariable Cox regression model was used. Covariates included in the multivariable model were selected if they were significantly different between the groups or had prognostic values, which were as follows: type of treatment strategy (multivessel percutaneous coronary intervention [PCI] or infarct-related artery [IRA]-only PCI); age; sex; pulse rate at initial presentation; Killip class; chest pain at initial presentation; diabetes mellitus; baseline white blood cell count; left ventricular ejection fraction; left main artery or left anterior descending artery as the culprit vessel; left main coronary artery disease; three-vessel disease; type B2 or C lesion according to the American College of Cardiology/American Heart Association classification; puncture site; use of intravascular imaging devices; stenting; cardiogenic shock, new heart failure, or acute kidney injury during hospitalisation; and use of beta-blockers, renin-angiotensin-aldosterone system blockers, or statins at discharge. The assumption of proportionality was assessed graphically using the log-minus-log plot, and the Cox proportional

hazards models for all clinical endpoints satisfied the proportional hazard assumption. Second, Cox proportional hazards regression in a propensity score-matched population was performed. A multivariable logistic regression model was used to generate the propensity scores, which indicate the probability of the patients being treated with the multivessel PCI strategy. All available covariates were included in this model, exactly following the recommendations of the analysis using the propensity score. For the propensity score matching, a 1:1 matching process without replacements was performed using a greedy algorithm with a calliper width of 0.2 standard deviations, yielding 163 patients in the multivessel PCI group matched with 163 controls in the IRA-only PCI group. Balance between the two groups after propensity score matching was assessed by calculating the percent standardised mean differences in the covariates used in the propensity score generation. The percent standardised mean differences after propensity score matching were within ±10% across all matched covariates, demonstrating successful balance achievement between the groups **(Supplementary Table 1)**.

To identify the independent predictors of major adverse cardiac events and all-cause death at one year, we used the multivariable stratified Cox proportional hazards model. C-statistics with 95% confidence intervals were calculated to validate the discriminant function of the model. In addition, comparisons of the primary endpoint between the multivessel PCI and IRA-only PCI groups according to the subgroups of interest were followed, and the interaction between the treatment effect and these subgroups was evaluated using the Cox regression model. In all analyses, the participating university hospitals were included as random effects.

All probability values were two-sided, and p-values of <0.05 were considered statistically significant. The statistical packages SPSS, Version 22.0 (IBM Corp., Armonk, NY, USA) and SAS version 9.4 (SAS Institute Inc., Cary, NC, USA) were used for the analyses.

Supplementary Table 1. Percent standardised differences of variables among unadjusted and propensity score-matched cohort, and baseline characteristics of propensity score-matched cohort.

	Percent standardised differences		Propensity score-matched cohort			
	Unadjusted (n=537)	PS matched (n=326)	All (n=326)	Multivessel PCI (n=163)	IRA-only PCI (n=163)	*p*-value
Age	-9.80	-2.85	75.4±9.6	75.3±9.4	75.6±9.9	0.791
Sex						
Female	11.95	-4.90	168 (51.5)	82 (50.3)	86 (52.8)	0.656
Vital sign at presentation						
Systolic blood pressure (mmHg)	3.68	-6.69	130.5±32.3	129.4±35.3	131.7±29.0	0.506
Diastolic blood pressure (mmHg)	2.71	-3.90	75.6±17.2	75.3 ±0.713	76.0±17.0	0.713
Heart rate (frequency/min)	13.90	-1.96	85.3±23.6	85.1±23.8	85.5±23.5	0.875
Body mass index (kg/m^2)	29.17	4.57	22.2±3.4	22.2±3.4	22.1±3.4	0.624
Diabetes mellitus	9.05	1.25	173 (53.1)	87 (53.4)	86 (52.8)	0.913
Hypertension	16.66	-5.04	271 (83.1)	134 (82.2)	137 (84.0)	0.663
Dyslipidaemia	8.28	3.83	32 (9.8)	17 (10.4)	15 (9.2)	0.696
Previous myocardial infarction	1.28	3.95	30 (9.2)	16 (9.8)	14 (8.6)	0.707
Previous cerebrovascular accident	-21.00	-5.84	43 (13.2)	20 (12.3)	23 (14.1)	0.614
Current smoker	-9.37	0.000	46 (14.1)	23 (14.1)	23 (14.1)	>0.99
Killip classification						0.961
1			157 (48.2)	78 (47.9)	79 (48.5)	
2	5.63	-1.65	53 (16.3)	26 (16.0)	27 (16.6)	
3	7.26	-1.41	83 (25.5)	41 (25.2)	42 (25.8)	
4	4.76	5.59	33 (10.1)	18 (11.0)	15 (9.2)	
Acute myocardial infarction						
NSTEMI	8.93	9.95	204 (62.6)	106 (65.0)	98 (60.1)	0.334
Laboratory findings						
WBC (10^3/L)	-1.09	2.24	11,070±5,129	11,130±5,410	11,000±4,750	0.831
Haemoglobin (g/dL)	-12.10	-0.85	10.9±1.8	10.9±1.5	10.9±1.9	0.940
eGFR (mL/min/1.73 m^2)	-9.62	-4.80	19.1±7.3	19.0±7.5	19.4±7.2	0.660
Glucose (mg/dL)	-3.55	-1.25	204.6±122.2	203.9±106.4	205.3±136.5	0.918
CK-MB (ng/mL)	-5.46	-2.39	73.9±106.8	72.0±103.1	75.8±110.6	0.749
Troponin-I (ng/mL)	5.92	-4.63	42.4±83.4	39.0±81.0	45.8±86.0	0.461
Total cholesterol (mg/dL)	2.52	-8.45	159.8±51.5	157.8±45.7	161.8±56.8	0.480
LDL-C (mg/dL)	2.36	0.39	94.0±36.4	94.0±36.5	93.9±36.5	0.972
Echocardiography						
LVEF (%)	-18.62	2.68	44.5±12.0	44.6±12.2	44.3±11.8	0.803

Medications at discharge						
Statin	4.18	1.46	249 (76.4)	125 (76.7)	124 (76.1)	0.897
Potent $P2Y_{12}$ inhibitors	-3.00	9.47	64 (19.6)	35 (21.5)	29 (17.8)	0.345
Approach						
Transfemoral	0.97	-1.54	257 (78.8)	128 (78.5)	129 (791.)	0.895
Culprit						0.645
Left anterior descending	7.55	8.09	139 (42.6)	66 (40.5)	73 (44.8)	
Left circumflex	0.64	2.52	59 (18.1)	32 (19.6)	27 (16.6)	
Right coronary artery	43.81	0.000	128 (39.3)	65 (39.9)	63 (38.7)	
Three-vessel disease	5.04	6.13	149 (45.7)	77 (47.2)	72 (44.2)	0.589
Lesion type B2/C	-8.97	5.32	281 (86.2)	142 (87.1)	139 (85.3)	0.623
Preprocedural TIMI						0.970
1	10.73	1.44	49 (15.0)	23 (14.1)	26 (16.0)	
2	17.68	2.67	65 (19.9)	33 (20.2)	32 (19.6)	
3	-1.61	6.13	92 (28.2)	47 (28.8)	45 (27.6)	
Intravascular ultrasound	14.65	1.51	59 (18.1)	30 (18.4)	29 (17.8)	0.885
Glycoprotein IIb/IIIa inhibitor	-3.51	2.48	25 (7.7)	13 (8.0)	12 (7.4)	0.836
Thrombus aspiration	-42.24	-8.46	44 (13.5)	20 (12.3)	24 (14.7)	0.481

CK-MB: creatine kinase-myocardial band isoform; eGFR: estimated glomerular filtration rate; IRA: infarct-related artery; LDL-C: low-density lipoprotein cholesterol; LVEF: left ventricular ejection fraction; NSTEMI: non-ST-segment elevation myocardial infarction; PCI: percutaneous coronary intervention; PS: propensity score; TIMI: Thrombolysis In Myocardial Infarction; WBC: white blood cell

Potent $P2Y_{12}$ inhibitors: prasugrel or ticagrelor.

Supplementary Table 2. Baseline clinical characteristics.

	Overall population (n=537)	**Multivessel PCI (n=263)**	**IRA-only PCI (n=274)**	***p*-value**
Age, years	74.9±10.2	74.3±10.0	75.5±10.4	0.179
Sex (male)	277 (51.6)	135 (51.3)	125 (45.6)	0.186
Vital sign at presentation				
Systolic blood pressure (mmHg)	128.5±34.1	129.4±36.3	127.6±32.0	0.550
Diastolic blood pressure (mmHg)	74.2±19.2	74.6±19.3	73.8±19.1	0.666
Heart rate (frequency/min)	84.6±24.0	86.2±22.9	83.1±25.0	0.132
Body mass index (kg/m^2)	22.1±3.4	22.6±3.6	21.5±3.2	<0.001
Diabetes mellitus	309 (57.5)	158 (60.1)	151 (55.1)	0.244
Hypertension	433 (80.6)	220 (83.7)	213 (77.7)	0.083
Dyslipidaemia	54 (10.1)	30 (11.4)	24 (8.8)	0.308
Previous myocardial infarction	57 (10.6)	28 (10.6)	29 (10.6)	0.981
Previous PCI	96 (17.9)	48 (18.3)	48 (17.5)	0.825
Previous cerebrovascular accident	78 (14.5)	30 (11.4)	48 (17.5)	0.045
Smoking				0.196
Current	81 (15.7)	35 (13.8)	46 (17.4)	
Ex	115 (22.2)	51 (20.2)	64 (24.2)	
Never	321 (62.1)	167 (66.0)	154 (58.3)	
Killip classification				0.507
1	263 (49.0)	120 (45.6)	143 (52.2)	
2	82 (15.3)	43 (16.3)	39 (14.2)	
3	124 (23.1)	65 (24.7)	59 (21.5)	
4	68 (12.7)	35 (13.3)	33 (12.0)	
Killip classification ≥2	274 (51.0)	143 (54.4)	131 (47.8)	0.128
Acute myocardial infarction				<0.001
STEMI	201 (37.4)	74 (28.1)	127 (46.4)	
NSTEMI	336 (62.6)	189 (71.9)	147 (53.6)	
Laboratory findings				
WBC (10^3/L)	11,228±5,105	11,171±5,546	11,283±4,652	0.800
Haemoglobin (g/dL)	10.9±1.8	10.8±1.7	11.0±1.9	0.249
Creatinine (mg/dL)	3.37±2.90	3.61±3.10	3.13±2.68	0.056

eGFR (mL/min/1.73 m^2)	19.1±7.5	18.7±7.8	19.5±7.2	0.196
Glucose (mg/dL)	213.4±127.4	210.6±113.8	216.1±139.5	0.622
HbA1C (%)	6.73±1.62	6.69±1.47	6.78±1.76	0.619
CK-MB (ng/mL)	88.1±163.5	86.9±196.0	89.2±125.0	0.870
Troponin-I (ng/mL)	53.0±136.7	59.0±173.8	46.8±81.6	0.334
Total cholesterol (mg/dL)	158.8±50.7	159.5±48.3	158.1±53.1	0.758
LDL-C (mg/dL)	94.1±39.9	94.7±40.6	93.6±39.3	0.767
hs-CRP (mg/L, n=332)	3.99±11.66	3.30±5.32	4.60±15.17	0.309
NT-proBNP (pg/mL, n=338)	15,000±24,369	13,140±12,082	16,575±31,155	0.197
Echocardiography				
Left ventricular ejection fraction (%)	44.9±12.9	43.4±13.5	46.3±12.1	0.012
Mitral regurgitation 3, 4	20 (4.4)	13 (6.1)	7 (2.9)	0.102
Vital sign at discharge				
Systolic blood pressure (mmHg)	116.5±25.9	116.8±24.6	116.2±27.1	0.781
Diastolic blood pressure (mmHg)	66.3±14.6	65.7±14.2	66.8±15.0	0.446
Heart rate (frequency/min)	71.7±16.4	73.1±16.6	70.4±16.1	0.081
Medications at discharge				
Aspirin	536 (99.8)	263 (100.0)	273 (99.6)	>0.99
Clopidogrel	469 (87.3)	233 (88.6)	236 (86.1)	0.391
Prasugrel	22 (4.1)	12 (4.4)	10 (3.8)	0.736
Ticagrelor	81 (15.1)	39 (14.8)	42 (15.3)	0.872
Cilostazol	80 (14.9)	50 (19.0)	30 (10.9)	0.009
ACEI or ARB	348 (64.8)	165 (62.7)	183 (66.8)	0.326
Beta-blocker	379 (70.6)	186 (70.7)	193 (70.4)	0.942
Statin	409 (76.2)	202 (76.8)	207 (75.5)	0.732
Oral anticoagulant	18 (3.4)	4 (1.5)	14 (5.1)	0.029

ACEI: angiotensin-converting enzyme inhibitor; ARB: angiotensin II receptor blocker; CK-MB: creatine kinase myocardial band isoform; eGFR: estimated glomerular filtration rate; HbA1C: haemoglobin A1C; hs-CRP: high-sensitivity C-reactive protein; IRA: infarct-related artery; LDL-C: low-density lipoprotein cholesterol; NSTEMI: non-ST-segment elevation myocardial infarction; NT-proBNP: N-terminal pro B-type natriuretic peptide; PCI: percutaneous coronary intervention; STEMI: ST-segment elevation myocardial infarction; TIMI: Thrombolysis In Myocardial Infarction; WBC: white blood cell

Supplementary Table 3. **Baseline angiographic and procedural characteristics.**

	Overall population (n=537)	**Multivessel PCI (n=263)**	**IRA-only PCI (n=274)**	***p*-value**
Approach				0.547
Transradial	105 (19.6)	51 (19.4)	54 (19.7)	
Transfemoral	432 (80.4)	212 (80.6)	220 (80.3)	
Culprit				<0.001
Left anterior descending	202 (37.6)	74 (28.1)	128 (46.7)	
Left circumflex	85 (15.8)	45 (17.1)	40 (14.6)	
Right coronary artery	206 (38.4)	100 (38.0)	106 (38.7)	
Left main	44 (8.2)	44 (16.7)	0 (0.0)	
Three-vessel disease	251 (46.7)	127 (48.3)	124 (45.3)	0.481
Lesion type B2/C	471 (87.7)	226 (85.9)	245 (89.4)	0.219
Preprocedural TIMI 2, 3	268 (49.9)	140 (53.2)	128 (46.7)	0.131
Timing of non-IRA PCI				
Immediate PCI		205 (77.9)		
Staged		58 (22.1)		
Intravascular ultrasound	94 (17.5)	54 (20.5)	40 (14.6)	0.070
Glycoprotein IIb/IIIa inhibitor	38 (7.1)	18 (6.8)	20 (7.3)	0.837
Thrombus aspiration	85 (15.8)	25 (9.5)	60 (21.9)	<0.001
Stenting	486 (90.5)	227 (86.3)	259 (94.5)	0.001
Post-procedural TIMI 2, 3	527 (98.1)	256 (97.3)	271 (98.9)	0.214
Successful PCI	521 (97.0)	253 (96.2)	268 (97.8)	0.143
Completeness of multivessel PCI				
Complete revascularisation		149 (56.7)		
Incomplete revascularisation		114 (43.3)		
Closure device	162 (30.2)	75 (28.5)	87 (31.8)	0.414

IRA: infarct-related artery; PCI: percutaneous coronary intervention; TIMI: Thrombolysis In Myocardial Infarction

Supplementary Table 4. Sensitivity analysis of baseline adjustment using propensity score matching for 1-year clinical outcomes according to treatment strategy.

	Overall (n=326)	**Multivessel PCI (n=163)**	**IRA-only PCI (n=163)**	***p*-value**
All-cause death	77 (23.6)	34 (20.9)	43 (26.4)	0.297
Reinfarction	14 (4.3)	5 (3.1)	9 (5.5)	0.283
All-cause death or reinfarction	87 (26.7)	38 (23.3)	49 (30.1)	0.218
Re-hospitalisation for heart failure	31 (9.5)	18 (11.0)	13 (8.0)	0.328
Any repeat revascularisation	25 (7.7)	11 (6.7)	14 (8.6)	0.691
Non-IRA repeat revascularisation	14 (4.3)	8 (4.9)	6 (3.7)	0.586
Definite or probable stent thrombosis	3 (0.9)	2 (1.2)	1 (0.6)	0.570
Major adverse cardiac events	124 (38.0)	60 (36.8)	64 (39.3)	0.774

IRA: infarct-related artery; PCI: percutaneous coronary intervention

Major adverse cardiac events: a composite of all-cause death, reinfarction, re-hospitalisation for heart failure, and any repeat revascularisation

Supplementary Table 5. Comparison of 1-year clinical outcomes according to treatment strategy in patients with STE-AMI.

	Overall (n=201)	Multivessel PCI (n=74)	IRA-only PCI (n=127)	Unadjusted in overall population		Adjusted in overall population	
				HR (95% CI)	*p*-value	HR (95% CI)	*p*-value
MACE	87 (43.3)	35 (47.3)	52 (40.9)	1.215 (0.791-1.865)	0.374	1.903 (1.123-3.223)	0.017
All-cause death	66 (32.8)	28 (37.8)	38 (29.9)	1.303 (0.799-2.122)	0.289	1.184 (0.587-2.387)	0.637
Reinfarction	8 (4.0)	4 (5.4)	4 (3.1)	1.740 (0.435-6.958)	0.433	1.549 (0.385-6.227)	0.538
Re-hospitalisation for heart failure	9 (4.5)	2 (2.7)	7 (5.5)	0.483 (0.100-2.325)	0.364	0.432 (0.090-2.080)	0.295
Any repeat revascularisation	14 (7.0)	6 (8.1)	8 (6.3)	1.309 (0.454-3.772)	0.618	1.242 (0.430-3.592)	0.689
Non-IRA repeat revascularisation	7 (3.5)	4 (5.4)	3 (2.4)	2.319 (0.519-10.361)	0.271	2.326 (0.521-10.394)	0.269

CI: confidence interval; HR: hazard ratio; IRA: infarct-related artery; PCI: percutaneous coronary intervention; STE-AMI: ST-segment elevation acute myocardial infarction

Major adverse cardiac events (MACE): a composite of all-cause death, reinfarction, re-hospitalisation for heart failure, and any repeat revascularisation

Supplementary Table 6. Comparison of 1-year clinical outcomes according to treatment strategy in patients with non-STE-AMI.

	Overall (n=336)	Multivessel PCI (n=189)	IRA-only PCI (n=147)	Unadjusted in overall population		Adjusted in overall population	
				HR (95% CI)	*p*-value	HR (95% CI)	*p*-value
MACE	132 (39.3)	75 (39.7)	57 (38.8)	1.005 (0.712-1.418)	0.977	1.118 (0.776-1.611)	0.551
All-cause death	82 (24.4)	43 (22.8)	39 (26.5)	0.851 (0.552-1.313)	0.465	0.748 (0.464-1.206)	0.234
Reinfarction	19 (5.7)	8 (4.2)	11 (7.5)	0.555 (0.223-1.381)	0.206	0.451 (0.180-1.133)	0.090
Re-hospitalisation for heart failure	34 (10.1)	23 (12.2)	11 (7.5)	1.677 (0.817-3.440)	0.159	1.717 (0.836-3.526)	0.141
Any repeat revascularisation	24 (7.1)	12 (6.3)	12 (8.2)	0.771 (0.346-1.716)	0.524	0.734 (0.329-1.640)	0.451
Non-IRA repeat revascularisation	12 (3.6)	6 (3.2)	6 (4.1)	0.775 (0.250-2.401)	0.658	0.611 (0.190-1.967)	0.408

CI: confidence interval; HR: hazard ratio; IRA: infarct-related artery; PCI: percutaneous coronary intervention; STE-AMI: ST-segment elevation acute myocardial infarction

Major adverse cardiac events (MACE): a composite of all-cause death, reinfarction, re-hospitalisation for heart failure, and any repeat revascularisation

Supplementary Table 7. Independent predictors of MACE or all-cause death.

	Median (IQR) or number (%)	Wald chi-square	Hazard ratio (95% confidence interval)	*p*-value
Major adverse cardiac events				
Diastolic blood pressure at discharge	70.0 (60.0-75.5)	14.907	0.973 (0.960-0.987)	<0.001
Chest pain at presentation	341 (63.5)	5.178	0.678 (0.485-0.948)	0.023
Discharge medications				
RAA blockers	348 (64.8)	4.716	0.683 (0.485-0.964)	0.030
Statin	409 (76.2)	4.481	0.656 (0. 444-0.969)	0.034
Pulse rate at presentation	84.0 (70.0-101.0)	4.089	1.008 (1.000-1.016)	0.043
Left ventricular ejection fraction (%)	45.0 (35.3-55.0)	3.965	0.986 (0.973-1.000)	0.046
Stenting	486 (90.5)	3.778	0.605 (0.364-1.004)	0.052
Left main artery or LAD as culprit vessel	246 (45.8)	3.230	1.359 (0.973-1.900)	0.072
All-cause death				
Cardiogenic shock during index hospitalisation	106 (19.7)	23.588	2.890 (1.883-4.435)	<0.001
Discharge medication, statin	409 (76.2)	12.472	0.481 (0.320-0.722)	<0.001
Left ventricular ejection fraction (%)	45.0 (35.3-55.0)	12.324	0.973 (0.958-0.988)	<0.001
Age	77.0 (69.0-82.0)	5.807	1.024 (1.004-1.044)	0.016
Discharge medication, RAA blockers	348 (64.8)	5.219	0.631 (0.425-0.937)	0.022
Preprocedural TIMI 2 or 3	268 (49.9)	3.331	0.684 (0.455-1.028)	0.068
Non-STE-AMI	336 (62.6)	3.119	0.678 (0.441-1.044)	0.077

IRA: infarct-related artery; IQR: interquartile range; LAD: left anterior descending; NSTEMI: non-ST-segment elevation myocardial infarction; PCI: percutaneous coronary intervention; RAA: renin-angiotensin-aldosterone; TIMI: Thrombolysis In Myocardial Infarction

MACE (major adverse cardiac events): a composite of all-cause death, reinfarction, re-hospitalisation for heart failure, and any repeat revascularisation