



Complete or incomplete revascularization in patients with left main culprit lesion acute myocardial infarction with multivessel disease: a retrospective observational study

Sun Oh Kim¹, Hong-Ju Kim¹, Jong-Il Park¹, Kang-Un Choi¹, Jong-Ho Nam¹, Chan-Hee Lee¹, Jang-Won Son¹, Jong-Seon Park¹, Sung-Ho Her², Ki-Yuk Chang³, Tae-Hoon Ahn⁴, Myung-Ho Jeong⁵, Seung-Woon Rha⁶, Hyo-Soo Kim⁷, Hyeon-Cheol Gwon⁸, In-Wan Seong⁹, Kyung-Kuk Hwang¹⁰, Seung-Ho Hur¹¹, Kwang-Soo Cha¹², Seok-Kyu Oh¹³, Jei-Keon Chae¹⁴, Ung Kim¹

For further information on the authors' affiliations, see [Additional information](#).

Background: Complete revascularization has demonstrated better outcomes in patients with acute myocardial infarction (AMI) and multivessel disease. However, in the case of left main (LM) culprit lesion AMI with multivessel disease, there is limited evidence to suggest that complete revascularization is better.

Methods: We reviewed 16,831 patients in the Korea Acute Myocardial Infarction Registry who were treated from July 2016 to June 2020, and 399 patients were enrolled with LM culprit lesion AMI treated with percutaneous coronary intervention. We categorized the patients as those treated with complete revascularization (n = 295) or incomplete revascularization (n = 104). The study endpoint was major adverse cardiac and cerebrovascular events (MACCE), a composite of all-cause death, myocardial infarction, ischemia-driven revascularization, stent thrombosis, and stroke. We performed propensity score matching (PSM) and analyzed the incidence of MACCE at 1 year.

Results: After PSM, the two groups were well balanced. There was no significant difference between the two groups in MACCE at 1 year (12.1% vs. 15.2%; hazard ratio, 1.28; 95% confidence interval, 0.60–2.74; $p = 0.524$) after PSM. The components of MACCE and major bleeding were also not significantly different.

Conclusion: There was no significant difference in clinical outcomes between the groups treated with complete or incomplete revascularization for LM culprit lesion AMI with multivessel disease.

Keywords: Left main disease; Multivessel disease; Myocardial infarction; Percutaneous coronary revascularization

Introduction

In the era of drug-eluting stents, percutaneous coronary intervention (PCI) has become the primary treatment for acute myocardial infarction (AMI) [1-3]. A significant proportion of patients with

AMI present with multivessel disease [4-6]. Recent studies have suggested that complete revascularization in these cases, which addresses the culprit and non-culprit lesions, is superior to revascularization of the culprit lesion alone [7-10].

However, patients with AMI and the left main (LM) coronary

Received: November 5, 2024 • Revised: December 3, 2024 • Accepted: December 7, 2024 • Published online: December 19, 2024

Corresponding author: Ung Kim, MD, PhD

Division of Cardiology, Department of Internal Medicine, Yeungnam University Medical Center, 170 Hyeonchung-ro, Nam-gu, Daegu 42415, Korea

Tel: +82-53-620-3313 • E-mail: woongwa@yu.ac.kr

© 2025 Yeungnam University College of Medicine, Yeungnam University Institute of Medical Science

This is an Open Access article distributed under the terms of the Creative Commons Attribution Non-Commercial License (<https://creativecommons.org/licenses/by-nc/4.0/>) which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

artery as the culprit lesion, despite their lower prevalence, exhibit higher mortality rates than patients with AMI and other vessels as the culprit lesion [6,11-13]. In cases of multivessel disease, there is a high risk of cardiogenic shock [14-16]. Consequently, most studies, designed with “stable” patients in mind, either exclude cases where the LM coronary artery is the culprit lesion or include only a minimal number of such patients [7,9,10]. As a result, there is limited evidence to support the superiority of complete revascularization in LM culprit lesion AMI.

This study aimed to evaluate whether complete revascularization leads to improved clinical outcomes compared to incomplete revascularization in patients with LM culprit lesion AMI and multivessel disease.

Methods

Ethics statement: The Institutional Review Board (IRB) of each center approved the study protocol, and the study was performed in accordance with the Declaration of Helsinki. This study was approved by the IRB of Yeungnam University Hospital (IRB No: 2016-03-017 KAMIR-V). Written informed consent was obtained from all study participants.

1. Study design and population

The Korea Acute Myocardial Infarction Registry (KAMIR) is a nationwide, prospective observational multicenter registry of Korean patients with AMI. Between July 2016 and June 2020, 16,831 patients from 50 centers were registered. All the participating centers were highly experienced in primary PCI and used a common protocol. After registration, clinical follow-up was performed 6 months after discharge, and annual follow-up was performed for up to 3 years. Specialized research coordinators collected data in a structured format, and the KAMIR Steering Committee defined all variables. Data were obtained from this registry.

A flowchart of the study is shown in Fig. 1. A total of 595 patients who had LM culprit lesion AMI and were treated with PCI using a drug-eluting stent. Four patients with missing data and 112 patients without multivessel disease were excluded. Among the remaining patients, 80 had cardiogenic shock that required mechanical support, such as extracorporeal membrane oxygenation (ECMO) and/or intra-aortic balloon pump (IABP), or underwent cardiopulmonary resuscitation (CPR). To minimize bias, these patients were excluded because they would have been treated in urgent situations, which might have influenced the choice of treatment. The remaining 399 patients with multivessel disease were categorized into the incomplete revascularization group and

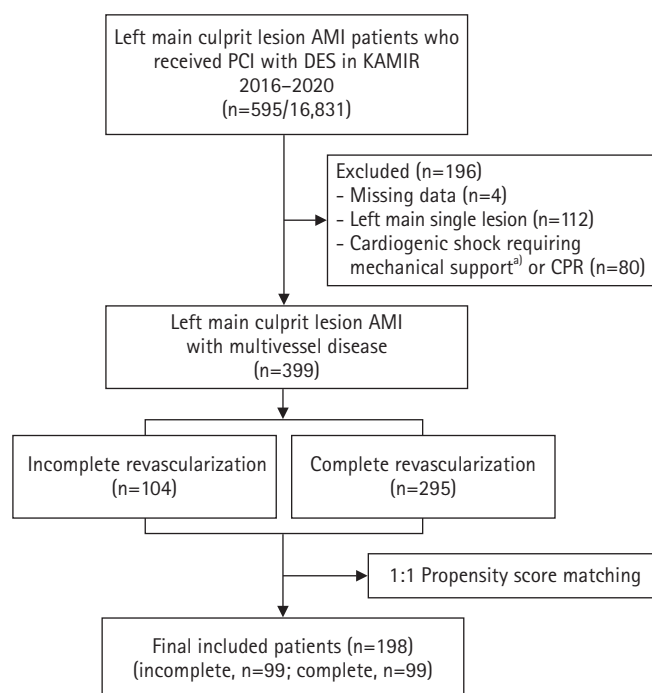


Fig. 1. Study design and population. AMI, acute myocardial infarction; PCI, percutaneous coronary intervention; DES, drug-eluting stent; KAMIR, Korea Acute Myocardial Infarction Registry; CPR, cardiopulmonary resuscitation. ^{a)}Extracorporeal membrane oxygenation and/or intra-aortic balloon pump.

complete revascularization groups. The 104 patients who received treatment for the LM coronary artery but not for all other non-culprit lesions were classified into the incomplete revascularization group. Subsequently, 295 patients who were treated for both the LM coronary artery and all the non-culprit lesions were assigned to the complete revascularization group.

2. Procedure and medical treatment

Coronary angiography (CAG) and PCI were performed using standard techniques. During the procedure, treatment strategies such as pre-balloon dilation, direct stent implantation, post-balloon dilation, imaging-guided PCI (e.g., intravascular ultrasound or optical coherence tomography), and administration of glycoprotein IIb/IIIa inhibitors were left to the discretion of each operator. Medications were prescribed using standardized regimens. All patients were administered a loading dose of aspirin 200 mg orally and a loading dose of P2Y₁₂ inhibitor (clopidogrel 300 mg or 600 mg, ticagrelor 180 mg, or prasugrel 60 mg) before or during the procedure, and dual antiplatelet therapy (aspirin 100 mg/day and P2Y₁₂ inhibitor: clopidogrel 75 mg/day, ticagrelor 180 mg/day, or prasugrel 10 mg/day) was administered after the procedure for at least 1 year. All other medications were at the discretion of each physician.

3. Study endpoints and definitions

The primary endpoint was a composite of major adverse cardiac and cerebrovascular events (MACCE: all-cause death, myocardial infarction [MI], ischemia-driven revascularization, stent thrombosis, and stroke) at 1 year.

All-cause death was classified as cardiac death if non-cardiac causes could not be determined. Cardiac death was defined as death due to proximate cardiac causes (MI, pump failure, fatal arrhythmia, unwitnessed death, or all procedure-related deaths, including those related to concomitant treatment). Non-cardiac death was defined as death due to non-coronary vascular causes (cerebrovascular disease, pulmonary embolism, ruptured aortic aneurysm, dissecting aneurysm, or other vascular diseases) or non-cardiovascular causes (infection, malignancy, sepsis, pulmonary causes, accident, suicide, or trauma) [17]. MI was defined as having an elevated creatine kinase-myocardial band (CK-MB), troponin I, or troponin T level above the normal range, and typical ischemic symptoms combined with newly developed ischemic electrocardiogram (ECG) changes. Ischemia-driven revascularization was defined as a composite of all target and non-target revascularization via repeat PCI or coronary artery bypass graft surgery. Stent thrombosis was defined using Academic Research Consortium definitions [18]. Stroke was defined as cerebrovascular disease causing a prolonged neurological deficit for at least 24 hours or death.

The secondary endpoints were the individual components of the primary endpoint and major bleeding at 1 year. Major bleeding was defined as types 3 to 5 bleeding according to the Bleeding Academic Research Consortium [19].

A non-culprit lesion was defined as an epicardial coronary artery that was not related to an infarct, was not an extended lesion from the LM coronary artery, and had at least 70% stenosis of the vessel diameter, as visually estimated on CAG. In the complete revascularization group, treatment of the non-culprit epicardial coronary arteries was performed immediately during the index PCI or as a staged PCI during hospitalization.

AMI was defined as elevation above the 99th percentile upper reference limit of at least one cardiac enzymes such as CK-MB, troponin I, and troponin T and having at least one of the following (ischemic symptoms, ischemic ECG changes, developing pathological Q waves, or newly developed regional wall motion abnormality, which was compatible with the coronary territory ischemia observed on the echocardiography) [20]. ST-elevation myocardial infarction (STEMI) was defined as ≥ 2 mm ST segment elevation on two contiguous precordial leads or ≥ 1 mm ST segment elevation on at least two limb leads on ECG. All other ECG findings were defined as non-STEMI.

Cardiogenic shock was defined as sustained hypotension with systolic blood pressure < 90 mmHg for ≥ 30 minutes.

4. Statistical analysis

Categorical variables are reported as numbers and percentages. Continuous variables are reported as mean \pm standard deviation or median values with interquartile ranges (IQR, Q1–Q3). Comparisons of categorical variables between the two groups were performed using either Pearson chi-square test or Fisher exact test, and comparisons of continuous variables were performed using either Student *t*-test or the Mann-Whitney U-test. A 1:1 propensity score matching (PSM) was performed to reduce the confounding factors of baseline and procedural characteristics based on significant variables ($p < 0.05$). To compare outcomes between the groups, the cumulative incidence of primary and secondary endpoints was estimated using the Kaplan-Meier method, and the curves were compared using the log-rank test. Cox proportional hazards regression analysis was performed to identify potential risk factors (baseline, clinical, and procedural variables) for the primary endpoint. Variables with $p < 0.20$ in the univariate analysis were included in the multivariate analysis. Statistical significance was set at $p < 0.05$. All analyses were performed using R software version 4.3.0 (The R Foundation for Statistical Computing, Vienna, Austria).

Results

1. Characteristics of the study population

Most baseline characteristics did not show any significant differences between the two groups; however, a few characteristics exhibited significant differences (Table 1). In the incomplete revascularization group, the patients were older (69.8 ± 10.4 vs. 67.2 ± 10.7 years, $p = 0.027$), and there was a higher proportion of hypertensive patients (63.5% vs. 48.5%, $p = 0.012$). The proportions of clinical presentation were similar between the two groups (STEMI, 32.7% vs. 31.9%; $p > 0.999$) and Killip classification (≥ 2 , 23.7% vs. 25.9%; $p = 0.769$). However, the left ventricular ejection fraction (LVEF) was significantly lower in the incomplete revascularization group (48.5% [IQR, 39.0%–57.9%] vs. 52.0% [IQR, 45.9%–59.0%], $p = 0.030$). Laboratory findings and medications administered at discharge were not significantly different between the two groups.

Among the procedural characteristics (Table 2), a significant difference was observed in the number of non-culprit vessels per patient between the two groups. The complete revascularization group had a higher proportion of single non-culprit lesions (10.6% vs. 44.7%, $p < 0.001$). Regarding the location of the non-culprit lesion, the number of left circumflex (LCX) and right coronary ar-

Table 1. The baseline and clinical characteristics of the included patients

Characteristic	Before PSM group			PSM group		
	Incomplete (n = 104)	Complete (n = 295)	p-value	Incomplete (n = 99)	Complete (n = 99)	p-value
Age (yr)	69.8 ± 10.4	67.2 ± 10.7	0.027	69.1 ± 10.2	67.8 ± 9.6	0.329
Male sex	81 (77.9)	226 (77.6)	>0.999	78 (78.8)	77 (77.8)	>0.999
Hypertension	66 (63.5)	143 (48.5)	0.012	61 (61.6)	52 (52.5)	0.251
Diabetes mellitus	40 (38.5)	112 (38.0)	>0.999	39 (39.4)	41 (41.4)	0.885
Dyslipidemia	17 (16.3)	36 (12.2)	0.367	17 (17.2)	11 (11.1)	0.308
Atrial fibrillation	9 (8.7)	14 (4.7)	0.220	9 (9.1)	4 (4.0)	0.251
Previous MI	10 (9.6)	25 (8.5)	0.879	10 (10.1)	8 (8.1)	0.805
Previous PCI	15 (14.4)	37 (12.5)	0.749	15 (15.2)	12 (12.1)	0.679
Previous heart failure	3 (2.9)	6 (2.0)	0.702	2 (2.0)	5 (5.1)	0.445
Previous stroke	13 (12.5)	19 (6.4)	0.081	12 (12.1)	9 (9.1)	0.644
Current smoker	25 (24.0)	90 (30.5)	0.260	24 (24.2)	23 (23.2)	>0.999
Clinical presentation			0.973			>0.999
Non-STEMI	70 (67.3)	201 (68.1)		67 (67.7)	68 (68.7)	
STEMI	34 (32.7)	94 (31.9)		32 (32.3)	31 (31.3)	
Killip class ≥ 2 at admission	23 (22.1)	70 (23.7)	0.769	21 (21.2)	26 (26.3)	0.369
LVEF (%)	48.5 (39.0–57.9)	52.0 (45.9–59.0)	0.030	51 (39.2–58.0)	52.4 (44.0–60.0)	0.197
Glycated hemoglobin (%)	6.0 (5.5–7.2)	6.1 (5.6–6.9)	0.939	6.0 (5.5–7.2)	6.0 (5.6–7.3)	0.931
LDL-C (mg/dL)	103.0 (77.0–136.0)	103.5 (76.0–138.0)	0.873	106.5 ± 40.2	102.4 ± 36.8	0.496
Peak creatinine (mg/dL)	1.1 (0.9–1.6)	1.0 (0.9–1.3)	0.073	1.1 (0.9–1.6)	1.0 (0.9–1.5)	0.601
Medication at discharge						
Aspirin	103 (99.0)	289 (98.0)	0.971	98 (99.0)	96 (97.0)	0.996
P2Y ₁₂ inhibitor	103 (99.0)	295 (100)	>0.999	98 (99.0)	96 (97.0)	0.996
Clopidogrel	71 (68.3)	161 (54.6)	0.022	66 (66.7)	60 (60.6)	0.514
Prasugrel	8 (7.7)	24 (8.1)	>0.999	8 (8.1)	8 (8.1)	>0.999
Ticagrelor	24 (24.2)	110 (37.3)	0.021	24 (24.2)	28 (28.3)	0.430
Beta-blocker	86 (82.7)	219 (74.2)	0.126	82 (82.8)	74 (74.7)	0.267
ACE inhibitor or ARB	77 (77.8)	207 (70.2)	0.596	72 (72.7)	73 (73.7)	0.903
Statin	95 (96.0)	276 (93.6)	0.387	91 (91.9)	92 (92.9)	0.780
Ezetimibe	11 (10.6)	38 (12.9)	0.633	10 (10.1)	15 (15.2)	0.377
OAC	3 (2.9)	17 (5.8)	0.360	3 (3.0)	5 (5.1)	0.495
SGLT2 inhibitor	6 (5.8)	14 (4.7)	0.887	6 (6.1)	2 (2.0)	0.279

Values are presented as mean ± standard deviation, number (%), or median (interquartile range).

PSM, propensity score matching; MI, myocardial infarction; PCI, percutaneous coronary intervention; STEMI, ST-elevation myocardial infarction; LVEF, left ventricular ejection fraction; LDL-C, low-density lipoprotein cholesterol; ACE, angiotensin-converting enzyme; ARB, angiotensin receptor blocker; OAC, oral anticoagulant; SGLT2, sodium-glucose cotransporter 2.

$p < 0.05$, significant difference.

tery (RCA) lesions was higher in the incomplete revascularization group (LCX, 73.1% vs. 52.5%, $p < 0.001$; RCA, 86.5% vs. 36.9%, $p < 0.001$). Staged PCI was performed in 63 patients (21.4 %) in the complete revascularization group.

PSM was performed based on age, presence of hypertension, LVEF, and number of non-culprit vessels per patient. After PSM, the two groups were well balanced, except for the number of non-culprit RCA lesions (85 [85.9%] vs. 49 [49.5%], $p < 0.001$).

2. Primary and secondary endpoints

The outcome comparisons between the incomplete and complete revascularization groups at 1-year follow-up are shown in Table 3 and Fig. 2. The cumulative incidence of the primary endpoint

(MACCE) showed no significant difference between the two groups (12 [12.1%] vs. 15 [15.2%]; hazard ratio [HR], 1.28; 95% confidence interval [CI], 0.60–2.74; $p = 0.524$) (Fig. 2). For the secondary endpoints, each MACCE component and the incidence of major bleeding were similar between the two groups (Fig. 3). Unexpectedly, although not significant, stroke occurred more frequently in the complete revascularization group (1 [1.0%] vs. 4 [4.0%]; HR, 4.17; 95% CI, 0.47–37.34; $p = 0.201$).

3. Independent predictor of major adverse cardiac and cerebrovascular events in left main culprit lesion acute myocardial infarction with multivessel disease

In the univariate Cox proportional regression analysis, hyperten-

Table 2. Procedural characteristics

Variable	Before PSM			After PSM		
	Incomplete (n = 104)	Complete (n = 295)	p-value	Incomplete (n = 99)	Complete (n = 99)	p-value
No. of LM stents			> 0.999			> 0.999
1	92 (88.5)	262 (88.8)		87 (87.9)	87 (87.9)	
2	12 (11.5)	33 (11.2)		12 (12.1)	12 (12.1)	
Location of non-culprit lesion						
LAD	89 (85.6)	236 (80.0)	0.266 ^{a)}	84 (84.8)	85 (85.9)	> 0.999 ^{a)}
LCX	76 (73.1)	155 (52.5)	< 0.001 ^{a)}	72 (72.7)	70 (70.7)	0.875 ^{a)}
RCA	90 (86.5)	109 (36.9)	< 0.001 ^{a)}	85 (85.9)	49 (49.5)	0.001 ^{a)}
No. of non-culprit vessels per patient			< 0.001			0.828
1	11 (10.6)	132 (44.7)		11 (11.1)	13 (13.1)	
≥ 2	93 (89.4)	163 (55.3)		88 (88.9)	86 (86.9)	
Staged PCI		63 (21.4)			21 (21.2)	
Puncture site			0.182			0.114
Radial artery	49 (47.1)	115 (39.0)		48 (48.5)	36 (36.4)	
Femoral artery	55 (52.9)	180 (61.0)		51 (51.5)	63 (63.6)	
GpIIb/IIIa inhibitor	6 (5.8)	30 (10.2)	0.251	6 (6.1)	9 (9.1)	0.591
Intravascular image	70 (67.3)	176 (59.7)	0.302	69 (69.7)	59 (59.6)	0.181

Values are presented as number (%).

PSM, propensity score matching; LM, left main; LAD, left anterior descending; LCX, left circumflex; RCA, right coronary artery; PCI, percutaneous coronary intervention; GpIIb/IIIa, glycoprotein IIb/IIIa.

^{a)}p-value is for the comparison of each non-culprit epicardial vessel between the two groups. $p < 0.05$, significant difference.

Table 3. One-year clinical endpoint according to treatment of complete or incomplete revascularization

Endpoint	Before PSM (n = 399)				After PSM (n = 198)			
	Incomplete (n = 104)	Complete (n = 295)	aHR (95% CI)	p-value	Incomplete (n = 99)	Complete (n = 99)	aHR (95% CI)	p-value
Primary endpoint ^{a)}	11 (10.6)	29 (9.8)	0.94 (0.47–1.88)	0.862	12 (12.1)	15 (15.2)	1.28 (0.60–2.74)	0.524
Individual clinical endpoint								
All-cause deaths	4 (3.8)	8 (2.7)	0.71 (0.21–2.36)	0.578	5 (5.1)	5 (5.1)	1.02 (0.30–3.54)	0.971
Cardiovascular death	4 (3.8)	5 (1.7)	0.44 (0.12–1.65)	0.226	4 (4.0)	3 (3.0)	0.77 (0.17–3.44)	0.733
Myocardial infarction	3 (2.9)	5 (1.7)	0.60 (0.14–2.50)	0.479	3 (3.0)	4 (4.0)	1.43 (0.32–6.40)	0.636
Ischemia-driven revascularization	6 (5.8)	19 (6.4)	1.13 (0.45–2.84)	0.790	6 (6.1)	8 (8.1)	1.40 (0.49–4.03)	0.534
Stent thrombosis	0 (0)	3 (1.0)			0 (0)	2 (2.0)		
Stroke	1 (1.0)	5 (1.7)	1.85 (0.22–15.80)	0.576	1 (1.0)	4 (4.0)	4.17 (0.47–37.34)	0.165
Major bleeding (BARC type 3–5)	0 (0)	2 (0.7)			0 (0)	1 (1.0)		

Values are presented as numbers (%) unless otherwise specified.

PSM, propensity score matching; aHR, adjusted hazard ratio; CI, confidence interval; BARC, Bleeding Academic Research Consortium.

^{a)}Composite of all-cause deaths, myocardial infarction, ischemia driven revascularization stent thrombosis, or stroke. $p < 0.05$, significant difference.

sion (HR, 1.88; 95% CI, 0.97–3.64; $p = 0.062$), diabetes mellitus (HR, 2.00; 95% CI, 1.07–3.72; $p = 0.030$), previous history of MI (HR, 1.91; 95% CI, 0.75–4.87; $p = 0.178$), and current smoking status (HR, 0.59; 95% CI, 0.27–1.28; $p = 0.185$) showed a trend towards association with MACCE. In the multivariate analysis, which included these variables along with age and sex, diabetes mellitus was identified as the only independent predictor of MACCE (HR, 1.98; 95% CI, 1.06–3.69; $p = 0.031$) (Table 4).

Discussion

The main finding of this study was that in patients with LM culprit lesion AMI with multivessel disease, there was no significant difference in the 1-year clinical outcomes of MACCE between the incomplete and complete revascularization groups. However, the complete revascularization group had a slightly higher incidence of the primary endpoint, which was due to the higher occurrence of stroke. Specifically, all four cases of stroke in the complete revascularization group were ischemic strokes, and the one case in the incomplete revascularization group was a hemorrhagic stroke. Nota-

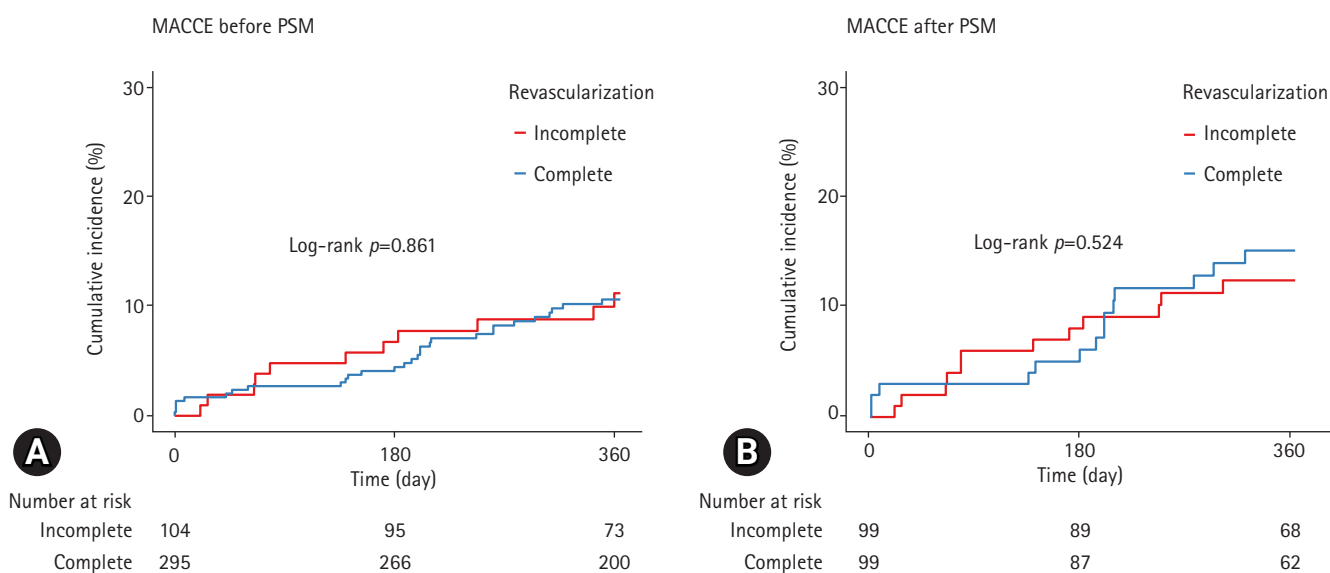


Fig. 2. Kaplan-Meier curve of primary endpoint in patients with left main culprit lesion acute myocardial infarction with multivessel disease comparing complete and incomplete revascularization groups. (A) Cumulative incidence of primary endpoint (major adverse cardiac and cerebrovascular events [MACCE]: composite of all-cause death, myocardial infarction, ischemia-driven revascularization, stent thrombosis, and stroke) before propensity score matching (PSM). (B) Cumulative incidence of primary endpoint (MACCE) after PSM.

bly, all stroke events occurred more than 3 months after the index procedure. Unfortunately, more detailed information about these stroke events was not available in the KAMIR, which limited our ability to determine the specific causes. The higher incidence of stroke in the complete revascularization group may be attributable to prolonged procedural times, additional vessel manipulation during multivessel treatment, or a higher burden of atherosclerosis in the patients selected for complete revascularization [21,22]. These factors could potentially lead to embolic events or damage to the vascular endothelium. However, further investigation is required to confirm this hypothesis.

Previous studies have shown that the clinical outcomes of patients with multivessel disease who underwent complete revascularization after AMI were better than those of patients who underwent culprit-only revascularization. This was mainly due to the significantly lower occurrence of MI and ischemia-driven revascularization in the 1- to 3-year follow-up period of the target group, which included fewer patients with cardiogenic shock or excluded them altogether [7,9,10]. In contrast, complete revascularization demonstrated less benefit than culprit-only revascularization in cases of AMI with multivessel disease accompanied by cardiogenic shock [23,24]. As reported by Thiele et al. [25] in 2017, the 30-day clinical outcomes showed that all-cause death and the rate of renal replacement therapy were significantly higher in patients who underwent complete revascularization, resulting in unfavorable outcomes. Based on these previous studies, our study targeted LM

culprit lesion AMI with multivessel disease. If cardiogenic shock was excluded, the 1-year clinical outcome would be expected to yield results similar to those of other studies on complete revascularization in AMI with multivessel disease.

In real-world clinical practice, performing complete revascularization can be challenging when patients are unstable. Consequently, it is highly likely that the condition of the patients in the incomplete revascularization group was more severe in the real-world data of the KAMIR. In this study, the initial plan was to exclude all patients with cardiogenic shock. However, it was challenging to analyze only patients who did not experience cardiogenic shock in cases of LM culprit lesion AMI with multivessel disease. Furthermore, as the KAMIR data included patients registered from 2016 to 2020, cardiogenic shock was not classified using the Society for Cardiovascular Angiography and Interventions (SCAI) cardiogenic shock classification proposed in 2019 [26]. Therefore, the analysis was conducted by classifying and excluding patients who required mechanical support, such as IABP and ECMO, or those who underwent CPR. However, it is important to note that even in cases where mechanical support was not provided, there would undoubtedly have been cases corresponding to stage C in the SCAI classification. Performing optimal PCI would have been difficult if the patient was unstable. Even if initially stable, it is common for the patient to become unstable during the LM disease PCI, suggesting that this variable likely influenced the performance of complete revascularization. This is likely a major variable ex-

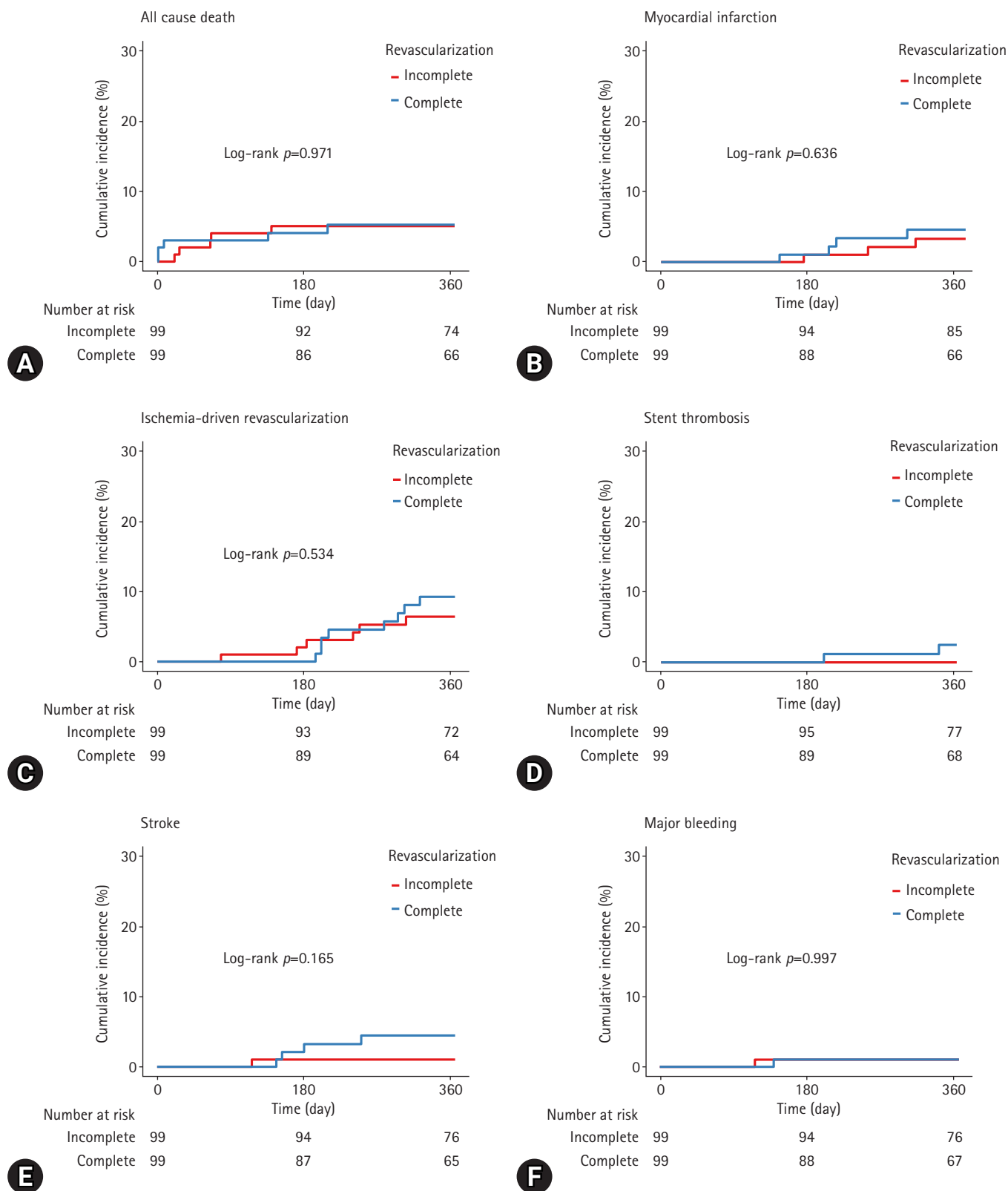


Fig. 3. Kaplan-Meier curve of secondary endpoints ([A] all-cause death, [B] myocardial infarction, [C] ischemia-driven revascularization, [D] stent thrombosis, [E] stroke, and [F] major bleeding) in propensity score-matched patients with left main culprit lesion acute myocardial infarction with multivessel disease comparing complete and incomplete revascularization groups.

Table 4. Predictors of major adverse cardiac and cerebrovascular events at 1 year in Cox proportional hazards regression analysis

Factor	Univariate analysis		Multivariate analysis	
	HR (95% CI)	p-value	HR (95% CI)	p-value
Age	1.48 (0.77–2.84)	0.234	1.00 (0.97–1.04)	0.814
Male sex	0.89 (0.43–1.82)	0.743	1.16 (0.55–2.44)	0.693
Hypertension	1.88 (0.97–3.64)	0.062	1.71 (0.87–3.35)	0.122
Diabetes mellitus	2.00 (1.07–3.72)	0.030	1.98 (1.06–3.69)	0.031
Dyslipidemia	0.52 (0.16–1.69)	0.277		
Previous MI	1.91 (0.75–4.87)	0.178	1.28 (0.50–3.33)	0.607
Previous PCI	1.65 (0.73–3.73)	0.229		
Current smoker	0.59 (0.27–1.28)	0.185	0.72 (0.33–1.59)	0.420
LVEF \leq 40%	1.18 (0.54–2.55)	0.684		
Previous stroke	1.35 (0.48–3.79)	0.570		
STEMI at admission	1.20 (0.63–2.28)	0.572		
Non-culprit vessel number \geq 2	1.13 (0.58–2.18)	0.727		

HR, hazard ratio; CI, confidence interval; MI, myocardial infarction; PCI, percutaneous coronary intervention; LVEF, left ventricular ejection fraction; STEMI, ST-elevation myocardial infarction.

$p < 0.05$, significant difference.

plaining why, in this study, stent thrombosis occurred only in the complete revascularization group, and there was no difference between the two groups in terms of MI or ischemia-driven revascularization events. Despite the various challenges encountered in the analysis, to the best of our knowledge, this study is the first to compare complete and incomplete revascularization PCI for LM culprit lesion AMI with multivessel disease.

However, this study had several limitations that must be addressed. First, the fundamental limitation of the study was the small number of participants. Second, there were various restrictions associated with the use of multicenter registry data, including missing data, a relatively short follow-up period of 1 year, and potential inconsistencies in variables due to data collection across multiple centers, all of which could have influenced the clinical outcomes. To address missing data, we conducted a complete case analysis, assuming that the relatively small proportion had a limited impact on the results. Nonetheless, potential bias may remain. Third, the observational nature of the registry and the retrospective nature of the study could lead to selection bias due to patients not being enrolled in the registry because of issues such as sudden death or periprocedural complications. Fourth, there was no detailed information on non-culprit lesion PCI in the KAMIR, including information on staged PCI performed after discharge, intermediate lesions, and physiology-guided PCI.

In conclusion, although this study found no significant difference between complete and incomplete revascularization in LM culprit AMI with multivessel disease, treatment decisions should be guided by patient-specific factors. Future randomized controlled trials are needed to confirm these findings and explore the long-term outcomes.

Additional information

¹Division of Cardiology, Department of Internal Medicine, Yeungnam University Medical Center, Daegu, Korea

²Division of Cardiology, Department of Internal Medicine, St. Vincent's Hospital, College of Medicine, The Catholic University of Korea, Suwon, Korea

³Division of Cardiology, Department of Internal Medicine, The Catholic University of Korea, Seoul St. Mary's Hospital, Seoul, Korea

⁴Division of Cardiology, Department of Internal Medicine, Korea University Anam Hospital, Seoul, Korea

⁵Division of Cardiology, Department of Internal Medicine, Chonnam National University Hospital, Gwangju, Korea

⁶Division of Cardiology, Department of Internal Medicine, Korea University Guro Hospital, Seoul, Korea

⁷Division of Cardiology, Department of Internal Medicine, Seoul National University Hospital, Seoul, Korea

⁸Division of Cardiology, Department of Internal Medicine, Samsung Medical Center, Sungkyunkwan University College of Medicine, Seoul, Korea

⁹Division of Cardiology, Department of Internal Medicine, Chungnam National University Hospital, Daejeon, Korea

¹⁰Division of Cardiology, Department of Internal Medicine, Chungbuk National University Hospital, Cheongju, Korea

¹¹Division of Cardiology, Department of Internal Medicine, Keimyung University Dongsan Medical Center, Daegu, Korea

¹²Division of Cardiology, Department of Internal Medicine, Pusan National University Hospital, Busan, Korea

¹³Division of Cardiology, Department of Internal Medicine, Wonkwang University Hospital, Iksan, Korea

¹⁴Division of Cardiology, Department of Internal Medicine, Chonbuk National University Hospital, Jeonju, Korea

Article information

Conflicts of interest

Ung Kim has been an editorial board member of *Journal of Yeungnam Medical Science* since 2014. He was not involved in the review process of this manuscript. There are no other conflicts of interest to declare.

Funding

None.

Author contributions

Conceptualization, Formal analysis: all authors; Data curation, Investigation, Visualization: SOK; Validation: HJK, JIP, KUC, JHN, CHL, JWS, JSP, SH Her, KYC, THA, MJ, SWR, HSK, HCG, IWS, KKH, SH Hur, KSC, SKO, JKC; Methodology: HJK, JIP; Supervision: HJK, JIP, KUC, JHN, CHL, JWS, JSP, SH Her, KYC, THA, MJ, SWR, HSK, HCG, IWS, KKH, SH Hur, KSC, SKO, JKC, UK; Writing-original draft: SOK; Writing-review & editing: all authors.

ORCID

Sun Oh Kim, <https://orcid.org/0000-0003-2851-7813>
 Hong-Ju Kim, <https://orcid.org/0000-0003-3510-3774>
 Jong-Il Park, <https://orcid.org/0000-0002-4337-4550>
 Kang-Un Choi, <https://orcid.org/0000-0002-3385-3152>
 Jong-Ho Nam, <https://orcid.org/0000-0001-5106-8361>
 Chan-Hee Lee, <https://orcid.org/0000-0001-9338-0679>
 Jang-Won Son, <https://orcid.org/0000-0002-8109-5018>
 Jong-Seon Park, <https://orcid.org/0000-0001-5242-2756>
 Sung-Ho Her, <https://orcid.org/0000-0002-1548-4154>
 Ki-Yuk Chang, <https://orcid.org/0000-0003-3456-8705>
 Tae-Hoon Ahn, <https://orcid.org/0000-0003-1544-1784>
 Myung-Ho Jeong, <https://orcid.org/0000-0003-2424-810X>
 Seung-Woon Rha, <https://orcid.org/0000-0001-9456-9852>
 Hyo-Soo Kim, <https://orcid.org/0000-0003-0847-5329>
 Hyeon-Cheol Gwon, <https://orcid.org/0000-0002-4902-5634>
 In-Whan Seong, <https://orcid.org/0000-0003-4628-0258>
 Kyung-Kuk Hwang, <https://orcid.org/0000-0003-3464-3023>
 Seung-Ho Hur, <https://orcid.org/0000-0002-3895-1915>
 Kwang-Soo Cha, <https://orcid.org/0000-0001-7980-4578>
 Seok-Kyu Oh, <https://orcid.org/0000-0001-7545-0143>
 Jei-Keon Chae, <https://orcid.org/0000-0001-5783-7950>
 Ung Kim, <https://orcid.org/0000-0002-6009-1843>

References

1. Byrne RA, Rossello X, Coughlan JJ, Barbato E, Berry C, Chieffo A, et al. 2023 ESC guidelines for the management of acute coronary syndromes. *Eur Heart J* 2023;44:3720–826.
2. Lawton JS, Tamis-Holland JE, Bangalore S, Bates ER, Beckie TM, Bischoff JM, et al. 2021 ACC/AHA/SCAI guideline for coronary artery revascularization: executive summary: a report of the American College of Cardiology/American Heart Association Joint Committee on clinical practice guidelines. *Circulation* 2022;145:e4–17.
3. Andersen HR, Nielsen TT, Rasmussen K, Thuesen L, Kelbaek H, Thayssen P, et al. A comparison of coronary angioplasty with fibrinolytic therapy in acute myocardial infarction. *N Engl J Med* 2003;349:733–42.
4. Goldstein JA, Demetriou D, Grines CL, Pica M, Shoukfeh M, O'Neill WW. Multiple complex coronary plaques in patients with acute myocardial infarction. *N Engl J Med* 2000;343:915–22.
5. Sorajja P, Gersh BJ, Cox DA, McLaughlin MG, Zimetbaum P, Costantini C, et al. Impact of multivessel disease on reperfusion success and clinical outcomes in patients undergoing primary percutaneous coronary intervention for acute myocardial infarction. *Eur Heart J* 2007;28:1709–16.
6. Jensen LO, Terkelsen CJ, Horváth-Puhó E, Tilsted HH, Maeng M, Junker A, et al. Influence of multivessel disease with or without additional revascularization on mortality in patients with ST-segment elevation myocardial infarction. *Am Heart J* 2015;170:70–8.
7. Mehta SR, Wood DA, Storey RF, Mehran R, Bainey KR, Nguyen H, et al. Complete revascularization with multivessel PCI for myocardial infarction. *N Engl J Med* 2019;381:1411–21.
8. Bainey KR, Alemayehu W, Armstrong PW, Westerhout CM, Kaul P, Welsh RC. Long-term outcomes of complete revascularization with percutaneous coronary intervention in acute coronary syndromes. *JACC Cardiovasc Interv* 2020;13:1557–67.
9. Biscaglia S, Guiducci V, Escaned J, Moreno R, Lanzilotti V, Santarelli A, et al. Complete or culprit-only PCI in older patients with myocardial infarction. *N Engl J Med* 2023;389:889–98.
10. Diletti R, den Dekker WK, Bennett J, Schotborgh CE, van der Schaaf R, Sabaté M, et al. Immediate versus staged complete revascularisation in patients presenting with acute coronary syndrome and multivessel coronary disease (BIOVASC): a prospective, open-label, non-inferiority, randomised trial. *Lancet* 2023;401:1172–82.
11. De Luca G, Suryapranata H, Thomas K, van 't Hof AW, de Boer MJ, Hoorntje JC, et al. Outcome in patients treated with prima-

- ry angioplasty for acute myocardial infarction due to left main coronary artery occlusion. *Am J Cardiol* 2003;91:235–8.
12. Kim U, Park JS, Kang SW, Kim YM, Park WJ, Lee SH, et al. Outcomes according to presentation with versus without cardiogenic shock in patients with left main coronary artery stenosis and acute myocardial infarction. *Am J Cardiol* 2012;110:36–9.
13. Neri R, Migliorini A, Moschi G, Valenti R, Dovellini EV, Antoniucci D. Percutaneous reperfusion of left main coronary disease complicated by acute myocardial infarction. *Catheter Cardiovasc Interv* 2002;56:31–4.
14. Tarantini G, D'Amico G, Tellaroli P, Colombo C, Brenner SJ. Meta-analysis of the optimal percutaneous revascularization strategy in patients with acute myocardial infarction, cardiogenic shock, and multivessel coronary artery disease. *Am J Cardiol* 2017;119:1525–31.
15. Sanborn TA, Sleeper LA, Webb JG, French JK, Bergman G, Parikh M, et al. Correlates of one-year survival inpatients with cardiogenic shock complicating acute myocardial infarction: angiographic findings from the SHOCK trial. *J Am Coll Cardiol* 2003;42:1373–9.
16. Karami M, Peters EJ, Lagrand WK, Houterman S, den Uil CA, Engström AE, et al. Outcome and predictors for mortality in patients with cardiogenic shock: a Dutch nationwide registry-based study of 75,407 patients with acute coronary syndrome treated by PCL. *J Clin Med* 2021;10:2047.
17. Cutlip DE, Windecker S, Mehran R, Boam A, Cohen DJ, van Es GA, et al. Clinical end points in coronary stent trials: a case for standardized definitions. *Circulation* 2007;115:2344–51.
18. Laskey WK, Yancy CW, Maisel WH. Thrombosis in coronary drug-eluting stents: report from the meeting of the circulatory system medical devices advisory panel of the food and drug administration center for devices and radiologic health, December 7-8, 2006. *Circulation* 2007;115:2352–7.
19. Kikkert WJ, van Geloven N, van der Laan MH, Vis MM, Baan J, Koch KT, et al. The prognostic value of bleeding academic research consortium (BARC)-defined bleeding complications in ST-segment elevation myocardial infarction: a comparison with the TIMI (Thrombolysis In Myocardial Infarction), GUSTO (Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Coronary Arteries), and ISTH (International Society on Thrombosis and Haemostasis) bleeding classifications. *J Am Coll Cardiol* 2014;63:1866–75.
20. Thygesen K, Alpert JS, Jaffe AS, Chaitman BR, Bax JJ, Morrow DA, et al. Fourth universal definition of myocardial infarction (2018). *Eur Heart J* 2019;40:237–69.
21. Werner N, Zahn R, Zeymer U. Stroke in patients undergoing coronary angiography and percutaneous coronary intervention: incidence, predictors, outcome and therapeutic options. *Expert Rev Cardiovasc Ther* 2012;10:1297–305.
22. Khatri P, Kasner SE. Ischemic strokes after cardiac catheterization: opportune thrombolysis candidates? *Arch Neurol* 2006;63:817–21.
23. Masiero G, Cardaioli F, Rodinò G, Tarantini G. When to Achieve complete revascularization in infarct-related cardiogenic shock. *J Clin Med* 2022;11:3116.
24. Khera R, Secemsky EA, Wang Y, Desai NR, Krumholz HM, Maddox TM, et al. Revascularization practices and outcomes in patients with multivessel coronary artery disease who presented with acute myocardial infarction and cardiogenic shock in the US, 2009-2018. *JAMA Intern Med* 2020;180:1317–27.
25. Thiele H, Akin I, Sandri M, Fuernau G, de Waha S, Meyer-Saracaei R, et al. PCI Strategies in patients with acute myocardial infarction and cardiogenic shock. *N Engl J Med* 2017;377:2419–32.
26. Baran DA, Grines CL, Bailey S, Burkhoff D, Hall SA, Henry TD, et al. SCAI clinical expert consensus statement on the classification of cardiogenic shock: this document was endorsed by the American College of Cardiology (ACC), the American Heart Association (AHA), the Society of Critical Care Medicine (SCCM), and the Society of Thoracic Surgeons (STS) in April 2019. *Catheter Cardiovasc Interv* 2019;94:29–37.