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Primary Percutaneous Coronary Intervention for Unprotected Left Main Disease in Patients With Acute ST-Segment Elevation Myocardial Infarction

The AMIS (Acute Myocardial Infarction in Switzerland) Plus Registry Experience

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Objectives This study sought to assess outcomes in patients with ST-segment elevation myocardial infarction undergoing primary percutaneous coronary intervention (PCI) for unprotected left main (LM) disease.

Background Limited data are available on outcomes in patients with ST-segment elevation myocardial infarction undergoing LM PCI.

Methods Of 9,075 patients with ST-segment elevation myocardial infarction enrolled in the AMIS (Acute Myocardial Infarction in Switzerland) Plus registry between 2005 and June 30, 2010, 6,666 underwent primary PCI. Of them, 348 (5.2%; mean age: 63.5 ± 12.6 years) underwent LM PCI, either isolated (n = 208) or concomitant to PCI for other vessel segments (n = 140). They were compared with 6,318 patients (94.8%; mean age: 61.9 ± 12.5 years) undergoing PCI of non-LM vessel segments only.

Results The LM patients had higher rates of cardiogenic shock (12.2% vs. 3.5%; $p < 0.001$), cardiac arrest (10.6% vs. 6.3%; $p < 0.01$), in-hospital mortality (10.9% vs. 3.8%; $p < 0.001$), and major adverse cardiac and cerebrovascular events (12.4% vs. 5.0%; $p < 0.001$) than non-LM PCI. Rates of mortality and major adverse cardiac and cerebrovascular events were highest for concurrent LM and non-LM PCI (17.9% and 18.6%, respectively), intermediate for isolated LM PCI (6.3% and 8.3%, respectively), and lowest for non-LM PCI (3.8% and 5.0%, respectively). Rates of mortality and major adverse cardiac and cerebrovascular events for LM PCI were higher than for non-LM multivessel PCI (10.9% vs. 4.9%, $p < 0.001$, and 12.4% vs. 6.4%, $p < 0.001$, respectively). LM disease independently predicted in-hospital death (odds ratio: 2.36; 95% confidence interval: 1.34 to 4.17; $p = 0.003$).

Conclusions Emergent LM PCI in the context of acute myocardial infarction, even including 12% cardiogenic shock, appears to have a remarkably high (89%) in-hospital survival. Concurrent LM and non-LM PCI has worse outcomes than isolated LM PCI. (J Am Coll Cardiol Intv 2011;4:627–33)

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Unprotected left main (LM) coronary artery disease, which is observed in 3% to 5% of patients undergoing coronary angiography, has major prognostic implications (1,2). Based on early randomized trials (3,4), current guidelines (5) support the use of coronary artery bypass graft surgery in patients with unprotected LM disease. However, recent data show that percutaneous coronary intervention (PCI) may be a safe and effective alternative to surgical revascularization in selected patients (6–10). However, unprotected LM disease still poses a significant challenge to the interventional cardiologist. Primary PCI has become the standard treatment for patients presenting with acute ST-segment elevation myocardial infarction (STEMI), including those with LM occlusion. Unlike patients with stable angina (8,9,11), however, limited data are available on patients with unprotected LM disease presenting with acute coronary syndromes (ACS) including STEMI (12–16). Therefore, we analyzed the clinical characteristics and outcomes of patients with STEMI treated with primary PCI for unprotected LM disease who were included in the nationwide acute myocardial infarction in Switzerland AMIS (Acute Myocardial Infarction in Switzerland) Plus registry between January 1, 2005, and June 30, 2010. These patients were compared with patients with STEMI treated with PCI of other vessel segments and included in this registry during the same period.

Abbreviations and Acronyms

ACS = acute coronary syndrome(s)

LM = left main

MACCE = major adverse cardiac and cerebrovascular event(s)

PCI = percutaneous coronary intervention

STEMI = ST-segment elevation myocardial infarction

Methods

Patients. The AMIS Plus registry is a nationwide prospective registry of patients admitted with ACS to 76 hospitals in Switzerland (17). The registry was initiated in 1997, and patient recruitment has been ongoing since then. Participating centers, ranging from community institutions to large tertiary facilities, provide data for each patient through a standardized Internet- or paper-based questionnaire. The questionnaire includes 200 items addressing medical history, cardiovascular risk factors, symptoms, out-hospital management, reperfusion therapy, hospital course, the diagnostic test used, length of stay, and discharge medication. Data collection is centralized at the Institute of Social and Preventive Medicine of the University of Zürich, and checked for consistency. Incomplete questionnaires are returned to the enrollment centers for completion. The registry was approved by the Over-Regional Ethics Committee for Clinical Studies, the Swiss Board for Data Security, and the appropriate Cantonal Ethic Commissions. AMI patients who had ST-segment elevation or new left bundle branch block on their initial electrocardiograms were classified as having STEMI. All patients presenting with STEMI and treated with primary PCI, and who were entered into the AMIS Plus registry from January 1, 2005, to June 30, 2010, were included in the analysis. STEMI patients were subdivided into those treated with PCI for unprotected LM disease (LM PCI), either alone (isolated LM PCI) or in combination with PCI of other vessel segments (nonisolated LM PCI), and those treated with PCI of vessel segments other than LM (non-LM PCI). The study's primary endpoints included in-hospital mortality and rates of major adverse cardiac and cerebrovascular events (MACCE), defined as cumulative composite of death, reinfarction, and cerebrovascular events. Bleeding was defined as major bleeding including intracerebral hemorrhage, local bleeding requiring surgical treatment, requirement for blood transfusions, or a drop in blood hemoglobin of more than 3.0 g/dl.

Statistical analyses. Data are presented as the proportion of valid cases for discrete variables and as mean ± SD and/or medians with interquartile ranges for continuous variables. Differences in baseline characteristics were compared using the unpaired *t* test or Mann-Whitney *U* test if appropriate and the Pearson chi-square test. Statistics for each table are based on all cases with valid data in the specified ranges for all variables in each table. The 95% confidence intervals for the odds ratios were calculated for treatment assignments and outcome using multivariate logistic regression, with backward stepwise variable selection. The following predictors were included in the analysis: LM PCI, age, sex, history of hypertension, history of dyslipidemia, current smoker, resuscitation, cardiogenic shock (Killip class IV) at admission, heart rate, systemic blood pressure, and a Charlson score >2. The Charlson comorbidity index takes into account the number and the seriousness of comorbid disease (18,19). A p value <0.05 was considered significant. The SPSS software (version 17.0, SPSS Inc., Chicago, Illinois) was used for all statistical analyses.

Results

Patient population. The AMIS Plus registry included 9,075 patients admitted for STEMI to 61 Swiss hospitals between January 1, 2005, and June 30, 2010 (Fig. 1). Of them, 7,049 patients underwent primary PCI. After exclusion of 383 patients because of incomplete data, 6,666 patients were included in our analysis. They were subdivided into 348 patients (5.2%) treated with LM PCI and 6,318 patients (94.8%) treated with PCI of other vessel segments (non-LM group). Of the 348 patients in the LM group, 208 (58%) underwent isolated LM PCI and 140 patients (42%) underwent combined PCI of LM and other vessel segments (nonisolated LM PCI).

Baseline clinical and angiographic data. Baseline clinical and angiographic data in the LM and non-LM PCI groups, as well as in the isolated and nonisolated LM subgroups, are shown in Table 1. The mean age of the overall study population was 62.0 ± 12.5 years. The sex distribution was

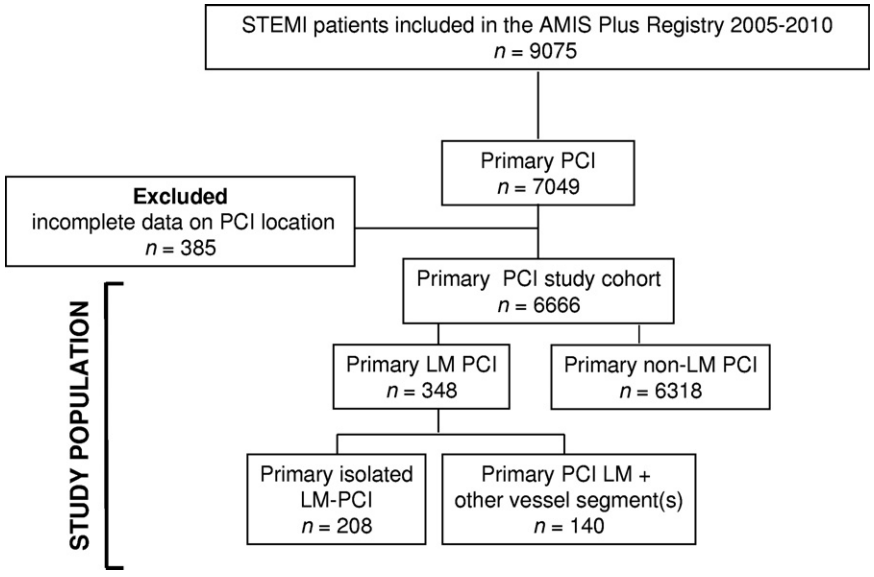

Figure 1. Study Flow Diagram

The flow chart shows the breakdown of included and excluded patients. AMIS = Acute Myocardial Infarction in Switzerland; LM = left main; PCI = percutaneous coronary intervention; STEMI = ST-segment elevation myocardial infarction.

77% men and 23% women. The risk profile of the overall study population was relatively high: 16.7% patients were older than 75 years; 11.1% and 3.3% had a history of myocardial infarction and stroke, respectively; and 3.4% had chronic renal failure. The LM PCI patients were slightly older, and slightly more were women compared with non-LM PCI patients. The prevalence of cardiovascular risk factors and comorbidities, as reflected by a Charlson weighted index >2, was comparable in the 2 groups. The LM PCI patients presented more frequently with dyspnea, low left ventricular ejection fraction, cardiogenic shock, cardiac arrest, and need for pharmacological inotropic or mechanical (intra-aortic balloon pump) support. Median door-to-balloon time was longer in LM versus non-LM PCI patients (70 vs. 60 min, respectively; $p = 0.032$). Patients with isolated LM PCI were younger and tended to have fewer comorbidities than those with nonisolated LM PCI did. Of nonisolated LM PCI patients, 6.9% had disease of 1 coronary vessel besides LM, 48.1% of 2 vessels besides LM, and 45.0% of 3 vessels besides LM. Of non-LM PCI patients, 44.3% had 1-vessel disease, 29.9% had 2-vessel disease, and 25.8% had 3-vessel disease. The number of drug-eluting stents as a percentage of all implanted stents was similar in LM PCI and non-LM PCI patients (78.3% vs. 73.8%, respectively; $p = 0.09$).

In-hospital outcomes. In-hospital outcomes are summarized in Table 2. In-hospital mortality rates were higher for LM than non-LM PCI patients (10.9% vs. 3.8%; $p < 0.001$), as were in-hospital rates of MACCE (12.4% vs. 5.0%; $p < 0.001$), and the proportion of patients presenting with cardiogenic shock on admission (12.2% vs. 3.5%; $p < 0.001$). In both groups, in-hospital mortality was extremely high in patients presenting with cardiogenic shock on admission, with a significant difference between LM and non-LM PCI (54.8% vs. 35.5%, respectively; $p = 0.024$). Patients developing cardiogenic shock after admission during hospitalization also were more frequent in the LM versus non-LM PCI group (6.1% vs. 3.3%; $p < 0.01$), although the associated in-hospital mortality rates were similar (38.0% and 38.1%, respectively). Among hemodynamically stable (Killip classes I to III) patients on admission, in-hospital mortality associated with LM PCI (5.0%) was almost twice as high as in the non-LM PCI group (2.7%). Rates of reinfarction, cerebrovascular events, and bleeding were similar for LM and non-LM PCI. Subgroup analysis of patients treated for LM PCI showed higher in-hospital mortality rates (17.9% vs. 6.3%; $p < 0.001$) and higher rates of MACCE (18.6% vs. 8.3%; $p = 0.007$) in those treated with concurrent PCI of LM and other vessel segments (i.e., nonisolated LM) compared with isolated LM PCI. These differences remained significant even when the analysis was restricted to the subgroup of non-LM patients who underwent PCI of multiple vessels (mortality: 4.9%, rates of MACCE: 6.4%; $p < 0.001$ vs. LM PCI for both parameters). An additional subgroup analysis based on age showed approximately 2- and 4-fold increases in death rates in the LM PCI and non-LM PCI groups, respectively, for patients aged >75 years compared with younger ones. In elderly patients, however, death rates in LM PCI patients were still approximately 2-fold higher than in non-LM

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Table 1. Clinical and Angiographic Data at Baseline

	LM PCI (n = 348)	Non-LM PCI (n = 6,318)	p Value	Isolated LM PC (n = 208)	Nonisolated LM PCI (n = 140)	p Value
Demographics						
Age, yrs	63.5 ± 12.6	61.9 ± 12.5	0.021	61.7 ± 12.5	66.2 ± 12.3	0.001
Women	87/348 (25.0)	1,455/6,318 (23.0)	0.40	53/208 (25.5)	34/140 (24.3)	0.90
Clinical presentation						
Chest pain	295/332 (88.9)	5,572/6,130 (90.9)	0.21	181/202 (89.6)	114/130 (87.7)	0.60
Dyspnea	107/307 (34.9)	1,287/5,507 (23.4)	<0.001	58/183 (31.7)	49/124 (39.5)	0.18
Resuscitation before admission	37/348 (10.6)	400/6,318 (6.3)	0.004	16/208 (7.7)	21/140 (15.0)	0.034
Killip class I	241/344 (70.1)	5,408/6,279 (86.1)	<0.001	158/206 (76.7)	83/138 (60.1)	0.001
Killip class II	44/344 (12.8)	532/6,279 (8.5)	0.008	21/206 (10.2)	23/138 (16.7)	0.099
Killip class III	17/344 (4.9)	122/6,279 (1.9)	<0.001	7/206 (3.4)	10/138 (7.2)	0.13
Cardiogenic shock at admission	42/344 (12.2)	217/6,279 (3.5)	<0.001	20/206 (9.7)	22/138 (15.9)	0.09
Charlson weighted index ≥2	50/340 (14.7)	843/6,152 (13.7)	0.57	20/204 (9.8)	30/136 (22.1)	0.003
Times, min						
Symptom-to-admission	187 (91, 485)	180 (100, 390)	0.40	186 (88, 488)	210 (120, 420)	0.46
Median door-to-balloon	70 (23, 149)	60 (24, 240)	0.032	70 (22, 158)	69 (27, 143)	0.88
Risk factors						
Family history of CAD	94/300 (31.3)	1,884/5,539 (34.0)	0.35	56/184 (30.4)	38/116 (32.8)	0.70
Hypertension	174/318 (54.7)	3,215/5,909 (54.4)	0.95	97/192 (50.5)	77/126 (61.1)	0.067
Dyslipidemia	142/295 (48.1)	2,764/5,519 (50.1)	0.55	81/175 (46.3)	61/120 (50.8)	0.48
Diabetes	57/324 (17.6)	919/5,992 (15.3)	0.27	31/194 (16.0)	26/130 (20.0)	0.37
Smoking, current	126/314 (40.1)	2,691/5,748 (46.8)	0.023	79/190 (41.6)	47/124 (37.9)	0.56
Obesity, BMI >30 kg/m^2	61/265 (23.0)	1,065/5,367 (19.8)	0.21	38/166 (22.9)	23/99 (23.2)	0.99
Angiographic data						
1-vessel disease	—	2,788/6,290 (44.3)	—	—	9/131 (6.9)	—
2-vessel disease	—	1,882/6,290 (29.9)	—	—	63/131 (48.1)	—
3-vessel disease	—	1,620/6,290 (25.8)	—	—	59/131 (45.0)	—
Drug therapy						
Acetylsalicylic acid	337/346 (97.4)	6,205/6,306 (98.4)	0.19	203/206 (98.5)	134/140 (95.7)	0.17
Clopidogrel	301/346 (87.0)	5,872/6,294 (93.3)	<0.001	181/206 (87.9)	120/140 (85.7)	0.63
GP IIb/IIIa inhibitors	108/339 (31.9)	2,755/6,232 (44.2)	<0.001	70/203 (34.5)	38/136 (27.9)	0.24
Beta-blocker	207/344 (60.2)	4,132/6,238 (66.2)	0.023	133/207 (64.3)	74/137 (54.0)	0.072
ACE inhibitor	177/343 (51.6)	3,489 (56.2)	0.11	120/207 (58.0)	57/136 (41.9)	0.004
Calcium-channel blocker	9/340 (2.6)	297/6,183 (4.8)	0.07	3/204 (1.5)	6/136 (4.4)	0.16
Diuretic	62/343 (18.1)	944/6,212 (15.2)	0.17	30/204 (14.7)	32/139 (23.0)	0.063
HMG-CoA reductase Inhibitors	246/345 (71.3)	5,100/6,257 (81.5)	<0.001	159/206 (77.2)	87/139 (62.6)	0.004
Circulatory support						
IABP	48/343 (14.0)	400/6,244 (6.4)	<0.001	18/206 (8.7)	30/137 (21.9)	0.001
Vasopressor	56/341 (16.4)	641/6,194 (10.3)	0.001	181/206 (87.9)	120/140 (85.7)	0.63

Values are mean ± SD, n/N (%), or median (IQR). Data are shown for LM versus non-LM PCI and isolated LM versus nonisolated LM PCI.

ACE = angiotensin-converting enzyme; BMI = body mass index; CAD = coronary artery disease; GP = glycoprotein; HMG-CoA = 3-hydroxy-3-methylglutaryl coenzyme A; IABP = intra-aortic balloon pump; IQR = interquartile range; LM = left main; PCI = percutaneous coronary intervention.

PCI patients (p = 0.026). Similarly, elderly patients showed increased rates of MACCE, compared with younger ones, in both the LM PCI and the non-LM PCI groups.

Multivariable analysis. Results of multivariable logistic regression analysis are shown in Table 3. This analysis identified LM PCI, age, cardiogenic shock (Killip class IV), cardiac resuscitation, tachycardia, low systolic blood pressure, and an elevated Charlson weighted index as independent predictors of in-hospital mortality.

Discussion

PCI for unprotected LM disease has been performed with increasing frequency over the past decade. However, unprotected LM disease still poses a significant challenge to the interventional cardiologist, particularly with respect to primary PCI in patients with STEMI. Limited data are available on these patients. Here, we report on the largest series, to the best of our knowledge, of patients with STEMI treated with

Table 2. In-Hospital Outcomes

	LM PCI	Non-LM PCI	p Value	Isolated LM PCI	Nonisolated LM PCI	p Value
All patients	n = 348	n = 6,318		n = 208	n = 140	
In-hospital death	38/348 (10.9)	241/6,318 (3.8)	<0.001	13/208 (6.3)	25/140 (17.9)	0.001
In-hospital death, patients with CS at admission	23/42 (54.8)	77/217 (35.5)	0.024	11/20 (55.0)	12/22 (54.5)	1.0
MACCE, death, reinfarction, stroke	43/346 (12.4)	314/6,291 (5.0)	<0.001	17/206 (8.3)	26/140 (18.6)	0.007
CS developing during hospitalization	21/346 (6.1)	205/6,289 (3.3)	0.01	10/206 (4.9)	11/140 (7.9)	0.26
Reinfarction	4/346 (1.2)	56/6,289 (0.9)	0.55	3/206 (1.5)	1/140 (0.7)	0.65
Cerebrovascular events	5/346 (1.4)	42/6,289 (0.7)	0.10	2/206 (1.0)	3/140 (2.1)	0.40
Bleeding	10/346 (2.9)	180/6,289 (2.9)	0.87	3/206 (1.5)	7/140 (5.0)	0.097
Patients age ≤75 yrs	n = 274	n = 5,278		n = 174	n = 100	
In-hospital death	25/274 (9.1)	142/5,278 (2.7)	<0.001	10/174 (5.7)	15/100 (15.0)	0.015
In-hospital death, patients with CS at admission	17/33 (51.5)	60/182 (33.0)	0.049	8/15 (53.3.0)	9/18 (50.0)	1.0
MACCE, death, reinfarction, stroke	29/272 (10.7)	198/5,255 (3.8)	<0.001	13/172 (7.6)	16/100 (16.0)	0.041
CS developing during hospitalization	17/237 (7.2)	139/5,044 (2.8)	0.01	8/156 (5.1)	9/81 (11.1)	0.11
Reinfarction	4/272 (1.5)	39/5,255 (0.7)	0.16	3/172 (1.7)	1/100 (1.0)	1.00
Cerebrovascular events	4/272 (1.5)	32/5,255 (0.6)	0.10	1/172 (0.6)	3/100 (3.0)	0.14
Bleeding	5/272 (1.8)	138/5,255 (2.6)	0.56	2/172 (1.2)	3/100 (3.0)	0.36
Patients age >75 yrs	n = 74	n = 1,040		n = 34	n = 40	
In-hospital death	13/74 (17.6)	99/1,040 (9.5)	0.026	3/34 (8.8)	10/40 (25.0)	0.12
In-hospital death, patients with CS at admission	6/9 (66.7)	17/35 (48.6)	0.46	3/5 (60.0)	3/4 (75.0)	1.0
MACCE, death, reinfarction, stroke	14/74 (18.9)	116/1,036 (11.2)	0.059	4/34 (11.8)	10/40 (25.0)	0.23
CS developing during hospitalization	4/63 (6.3)	58/991 (5.9)	0.78	2/28 (7.1)	2/35 (5.7)	1.0
Reinfarction	0/74 (0)	17/1,034 (1.6)	0.62	0/34 (0)	0/40 (0)	
Cerebrovascular events	1/74 (1.4)	10/1,034 (1.0)	0.53	1/34 (2.9)	0/40 (0)	0.46
Bleeding	5/74 (6.8)	42/1,034 (4.1)	0.24	1/34 (2.9)	4/40 (10.0)	0.37

Values are n/N (%). Data are shown for LM versus non-LM PCI groups and isolated LM versus nonisolated LM PCI subgroups.
CS = cardiogenic shock; MACCE = major adverse cardiac and cerebrovascular event(s); other abbreviations as in Table 1.

primary LM PCI. Our results show that emergent LM PCI in the context of acute myocardial infarction, even including 12% cardiogenic shock, appears to have a remarkably high (89%) in-hospital survival. Nevertheless, patients with LM PCI had worse in-hospital outcomes than those treated with PCI of vessel segments other than LM. Of note, this difference persisted when the analysis was restricted to the subset of non-LM PCI patients with multivessel disease. By multivariable logistic regression analysis, LM disease was identified as an independent risk factor in patients with STEMI treated with primary PCI.

Table 3. Multivariable Analysis Models: Independent Predictors of In-Hospital Mortality in Patients With ACS Treated With Early PCI

Variable	Odds Ratio (95% CI)	p Value
Cardiogenic shock at admission	4.87 (2.84–8.35)	<0.001
Resuscitation	4.20 (2.60–6.77)	<0.001
Charlson index ≥2*	2.66 (1.76–4.02)	<0.001
Left main treated	2.36 (1.34–4.17)	0.003
Age, per 10-yr increase	1.96 (1.62–2.37)	<0.001
Heart rate, per 10 beats/min increase	1.23 (1.14–1.33)	<0.001
Systolic BP, per 5 mm Hg increase	0.88 (0.85–0.91)	<0.001

All covariates were assessed at admission. *The Charlson comorbidity index gives an estimate of survival based on the following variables: cerebrovascular disease, chronic pulmonary disease, congestive heart failure, connective tissue disease, dementia, hemiplegia, leukemia, malignant lymphoma, myocardial infarction, peripheral vascular disease, ulcer disease, diabetes mellitus, liver disease, renal disease, malignant solid tumor, and acquired immune deficiency syndrome status.
ACS = acute coronary syndrome(s); BP = blood pressure; CI = confidence interval; PCI = percutaneous coronary intervention.

An earlier report by Lee et al. (12) showed an 8% in-hospital mortality in 62 patients with ACS treated with LM PCI, compared with 10.9% in our patients with STEMI and LM PCI. Buszman et al. (16) reported a 12-month mortality of 6.3% in patients with non-STEMI treated with LM PCI. In a registry study, Montalescot et al. (13) reported an 11% in-hospital mortality in the subgroup of LM patients treated with PCI, very similar to the mortality rate in our study (10.9%). However, these earlier studies focused on patients with ACS (12,13) or non-STEMI (16), whereas our analysis was restricted to patients with STEMI. Moreover, in the registry study by Montalescot et al. (13), only 47% of the LM PCI patients underwent the procedure on the same day of symptom onset, whereas our analysis was restricted to patients treated with primary PCI. Another difference with this earlier study is that 92% of the LM PCI patients in this study had a concomitant disease of another coronary territory (13), whereas a majority of our LM PCI patients had isolated LM PCI.

Cardiogenic shock on admission, which was observed in more than 10% of our patients, was the main determinant of death in patients with STEMI treated with LM PCI, with an intrinsic mortality of approximately 50%. By contrast, mortality in hemodynamically stable LM PCI patients was nearly twice as high as in hemodynamically stable non-LM PCI patients, reflecting the prognostic impact of LM disease. With respect to the role of cardiogenic shock in this clinical setting, Hurtado et al. (15) reported a 66% prevalence of cardiogenic shock and a 61% in-hospital mortality in 71 consecutive patients treated with emergency PCI for LM disease. These data are in line with our results, highlighting the poor prognosis associated with cardiogenic shock (20) and the key role of an appropriate pharmacological and mechanical hemodynamic support, when needed.

In the present study, patients treated with concurrent PCI of LM and other vessel segments were at an ≈3-fold increased risk compared with those treated with isolated LM PCI. The latter were at a 2-fold increased risk compared with those treated with non-LM PCI. Interestingly, the prognostic role of isolated LM disease in patients with STEMI was moderate. By contrast, the prognostic impact of LM disease was greatest in the presence of additional lesions on other vessel segments. Of note, this subgroup of patients had a higher prevalence of multivessel disease, irrespective of LM lesions, compared with non-LM PCI patients, suggesting that LM disease might reflect an overall higher degree and a more widespread pattern of anatomical lesions in the former subgroup.

Not surprisingly, age was a major determinant of clinical outcomes. In patients age >75 years presenting with cardiogenic shock on admission, mortality was as high as 60% in patients with isolated LM PCI, and as high as 75% in those with nonisolated LM PCI, while being lower in younger patients. These results are of major interest because few studies have adequately described the impact of different treatment modalities in older patients with STEMI (21). Our results are in line with data from the SHOCK (Should We Emergently Revascularize Occluded Coronaries for Cardiogenic Shock?) trial (22) showing that older patients (age ≥75 years) presenting with AMI complicated by cardiogenic shock and treated with early revascularization have nearly double the 30-day mortality of younger patients (75.0% and 41.4%, respectively). Recent data clearly demonstrate the benefits of the invasive management of ACS in older patients (23).

Unprotected LM disease is a heterogeneous condition that includes various degrees of anatomic location and severity of LM lesions, and various possible sets of concurrent lesions of other coronary segments (24). In the AMIS Plus registry, patients were classified based on the vessel treated by PCI, rather than based solely on angiographic data, and the decision to perform PCI was at the discretion of the operator. Over the past 5 years, critical LM lesions in patients with STEMI have increasingly been managed invasively, more often with PCI than with coronary artery bypass graft surgery (24). Therefore, it seems reasonable to assume that, in patients with STEMI who underwent primary PCI and were enrolled in the AMIS Plus registry, very few LM stenoses, if any, were left untreated. It also should be emphasized that our analysis included a large subgroup of patients who underwent isolated LM PCI, unequivocally defining LM stenosis as the culprit lesion. Moreover, LM disease appeared to be associated with an increased risk even before undergoing primary PCI, as indicated by elevated resuscitation rates and a higher Charlson weighted index on admission.

Current guidelines (25) support PCI of non–infarct-related arteries during primary PCI in patients with STEMI and hemodynamic instability, whereas its role in hemodynamically stable patients remains more controversial (26,27). Our analysis provides important data on the outcomes of patients with STEMI undergoing simultaneous PCI of LM and other vessel segments, showing highest mortality and MACCE rates in this subgroup. Current strategies in patients with STEMI favor single-vessel acute PCI as the default approach (to treat only the infarct-related artery during the acute phase of STEMI), whereas acute multivessel PCI is restricted to exceptional patients with multiple critical (>90%) and potentially unstable lesions, and significant lesions of the noninfarct arteries being treated either medically or by staged revascularization procedures (28). Although these recommendations referred to multivessel disease in general, they appear to also apply to the management of non-LM lesions in the context of acute LM PCI.

Study limitations. Finally, a few limitations of our study, which are related to its nature of a registry study, must be acknowledged. Although the AMIS Plus registry provides data on the number of vessels treated with PCI, the precise anatomical distribution of the lesions is unknown. Clearly, it would have been of interest to know how LM lesions were distributed across the LM itself, including or not the bifurcation, and whether additional lesions were located on the right coronary artery versus the left anterior descending and/or the circumflex coronary artery. It also would have been of interest to know how many patients underwent LM PCI after having been rejected for coronary artery bypass graft surgery. Although we do not have precise data on this issue, these patients most likely constituted a small minority of all LM patients. An additional limitation is that patients who died before planned PCI were not included in the analysis.

Conclusions

Our data indicate that emergent LM PCI in the context of acute myocardial infarction has a remarkably high overall in-hospital survival, despite specific subsets of patients (including old, hemodynamically unstable patients, and

those undergoing concurrent LM and non-LM PCI) having a significantly increased risk.

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