Left Circumflex Occlusion in Acute Myocardial Infarction (from the National Cardiovascular Data Registry)

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Compared to occlusions of other major coronary arteries, patients presenting with acute left circumflex (LCx) occlusion usually have ST-segment elevation on the electrocardiogram <50% of the time, potentially delaying treatment and resulting in worse outcomes. In contemporary practice, little is known about the clinical outcomes of patients with LCx territory occlusion without ST-segment elevation myocardial infarction (STEMI). We identified patients with myocardial infarction from April 2004 to June 2009 in the CathPCI Registry treated with percutaneous coronary intervention for culprit LCx territory occlusion, excluding those with previous coronary artery bypass grafting. Logistic generalized estimating equation modeling was used to compare the outcomes, including in-hospital mortality between patients with STEMI and non-STEMI (NSTEMI) adjusting for differences in the baseline characteristics. Of the 27,711 patients with myocardial infarction and acute LCx territory occlusion, 18,548 (67%) presented with STEMI and 9,163 (33%) with NSTEMI. With the exception of a greater proportion of cardiac risk factors and cardiac history in the NSTEMI group, the demographic and baseline characteristics were clinically similar between the 2 groups, despite the statistical significance resulting from the large population. The patients with STEMI were more likely to have a proximal LCx culprit lesion (63% vs 27%, p <0.0001) and had greater risk-adjusted in-hospital mortality (odds ratio 1.36, 95% confidence interval 1.12 to 1.65, p = 0.002) compared to patients with NSTEMI. In conclusion, acute LCx territory occlusion often presents as NSTEMI, but patients with NSTEMI and occlusion have a lower mortality risk than those with STEMI, possibly because of factors such as the amount of myocardium involved, the lesion location along the vessel, and/or a dual blood supply. **(Am J Cardiol 2011;108:959–963)**

A 12-lead electrocardiogram (ECG) detects acute occlusions of the left anterior descending artery and right coronary artery in 70% to 92% of cases; however, the sensitivity for acute left circumflex artery (LCx) occlusion ranges from 32% to 48%,[1–4] likely because of its posterolateral location, which is farther from the chest wall, with a lack of corresponding electrocardiographic leads.[5–7] Patients presenting with non–ST-segment elevation myocardial infarction (NSTEMI) have better short-term outcomes because these infarcts are typically smaller. However, a subset of patients with NSTEMI might in fact be "STEMI-equivalents," with worse outcomes compared to patients presenting with ST-segment elevation due to delayed reperfusion in the NSTEMI group. In the present study, we assessed the incidence of acute occlusions in the LCx territory of patients who presented with NSTEMI and compared the outcomes between patients with STEMI and NSTEMI with acute LCx territory occlusion to determine whether acute occlusions of this territory in patients presenting with NSTEMI should be considered "STEMI-equivalents."

Methods

The present study was supported by the American College of Cardiology Foundation's National Cardiovascular Data Registry. The CathPCI Registry is a voluntary registry that receives data from >1,000 participating hospitals, including community, government, and university hospitals from rural, suburban, and urban areas throughout the United States. This is a standard data set with uniform data definitions, data entry and transmission requirements, and data quality checks. The details of the data collection process can be found at the National Cardiovascular Data Registry Web site (www.ncdr.com). For the purposes of the present study, we identified all patients with myocardial infarction who presented from April 2004 through June 2009 and underwent percutaneous coronary intervention (PCI) for culprit LCx territory occlusion. Because the acuity of the occluded lesions is not as well defined for patients with previous coronary artery bypass grafting (n = 2,951), these patients

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The views expressed in this report represent those of the authors and do not necessarily represent the official views of the American College of Cardiology Foundation's National Cardiovascular Data Registry, Washington, DC or its associated professional societies (available at http://www.ncdr.com).

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doi:10.1016/j.amjcard.2011.05.027

Table 1
Demographic and baseline clinical characteristics

Characteristic	STEMI (n = 18,548)	NSTEMI (n = 9,163)
Age (years)		
Median	58	58
Interquartile range	50–67	50–67
Men	13,916 (75.0%)	6,786 (74.1%)
Race		
White	15,236 (82.1%)	7,392 (80.7%)
Black	1,407 (7.6%)	856 (9.3%)
Hispanic	667 (3.6%)	308 (3.4%)
Other	1,200 (6.5%)	591 (6.5%)
Body mass index (kg/m^2)		
Median	28.3	28.9
Interquartile range	25.3–32.0	25.7–32.9
Previous myocardial infarction (>7 days)	2,472 (13.3%)	1,337 (14.6%)
Previous congestive heart failure	548 (3.0%)	414 (4.5%)
Diabetes mellitus	3,535 (19.1%)	2,011 (22.0%)
Previous renal failure	473 (2.6%)	232 (2.5%)
Cerebrovascular disease	995 (5.4%)	547 (6.0%)
Peripheral vascular disease	962 (5.2%)	526 (5.7%)
Hypertension	11,002 (59.3%)	5,936 (64.8%)
Smoker		
Former	3,715 (20.0%)	2,144 (23.4%)
Current	9,062 (48.9%)	4,111 (44.9%)
Dyslipidemia*	10,485 (56.5%)	5,735 (62.6%)
Previous percutaneous coronary intervention	2,536 (13.7%)	1,444 (15.8%)

*Documentation of total cholesterol >200 mg/dl, low-density lipoprotein ≥130 mg/dl, high-density lipoprotein <30 mg/dl, admission cholesterol >200 mg/dl, triglycerides >150 mg/dl, or initiation of treatment because of low-density lipoprotein level >100 mg/dl in patients with known coronary artery disease.

Table 2
Admission cardiac status stratified by myocardial infarction type

Cardiac Status	STEMI (n = 18,548)	NSTEMI (n = 9,163)	p Value
Symptom onset to admission (hours)			NA
≤6	15,354 (82.8%)	4,955 (54.1%)	
6–≤12	2,098 (11.3%)	2,077 (22.7%)	
12–≤24	1,096 (5.9%)	2,131 (23.3%)	
Congestive heart failure	1,756 (9.5%)	805 (8.8%)	0.0654
New York Heart Association class			<0.0001
I	4,063 (21.9%)	2,069 (22.6%)	
II	1,387 (7.5%)	908 (9.9%)	
III	2,911 (15.7%)	2,410 (26.3%)	
IV	10,185 (54.9%)	3,775 (41.2%)	
Cardiogenic shock	1,884 (10.2%)	377 (4.1%)	<0.0001

NA = not applicable.

were excluded from the analysis, yielding a final study population of 27,711 patients treated.

The patients with STEMI included those who presented within 24 hours of symptom onset with ST-segment elevation on the ECG and admission symptoms consistent with acute coronary syndrome. The patients with NSTEMI presented with admission symptoms consistent with acute coronary syndrome, symptom onset of ≤24 hours, and cardiac biomarkers of myonecrosis greater than the institutional upper limit of normal. Occlusion was defined as 100% preprocedure stenosis and Thrombolysis In Myocardial Infarction 0 or 1 flow in a LCx territory (defined as LCx or obtuse marginal artery) index lesion. The patients with STEMI who had undergone rescue or facilitated PCI with index lesions in the LCx territory and <100% stenosis or Thrombolysis In Myocardial Infarction flow >1 were also included. Because the data collection form for National Cardiovascular Data Registry did not capture the culprit lesion, for patients with multivessel PCI, the first lesion treated was assumed to be the infarct culprit lesion.

The patients were grouped according to the myocardial infarction type (STEMI vs NSTEMI). We compared the baseline demographic and patient characteristics, admission medications, cardiac status on admission, and catheterization laboratory data between the 2 groups. The Pearson chi-square test was used to compare categorical variables, and the Wilcoxon test was used for continuous variables. Logistic generalized estimating equation modeling was used to compare the in-hospital mortality between the patients with STEMI and NSTEMI, adjusting for differences in the baseline characteristics and variables in the National Cardiovascular Data Registry mortality model.[8]

We performed a secondary analysis of the patients with NSTEMI in an attempt to determine whether the prognosis associated with LCx occlusion is truly different from that associated with other coronary occlusions. We sampled all NSTEMI cases with 100% occlusions or Thrombolysis In Myocardial Infarction 0/1 flow as the culprit lesion during the same period and used logistic generalized estimating equation modeling to compare the in-hospital mortality for an occluded LCx culprit (n = 9,163) to occlusions in the left anterior descending artery (n = 7,093) and right coronary artery (n = 8,836) territories among patients with NSTEMI. All data analysis was performed by the National Cardiovascular Data Registry statistical analysis center at Duke Clinical Research Institute. A 2-tailed p value of ≤0.05 was considered significant.

Results

Of 27,711 consecutive patients without coronary artery bypass grafting who presented with STEMI or NSTEMI and underwent PCI for culprit LCx territory occlusion, 18,548 (67%) presented with STEMI and 9,163 (33%) with NSTEMI. Differences in the baseline and demographic characteristics between the 2 groups are listed in Table 1. The patients who presented with NSTEMI were more likely to have cardiac risk factors (ie, diabetes, dyslipidemia, and hypertension) and a history of coronary artery disease (previous myocardial infarction or PCI) than were the patients with STEMI.

No difference was found in the proportion of patients who presented with congestive heart failure between the 2 groups. However, patients with STEMI presented to the hospital earlier and were more likely to have cardiogenic shock and be in New York Heart Association class IV congestive heart failure on admission (Table 2). Compari-

Table 3
Admission medical management

Medication	STEMI (n = 18,548)	NSTEMI (n = 9,163)
Aspirin	16,759 (91.3%)	8,435 (92.8%)
β Blocker	12,295 (70.0%)	6,465 (73.2%)
Any glycoprotein IIb/IIIa inhibitor	14,036 (75.8%)	6,609 (72.2%)
Any low-molecular-weight heparin	2,045 (11.0%)	2,336 (25.5%)
Unfractionated heparin	13,481 (73.2%)	6,741 (74.2%)
Clopidogrel	13,507 (73.1%)	6,750 (74.1%)
Statins	5,544 (30.1%)	3,594 (39.5%)
Bivalirudin	3,066 (16.5%)	1,974 (21.6%)
Any thrombin inhibitor*	3,096 (16.7%)	1,982 (21.6%)

*Other than heparin products (i.e., bivalirudin, argatroban, lepirudin).

Table 4
Angiographic and procedural data

Catheterization Laboratory Data	STEMI (n = 18,548)	NSTEMI (n = 9,163)	p Value
Intra-aortic balloon pump	1,912 (10.3%)	513 (5.6%)	<0.0001
Multivessel disease	10,435 (56.3%)	5,307 (57.9%)	0.0049
Any drug-eluting stent	9,873 (53.2%)	5,230 (57.1%)	<0.0001
No stent	2,080 (11.2%)	1,296 (14.1%)	<0.0001
Preprocedure stenosis 100%	16,708 (90.1%)	9,163 (100%)	<0.0001
Previously treated lesion	865 (4.7%)	422 (4.6%)	0.8267
Percutaneous coronary intervention procedure success	16,937 (92.3%)	8,258 (90.6%)	0.3910

sons of the initial admission medical therapy among the groups are listed in Table 3. The patients with STEMI were more likely to receive glycoprotein IIb/IIIa inhibitors and unfractionated heparin ($p < 0.001$). The patients with NSTEMI were more likely to receive β blockers, statins, low-molecular-weight heparin, and thrombin inhibitors on admission ($p < 0.001$). The patients with STEMI were more likely to require insertion of an intra-aortic balloon pump during their hospitalization and were more likely to have a proximal lesion (lesion in the proximal portion of the LCx, obtuse marginal-1, or ramus arteries) than were patients with NSTEMI (62.8% vs 37.2%, $p < 0.0001$). The patients with NSTEMI were more likely to have multivessel disease and more frequently had a drug-eluting stent or no stent placed for their culprit lesion (Table 4). The success of the intervention was not different between the 2 groups. A small percentage (9.9%) of patients in the STEMI group had culprit lesions with stenosis <100%. This group represented those patients with STEMI who had undergone rescue or facilitated PCI.

Patients presenting with STEMI had greater unadjusted (5.5% vs 2.5%, $p < 0.0001$) and risk-adjusted (odds ratio 1.36, 95% confidence interval 1.12 to 1.65, $p = 0.002$) in-hospital mortality than those with NSTEMI. The patients with STEMI were also twice as likely to have in-hospital congestive heart failure (2.8% vs 1.5%, $p < 0.0001$) and cardiogenic shock (2.0% vs 1.2%, $p < 0.0001$) than the patients with NSTEMI. When analyzing the data of those who died, the patients with STEMI were more likely to die from cardiac causes (78.6% vs 70.5%, $p = 0.0005$) than those with NSTEMI (Table 5). In addition, other in-hospital adverse event rates such as bleeding and renal failure were greater for those with STEMI than those with NSTEMI (Table 5).

Table 5
Adverse in-hospital outcomes

Outcome	STEMI (n = 18,548)	NSTEMI (n = 9,163)	p Value
Mortality*	966 (5.5%)	224 (2.5%)	<0.0001
Cardiac death*†	759 (78.6%)	158 (70.5%)	0.0100
Cardiogenic shock	329 (2.0%)	103 (1.2%)	<0.0001
Congestive heart failure	4.66 (2.8%)	126 (1.5%)	<0.0001
Cerebrovascular accident	105 (0.6%)	40 (0.4%)	0.1506
Renal failure	248 (1.4%)	94 (1.1%)	0.0241
Any bleeding complication	890 (4.9%)	334 (3.7%)	<0.0001
Any vascular complication	153 (0.8%)	64 (0.7%)	0.2448
Any complication	2,193 (12.0%)	753 (8.3%)	<0.0001
Blood products after percutaneous coronary intervention	1,514 (8.3%)	613 (6.7%)	<0.0001

*Data excluded 1,116 patients (4.0%) who were transferred.
† Data not adjudicated in registry and might be inaccurate.

In a secondary analysis of all patients with NSTEMI with an occluded culprit lesion, those with LCx territory culprits had similar risk-adjusted mortality as those with left anterior descending artery culprits (odds ratio 0.90, 95% confidence interval 0.72 to 1.12, $p = 0.323$). In contrast, an right coronary artery culprit had a lower risk-adjusted mortality (odds ratio 0.74, 95% confidence interval 0.58 to 0.94, $p = 0.013$) compared to a left anterior descending artery culprit.

Discussion

In the present retrospective study analyzing a large, national cohort of patients with myocardial infarction undergoing PCI for LCx territory culprit occlusions, 67% presented with STEMI, greater than that in previous reports. STEMI secondary to LCx territory occlusion conferred a greater risk than NSTEMI, which might reflect the more proximal location of the occluded lesion resulting in larger infarcts, a greater risk of hemodynamic complications, and increased mortality.

Previous reports have indicated that ST-segment elevation in LCx territory occlusion are detected on the ECG in <50% of cases.[1–4,9] It is possible that the greater incidence of STEMI in the present study could be attributed to physician vigilance, with additional diagnostic modalities, including earlier angiography, additional electrocardiographic leads, or bedside echocardiography in trying to detect occlusions not apparent on the 12-lead ECG. Patients might have also been retrospectively classified as having STEMI after angiography revealed an occlusion. Furthermore, the inclusion of only PCI patients might have underestimated the true percentage of patients with LCx territory occlusion who present with NSTEMI because that criterion

eliminated patients with NSTEMI and LCx territory occlusion who were treated medically or died before PCI.

Previous studies of patients with NSTEMI have suggested that occluded culprit lesions are associated with worse outcomes than nonoccluded culprit lesions. Wang et al[5] determined that NSTEMI secondary to acute occlusion of any coronary artery conferred larger infarcts and greater risk-adjusted 6-month mortality than nonoccluded culprit lesions, similar to that of STEMI, and termed these cases "STEMI equivalents." In a previous study of the National Cardiovascular Data Registry, Dixon et al[6] also found that NSTEMI with an occluded culprit vessel was associated with greater rates of in-hospital mortality, cardiogenic shock, and congestive heart failure compared to NSTEMI without occlusion. In a subanalysis of the TRial to assess Improvement in Therapeutic Outcomes by optimizing platelet InhibitioN with prasugrel Thrombolysis In Myocardial Infarction 38 (TRITON-TIMI 38) study examining patients who presented with isolated anterior ST-segment depressions on the ECG, many were initially misdiagnosed as having NSTEMI, leading to a significant delay in PCI. Those who were misdiagnosed because of a lack of ST-segment elevation had greater recurrent myocardial infarction rates with larger infarcts and increased 30-day mortality.[10] A review by Krishnaswamy et al[11] also noted that a disproportionate number of patients with non–ST-segment elevation acute coronary syndrome with LCx occlusion and large infarcts enrolled in the Framingham and Fast Revascularization During Instability in Coronary Artery Disease (FRISC) II trial had worse 12-month outcomes,[12] and patients who had myocardial infarction due to LCx occlusion benefited equally from early reperfusion whether they had ST-segment elevation on the ECG or not.[9,11] Collectively, these studies have shown that patients with NSTEMI and occlusion have worse outcomes than those without occlusion. Also, in many of these studies, the occluded lesions were more frequently in the inferoposterior (likely LCx) territory. Our study took the next step by comparing patients with occluded LCx territory who presented with and without ST-segment elevation and showed that the risk of the patients with NSTEMI remained significantly lower than that of those with STEMI. A smaller, retrospective study at our institution revealed similar findings.[13]

Our findings of better in-hospital outcomes in patients with culprit LCx territory occlusion and NSTEMI compared to STEMI suggests that the term "STEMI equivalent" does not apply to all patients with NSTEMI and culprit vessel occlusion. Our secondary analysis comparing the adjusted mortality for occlusion of each major coronary in those with NSTEMI found no significant difference between the left anterior descending artery and LCx, and those patients with right coronary artery occlusion had lower mortality. Although ST-segment elevation might be more likely to occur in certain coronary arteries, we did not find evidence to suggest that the outcomes for patients with NSTEMI secondary to culprit occlusion were worse for the right coronary artery or left anterior descending artery territories than for the LCx territory.

The outcome results of our study support conventional knowledge that patients with STEMI have worse in-hospital outcomes than those with NSTEMI, rather than the hypothesis that NSTEMI secondary to culprit vessel occlusion is equivalent to STEMI. The electrocardiographic findings or initial clinical presentation seemed more predictive of the outcome than the angiographic findings. However, the lack of characteristic ST-segment elevation on the ECG in certain patients with LCx territory occlusion was not solely attributable to the insensitivity of the 12-lead ECG, because other factors such as the myocardium at risk or the lack of dual blood supply might be important in determining whether patients with LCx territory occlusion have ST-segment elevation on ECG.

The patients with STEMI in our study likely had worse outcomes because they had more myocardium at risk, and those who presented with NSTEMI had occlusions involving smaller coronary arteries that supplied relatively small amounts of myocardium. In a pathologic study of fatal infarcts, the size of the infarct was directly related to the ischemic bed size.[14] That the patients in our study with proximal LCx territory occlusion were much more likely to present with STEMI would be consistent with this theory.

Coronary dominance could also play a role. The prevalence of right coronary artery dominance in the population might be protective for acute occlusions of the LCx territory by providing collateral or dual flow, minimizing infarct size, and reducing the likelihood of having ST-segment elevation on the ECG. Similarly, patients with acute occlusions of a nondominant, small right coronary artery might not have typical ST-segment elevation on the inferior electrocardiographic leads.[15,16] Coronary dominance was not analyzed in our study because this variable was not collected in the registry during this study period. However, a recent study of 96 patients with myocardial infarction secondary to a LCx lesion found that ST-T changes on the ECG were significantly less likely to occur in patients with right or mixed coronary dominance.[17] We have also previously documented that patients with left or mixed coronary dominance were more likely to present with STEMI.[13]

The present study had several limitations not previously mentioned. It was subject to the differences in diagnostic and management strategies and clinical protocols among the different hospitals and clinicians. Because of the nature of the National Cardiovascular Data Registry, only in-hospital data were available. Therefore, we were unable to follow mortality longitudinally after discharge and did not have the mortality data for the small percentage of patients who were transferred. Although it can be assumed that the patients with STEMI had shorter revascularization times because of the earlier time to presentation and standard of care, the door-to-balloon times for the patients with NSTEMI were not available in the registry. Thus, we were unable to provide a comparison. The National Cardiovascular Data Registry does not have data for individual patients' electrocardiographic findings. Therefore, the diagnosis of STEMI or NSTEMI was determined by physician documentation and was subject to electrocardiographic interpretation and coding biases, making retrospective diagnosis of acute occlusions possible. We were also unable to analyze specific electrocardiographic findings that might be predictive of LCx territory occlusion or poor outcomes. As previously mentioned, we were unable to perform an analysis of cor-

onary dominance owing to a lack of data from earlier stages of the registry.

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