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ORIGINAL ARTICLE

Clinical impact of non-culprit lesions on 1-year mortality in very elderly patients with acute coronary syndrome

Satoshi Higuchi[1] · Yusuke Kabeya[2] · Kenichi Matsushita[1] · Hiroki Taguchi[1] · Haruhisa Ishiguro[1] · Hideyasu Kohshoh[1] · Hideaki Yoshino[1]

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Abstract Preventive percutaneous coronary intervention (PCI) for non-culprit lesions after primary PCI remains controversial in patients with acute coronary syndrome (ACS). We analyzed whether PCI for non-culprit lesions would be associated with a better long-term prognosis in very elderly (≥85 years) patients. This study included 91 consecutive patients with ACS (mean age, 88.2 ± 3.0, 52 % male). We investigated the association of residual lesions with 1-year mortality. Culprit lesions affected the left anterior descending artery (LAD) in 50 patients, the left circumflex artery (LCx) in 29, and the right coronary artery (RCA) in 31. Residual lesions affected LAD in 20 cases, LCx in 22, and RCA in 21 patients. Residual lesions in LAD were associated with a higher 1-year mortality ($p = 0.013$), whereas residual lesions in LCx or RCA were not ($p = 0.547$ and 0.473, respectively). A Cox regression model demonstrated that patients with residual lesions in LAD had an increased risk of 1-year mortality compared with those without residual lesions (hazard ratio, 2.39; 95 % confidence interval, 1.16–4.96; $p = 0.019$). Therefore, the option to not treat residual lesions in LAD of patients with PCI may be associated with a higher 1-year mortality. Further studies are needed to confirm these findings.

Keywords Very elderly · Acute coronary syndrome · Residual lesion · Percutaneous coronary intervention

✉ Hideaki Yoshino
yoshino@ks.kyorin-u.ac.jp

[1] Division of Cardiology, Department of Internal Medicine II, Kyorin University School of Medicine, 6-20-2 Shinkawa, Mitaka, Tokyo 181-8611, Japan

[2] Division of General Internal Medicine, Department of Internal Medicine, Tokai University Hachioji Hospital, 1838 Ishikawamachi, Hachioji, Japan

Introduction

Medical progress has extended the human lifespan in developed countries. Percutaneous coronary intervention (PCI) has become much safer and less invasive; therefore, very elderly (≥ 85 years) individuals have been increasingly referred for PCI, particularly in the clinical setting of acute coronary syndrome (ACS). In these patients, coronary angiography (CAG) sometimes shows multiple lesions [1]. The conventional strategy for ACS suggests that only the culprit lesion should be treated, followed by PCI for non-culprit lesions at a later session [2–4]. However, PCI for both culprit and non-culprit lesions during the initial procedure may be beneficial in reducing major adverse cardiovascular events (MACE) [5, 6]. In very elderly patients, non-culprit lesions are often treated only with optimal medication therapy in the absence of chest discomfort because of these patients' advanced age. As some elderly patients find it difficult to walk, chest discomfort becomes less likely to occur. It remains unclear whether we should perform PCI for non-culprit lesions.

Materials and methods

Study design

This retrospective study included 91 consecutive patients who were ≥85 years of age and admitted to the Kyorin University Hospital for ACS between 2007 and 2014. All patients underwent primary PCI. We reviewed all medical records to retrieve the patients' clinical information. We also evaluated their CAG, chest X-rays, and blood tests and categorized the patients according to the Killip

classification. [7] The institutional ethical review board at the Kyorin University School of Medicine approved the study.

Evaluation with CAG

We studied the culprit, non-culprit, and residual lesions in each patient. We defined the culprit lesions as the ones whose stenosis or occlusion was causing ACS. Lesions with ≥75 % stenosis or complete occlusion that were not associated with the development of ACS were defined as non-culprit lesions. Residual lesions were non-culprit lesions that were not treated with PCI during the admission. The decision of whether the lesion was a culprit or non-culprit lesion was based on findings from CAG, electrocardiogram, and echocardiography, according to the assessments of two interventionists. We performed quantitative coronary angiography (QCA) to evaluate each stenosis or occlusion, using the software Qangio XA, version 7.1 (Medis, Leiden, the Netherlands). Culprit lesions in the left anterior descending artery (LAD), left circumflex artery (LCx), and right coronary artery (RCA) are represented as cLAD, cLCx, and cRCA, respectively, whereas residual lesions in these arteries are represented as rLAD, rLCx, and rRCA, respectively.

Procedure

We evaluated the success rate of primary PCI for ACS. The procedure was deemed successful when achieving a <30 % angiographic residual stenosis and a thrombolysis in myocardial infarction flow grade 3 (TIMI3) after treatment of the culprit lesion. [8] Complete revascularization was defined as the absence of a ≥75 % stenosis in LAD, LCx, and RCA, and the absence of a ≥50 % stenosis in the left main trunk (LMT) after PCI. We also evaluated the type of device used for the intervention. The procedures included plain old balloon angioplasty (POBA), a bare metal stent (BMS), and drug-eluting stent (DES). The type of DES was also recorded.

Clinical outcome

We assessed the 1-year mortality and its associations with the presence of non-culprit or residual lesions. The cause of death was also evaluated. Furthermore, we evaluated whether PCI for the non-culprit lesion was related to the improvement in activities of daily living (ADL) at discharge. ADL was assessed at admission and discharge using the Barthel index [9]. According to the study, we defined the ADL as "good" when the Barthel index was ≥85. Data on mortality were obtained from medical records or telephone conversation with the patients' families.

Statistical analysis

Numerical data with normal distribution are presented as mean ± standard deviation. Otherwise, data are presented as median and interquartile range (quartile 1–3). Categorical variables were analyzed using Fisher's exact test and chi-square test and are expressed as absolute numbers or percentages. Continuous variables were analyzed using unpaired Student's *t* test or Mann–Whitney test. Bonferroni corrections were used for post hoc tests, and Kaplan–Meier statistics were used to estimate the cumulative 1-year mortality. The risk of mortality was assessed using the Cox regression analysis, which was adjusted for age, gender, and Killip classification and expressed as a hazard ratio (HR) and 95 % confidence interval (CI). To evaluate the HR of each vessel with a culprit lesion, we used as a reference a relevant non-culprit vessel or a vessel with no lesions. To assess HR of each vessel with residual lesions, the reference was the lesion treated with PCI or the relevant vessel with no lesions. As for the association between a residual lesion and ADL on discharge, we used logistic regression analysis to calculate the odds ratios (ORs), which was adjusted for age, gender, and ADL on admission. This analysis was restricted to patients discharged alive from the hospital. Differences were regarded significant when $p < 0.05$. All statistical analyses were performed using the software Stata (version 10; StataCorp, College Station, Texas, USA).

Results

Angiographic characteristics

The present study included 91 very elderly patients (mean age, 88.2 ± 3.0 years; range, 85–99 years; 52 % male). The median (interquartile range) Barthel index on admission in all patients was 100 (90–100). The median follow-up duration was 341 days (15–1302 days; mean, 681 days). Thirty-three patients (36 %) died in the year following the development of the ACS; 25 (76 %) of whom died of a cardiac disease. The culprit lesions were located in LAD in 50 cases, in LCx in 10 cases, and in RCA in 31 cases. QCA results were as follows: LAD, 96 % (90–100 %); LCx, 99 % (91–100 %); and RCA, 97 % (91–100 %) ($p < 0.001$). LCx was significantly narrower than LAD and RCA ($p < 0.001$ and 0.002, respectively). We did not detect culprit lesions in LMT; however, non-culprit lesions were found in LMT in eight patients. Single-vessel disease was detected in 45 patients, double-vessel disease in 25, and triple-vessel disease in 21. The success rate of PCI was 91 %. The guide wire could not advance beyond the culprit lesion in one patient, and the final coronary flow

was <TIMI3 in seven patients. Complete revascularization was not performed in 43 patients during hospitalization. In cases in which complete revascularization was performed, the culprit and non-culprit lesions were not treated simultaneously. Residual lesions were located as follows: LAD, 20 patients; LCx, 22 patients; and RCA, 21 patients. QCA results showed the following degrees of stenosis: LAD, 84 ± 13 %; LCx, 86 ± 13 %; and RCA, 80 ± 13 %; ($p = 0.206$). Patients' characteristics were classified according to the culprit and residual lesions (Tables 1, 2, respectively). Left ventricular ejection fraction was low, and heart failure was more common in patients with cLAD than in those with other lesions. DES and BMS were used at the primary PCI in 32 and 53 patients, respectively. There were no significant differences in the characteristics between the groups with and without rLAD, except for dyslipidemia and use of beta-blockers.

Relationship between angiographic findings and 1-year mortality

Cox regression analysis of 1-year mortality is shown in Table 3. The culprit lesions were not associated with the 1-year mortality. However, rLAD was associated with higher 1-year mortality ($p = 0.013$) (Fig. 1), whereas rLCx and rRCA were not ($p = 0.547$ and $p = 0.473$, respectively). All patients with rLAD ($n = 11$) died from heart failure with reduced ejection fraction within 2 months. The Cox regression model demonstrated that patients with residual lesions in LAD had an increased risk of 1-year mortality compared with that in those without rLAD lesions (HR, 2.39; 95 % CI, 1.16–4.96; $p = 0.019$). The significance persisted after adjustment for age, gender, and Killip classification (HR, 3.00; 1.41–6.37; $p = 0.004$). Patients without rLAD included those with cLAD who underwent PCI ($n = 46$) and those without cLAD on admission ($n = 25$). There was no significant difference in 1-year mortality between both groups ($p = 0.831$). Each patient in the non-rLAD group had a better 1-year prognosis than those with rLAD ($n = 20$) (patients without cLAD, $p = 0.034$ and patients with cLAD who underwent PCI, $p = 0.034$) (Fig. 2).

Although the number at risk reduced, the Cox regression model demonstrated that compared with patients without rLAD, those with rLAD had an increased risk of 3-year mortality (HR, 2.43; 95 % CI, 1.21–4.87; $p = 0.012$).

Association between residual lesion and ADL

The median Barthel index on admission in the present study was 100 (90–100). Therefore, ADL was classified as good in most patients. The presence of rLAD was associated with worse ADL at discharge (adjusted OR, 5.77; 95 % CI, 1.02–32.6; $p = 0.047$). Both rLCx and rRCA were not related to ADL.

Clinical impact of the Killip classification on 1-year mortality

A higher Killip class was significantly associated with worse 1-year prognosis ($p = 0.001$). Patients with Killip I ($n = 45$) or II ($n = 13$) had a better prognosis than those with Killip III ($n = 8$) or IV ($n = 3$) (OR, 4.61; 95 % CI 1.49–14.64; $p = 0.002$).

Discussion

Prognostic impact of residual lesions

The present study revealed that an rLAD is associated with a 1-year mortality even in very elderly patients. Patients without rLAD included those with cLAD who were treated with PCI and those without cLAD at the first CAG. The 1-year prognosis for both was similar. Thus, PCI may improve the prognosis in patients with LAD lesions and equate the prognosis to those without LAD lesions. Conversely, rLCx and rRCA were not related to the 1-year mortality.

The clinical significance of PCI differs according to the stability of the ischemia. In patients with stable angina pectoris, PCI is useful in alleviating the symptoms, but not in prolonging the lifespan in most cases. In patients with ACS, PCI is useful in improving both symptoms and lifespan. The COURAGE trial demonstrated no difference in myocardial infarction and major cardiovascular events between optimal medical treatment (OMT) with and without PCI in patients with stable angina pectoris. [10] Nevertheless, the results in the PCI group were not similar to those in the OMT group in all cases. A substudy of the COURAGE trial, which included patients who underwent myocardial perfusion single photon emission computed tomography, suggested that a ≥10 % ischemia is associated with worse event-free survival. [11] Hachamovitch et al. also found increasing survival benefit of PCI over medical treatments when patients had moderate-to-severe ischemia. [12] These studies suggest that a large area of the myocardium at risk is associated with a poor prognosis. There have been no randomized trials comparing PCI to OMT in patients with isolated culprit lesions in LAD. However, LAD generally feeds a broader territory than other vessels. Therefore, rLAD may be related to 1-year mortality. As the ischemic areas in LCx and RCA are usually narrower than those in LAD, rLCx and rRCA do not affect 1-year mortality. According to previous studies as well as the present one, OMT without PCI may be appropriate in very elderly

Table 1 Comparison of patient characteristics according to the culprit lesions

	Total ($n = 91$)	LAD ($n = 50$)	LCx ($n = 10$)	RCA ($n = 31$)	*P* value			
					All	LAD vs LCx	LAD vs RCA	LCx vs RCA
Background								
Age	88.2 ± 3.0	88.3 ± 3.3	86.9 ± 2.3	88.5 ± 2.8	NS	NS	NS	NS
Male	47 (52)	22 (44)	7 (70)	18 (58)	NS	NS	NS	NS
BMI	21.1 ± 3.1	20.7 ± 3.1	23.4 ± 3.3	21.2 ± 2.7	NS	0.021	NS	NS
sBP (mmHg)	125 ± 33	128 ± 36	131 ± 21	119 ± 32	NS	NS	NS	NS
dBP (mmHg)	69 ± 21	74 ± 24	67 ± 9	63 ± 18	NS	NS	0.023	NS
HR (beats per minute)	83 ± 31	91 ± 33	69 ± 18	73 ± 27	0.004	0.012	0.006	NS
Coronary risk factor								
Hypertension	73 (80)	39 (78)	10 (100)	24 (77)	NS	NS	NS	NS
Dyslipidemia	31 (34)	19 (38)	4 (40)	8 (26)	NS	NS	NS	NS
Diabetes mellitus	25 (27)	13 (26)	4 (40)	8 (26)	NS	NS	NS	NS
Current smoker	10 (11)	4 (8)	2 (20)	4 (13)	NS	NS	NS	NS
ACS classification								
STEMI	60 (66)	34 (68)	4 (40)	22 (71)	NS	NS	NS	NS
Non-STEMI	29 (32)	16 (32)	4 (40)	9 (29)	NS	NS	NS	NS
UAP	2 (2)	0 (0)	2 (20)	0 (0)	0.010	0.025	NS	NS
Examination on admission								
Hb (g/dl)	11.6 ± 1.7	11.6 ± 1.9	11.8 ± 1.0	11.6 ± 1.8	NS	NS	NS	NS
Cr (mg/dl)	0.9 (0.7–1.3)	0.8 (0.7–1.1)	0.9 (0.8–1.3)	1.1 (0.8–1.6)	0.021	NS	0.007	NS
CRP (mg/dl)	0.3 (0.2–1.3)	0.3 (0.2–1.1)	0.3 (0.1–0.5)	0.5 (0.1–4.5)	NS	NS	NS	NS
CK (IU/L)	1014 (351–3056)	946 (351–4242)	657 (96–1638)	1191 (432–2848)	NS	NS	NS	NS
BNP (pg/ml)	350 (143–823)	376 (148–910)	176 (63–466)	391 (240–792)	NS	NS	NS	0.018
LVEF (%)	46 ± 14	42 ± 13	53 ± 11	49 ± 13	0.006	0.016	0.009	NS
Complications								
Heart failure	60 (66)	37 (74)	3 (30)	20 (65)	0.034	0.012	NS	NS
CKD stage	3 (2–3)	2 (2–3)	3 (2–3)	3 (3–4)	0.016	NS	0.002	NS
Free wall rupture	9 (10)	9 (18)	0 (0)	0 (0)	0.017	NS	0.013	NA
AKI on admission	12 (13)	4 (8)	0 (0)	8 (26)	0.047	NS	0.032	NS
CI-AKI	13 (14)	7 (14)	2 (20)	4 (13)	NS	NS	NS	NS
Examination at discharge								
Hb (g/dl)	11.0 ± 1.9	11.3 ± 1.8	10.9 ± 1.3	10.5 ± 2.3	NS	NS	NS	NS
Cr (mg/dl)	1.0 (0.7–1.4)	0.9 (0.7–1.3)	1.1 (0.8–1.4)	1.1 (0.8–2.1)	NS	NS	NS	NS
Medication at discharge								
Aspirin	86 (95)	49 (98)	9 (90)	28 (90)	NS	NS	NS	NS
Clopidogrel	85 (93)	50 (100)	8 (80)	27 (87)	0.005	0.025	0.019	NS
ACE/ARB	59 (65)	34 (68)	6 (60)	19 (61)	NS	NS	NS	NS
β Blockade	54 (59)	30 (60)	8 (80)	16 (52)	NS	NS	NS	NS
Statin	45 (49)	27 (54)	4 (40)	14 (45)	NS	NS	NS	NS

Variables are *n* (%), mean ± SD, or median (interquartile range). $P < 0.05$ is considered significant

ACE/ARB angiotensin converting enzyme/angiotensin receptor blockers, *AKI* acute kidney injury, *BMI* body mass index, *CI-AKI* contrast-induced acute kidney injury, *CKD* chronic kidney disease, *dBP* diastolic blood pressure, *HR* heart rate, *LAD* left anterior descending artery, *LCx* left circumflex artery, *LVEF* left ventricular ejection fraction, *NSTEMI* non-ST-segment myocardial infarction, *NS* not significant, *RCA* right coronary artery, *sBP* systolic blood pressure, *STEMI* ST-segment elevation myocardial infarction, *UAP* unstable angina pectoris

Table 2 Comparison of clinical characteristics between those with residual lesion in left anterior descending artery (LAD) and those without

	Residual lesion in LAD ($n = 20$)	No residual lesion in LAD ($n = 71$)	P value
Background			
Age	88.4 ± 2.4	88.2 ± 3.2	NS
Male	11 (55)	36 (51)	NS
BMI	21.9 ± 3.1	20.9 ± 3.0	NS
sBP (mmHg)	109 ± 44	130 ± 28	NS
sDP (mmHg)	59 ± 28	72 ± 18	NS
HR (beats per minute)	71 ± 27	86 ± 32	NS
Coronary risk factor			
Hypertension	16 (80)	57 (80)	NS
Dyslipidemia	2 (10)	29 (41)	0.008
Diabetes mellitus	6 (30)	19 (27)	NS
Current smoker	3 (15)	7 (10)	NS
ACS classification			
STEMI	15 (75)	45 (63)	NS
Non-STEMI	4 (20)	25 (35)	NS
UAP	1 (5)	1 (1)	NS
Examination on admission			
Hb (g/dl)	11.7 ± 1.3	11.6 ± 1.9	NS
Cr (mg/dl)	1.0 (0.8–1.4)	0.8 (0.7–1.3)	NS
CRP (mg/dl)	0.4 (0.2–1.9)	0.3 (0.1–1.3)	NS
CK (IU/L)	1258 (560–4396)	1014 (315–2848)	NS
BNP (pg/ml)	319 (139–863)	380 (143–834)	NS
LVEF (%)	45 ± 15	46 ± 13	NS
Complications			
Heart failure	11 (55)	49 (69)	NS
CKD stage	3 (3–3)	3 (2–3)	NS
Free wall rupture	1 (5)	8 (11)	NS
AKI on admission	3 (15)	9 (13)	NS
CI-AKI	5 (25)	8 (11)	NS
Examination at discharge			
Hb (g/dl)	10.6 ± 1.9	11.1 ± 2.0	NS
Cr (mg/dl)	1.2 (0.8–2.0)	1 (0.7–1.3)	NS
Medication at discharge			
Aspirin	18 (90)	68 (96)	NS
Clopidogrel	19 (95)	66 (93)	NS
ACE/ARB	10 (50)	49 (69)	NS
β Blockade	8 (40)	46 (65)	0.042
Statin	8 (40)	37 (52)	NS

Variables are n (%), mean ± SD, or median (interquartile range). $P < 0.05$ is considered significant

Continuous variables were analyzed using Mann–Whitney test

Categorical variables were analyzed using the chi-square tests or Fisher's exact test

AKI acute kidney injury, *BMI* body mass index, *CI-AKI* contrast-induced acute kidney injury, *CKD* chronic kidney disease, *dBP* diastolic blood pressure, *HR* heart rate, *LAD* left anterior descending artery, *LCx* left circumflex artery, *LVEF* left ventricular ejection fraction, *NSTEMI* Non-ST-segment myocardial infarction, *NS* not significant, *RCA* right coronary artery, *sBP* systolic blood pressure, *STEMI* ST-segment elevation myocardial infarction, *UAP* unstable angina pectoris

Table 3 Cox regression analysis of relationship between angiographic findings and 1-year mortality

Variable	Univariate		Multivariate	
	HR (95 % CI)	*P* value	HR (95 % CI)	*P* value
Culprit lesion in LAD	0.91 (0.46–1.80)	NS	1.02 (0.51–2.04)	NS
Culprit lesion in LCx	0.45 (0.11–1.89)	NS	0.64 (0.15–2.83)	NS
Culprit lesion in RCA	1.46 (0.73–2.91)	NS	1.11 (0.54–2.29)	NS
Residual lesion in LAD	2.39 (1.16–4.96)	0.019	3.00 (1.41–6.37)	0.004
Residual lesion in LCx	1.26 (0.58–2.71)	NS	0.96 (0.42–2.19)	NS
Residual lesion in RCA	0.73 (0.30–1.77)	NS	0.65 (0.27–1.60)	NS
Complete revascularization	0.55 (0.27–1.09)	NS	0.63 (0.30–1.31)	NS

The multivariate model was adjusted for age, gender, and Killip classification

CI confidence interval, *HR* hazard ratio, *LAD* left anterior descending artery, *LCx* left circumflex artery, *NS* not significant, *RCA* right coronary artery

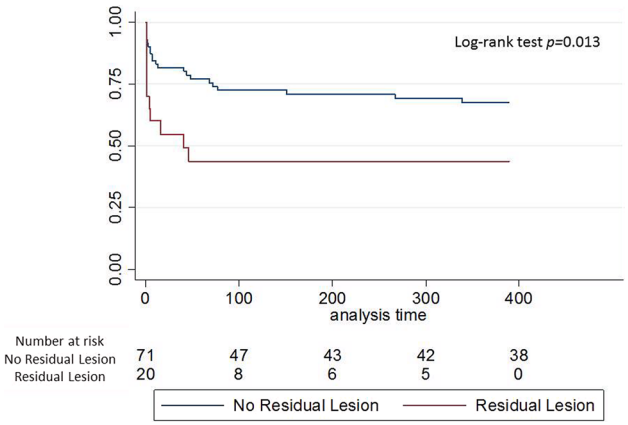

Fig. 1 Association between residual lesions in the left anterior descending artery and 1-year mortality. Residual lesions in the left anterior descending artery were associated with higher 1-year mortality

patients with rLCx or rRCA if they do not experience chest discomfort.

Differences in clinical impact among the culprit lesions

Here, there was no significant difference in 1-year mortality associated with culprit lesions. In the setting of ACS, lesions in LCx and RCA can be lethal. Fatal complications include free wall rupture in those with an acute occlusion

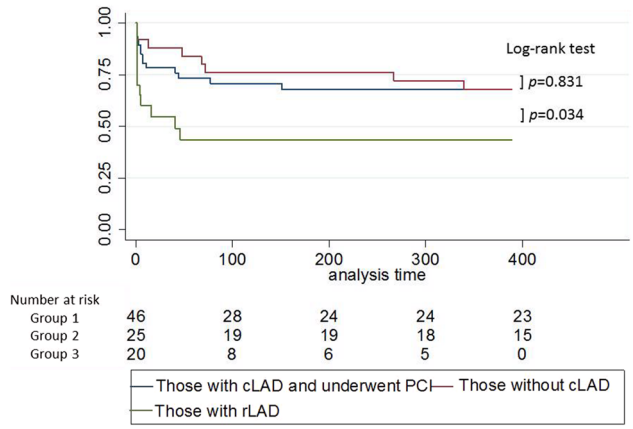

Fig. 2 The clinical impact of percutaneous coronary intervention (PCI) for non-culprit lesion in the left anterior descending artery (LAD). Group 1 (*upper line*), those without culprit lesions in the LAD (cLAD); Group 2 (*middle line*), those with cLAD who underwent PCI; and Group 3 (*lower line*), patients with residual lesions in LAD (rLAD). The patients with cLAD had similar 1-year prognosis as those with no LAD lesion after successful PCI

of LCx and right ventricular infarction in those with an acute occlusion of RCA. Lethal ventricular tachyarrhythmia can occur in both. As culprit lesions in vessels other than LAD can contribute to in-hospital mortality, there may be no difference in mortality among culprit lesions.

Is complete revascularization required for a better prognosis?

There is controversy as to whether and when PCI should be performed for non-culprit lesions in the clinical setting of ACS. The American College of Cardiology/American Heart Association (ACC/AHA) guidelines state that "Primary PCI should not be performed in a noninfarct artery at the time of primary PCI in patients with ST-segment elevation myocardial infarction (STEMI) who are hemodynamically stable," and "PCI is reasonable in a noninfarct artery at a time separate from primary PCI in patients with intermediate- or high-risk findings on noninvasive testing." [13] In accordance with the ACC/AHA guidelines, several studies have failed to indicate any benefit of PCI for non-culprit lesions during hospitalization. [14–17] Conversely, some studies have shown a benefit of complete revascularization. A meta-analysis by Sarathy et al. has revealed that the treatment of non-culprit lesions, in addition to culprit lesions, is associated with lower all-cause mortality in patients with STEMI. [18] Other studies [14, 19] have suggested that PCI for multiple lesions during primary PCI is related to a higher mortality. However, staged PCI for non-culprit lesions after the primary PCI resulted in a lower mortality. Kornowski et al. have demonstrated that the mortality of

patients following the deferred PCI strategy might be lower than that in those following the simultaneous PCI strategy. [20] According to these studies, angioplasty for non-culprit lesions during primary PCI appears to be associated with a higher mortality, whereas PCI for non-culprit lesions at a different time does not appear to be harmful. The present study did not show worsened outcomes associated with complete revascularization compared with those associated with culprit-only revascularization. The patients included in this study underwent PCI for non-culprit lesions at a different time from the primary PCI. The optimal timing to perform PCI for non-infarct arteries remains unclear. However, according to the findings of the present study, which showed cardiac death occurring within 2 months after discharge, a subsequent PCI for non-infarct arteries should be planned within 2 months after the primary PCI.

Association between rLAD and ADL

Residual lesions in LAD were independently associated with worse ADL on discharge. PCI for rLAD may be associated not only with prognosis at 1 year but also with a better ADL on discharge.

Study limitation

The small sample size in the present study could have limited our ability to detect other potential prognosis, such as residual lesions in LCx and RCA. Further studies with a sufficient number of very elderly patients may be required to confirm our findings.

To confirm the benefits of preventive PCI, a prospective study comparing patients with and without PCI for rLAD is required, although it may be difficult to conduct such studies among very elderly patients.

Compliance with ethical standards

Conflict of interest The authors have no conflict of interests to disclose.

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