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Outcomes of Primary Percutaneous Coronary Intervention in ST-Segment Elevation Myocardial Infarction Patients With Previous Coronary Bypass Surgery

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ABSTRACT

OBJECTIVES This study sought to determine the contemporary clinical characteristics and outcomes of patients with ST-segment elevation myocardial infarction (STEMI) and previous coronary artery bypass graft (CABG), including those with a saphenous vein graft culprit lesion.

BACKGROUND The outcome of STEMI patients with previous CABG is reported to be inferior to those without previous CABG, but limited data is available from the primary percutaneous coronary intervention era.

METHODS Data was extracted from a large, regional STEMI system's prospective database, which contained 3,542 unique STEMI episodes from March 4, 2003 through April 22, 2012.

RESULTS Previous CABG was present in 249 patients (7%). Despite higher comorbidity, patients with versus those without previous CABG had similar in-hospital (4.8% vs. 5.2%; $p = 0.82$) and 1-year (10.8% vs. 9.1%; $p = 0.36$) mortality, but 5-year (24.9% vs. 14.2%; $p < 0.001$) mortality was higher. Patients with previous CABG have similar door-to-balloon times. The culprit vessel was the saphenous vein graft in 84 patients (34%), a native vessel in 104 (42%), with no clear culprit in 59 (24%). The left internal mammary artery graft was not a culprit in any patient. Mortality at 30 days (8.3% vs. 3.9% vs. 1.7%, $p = 0.19$) and 1 year (14.3% vs. 9.0% vs. 6.8%; $p = 0.35$) was higher (but not statistically) with a saphenous vein graft culprit and was equivalent at 5 years (25.0% vs. 26.0% vs. 20.3%; $p = 0.71$).

CONCLUSIONS Patients with previous CABG treated in a regional STEMI system have similar outcomes as patients without previous CABG, although 5-year mortality is higher. The most common culprit location was a native vessel (42%). Outcomes have improved significantly compared with historical reports. (J Am Coll Cardiol Intv 2014;7:981–7)

The presence of previous coronary artery bypass graft (CABG) in a patient who presents with acute chest pain and possible ST-segment elevation myocardial infarction (STEMI) can pose a diagnostic and therapeutic challenge. Baseline electrocardiogram (ECG) morphology, coronary artery, and bypass graft anatomy are frequently abnormal and unknown. Current guidelines advocate rapid activation of the catheterization lab or emergency transport to a facility capable of performing percutaneous coronary intervention (PCI), which may preclude thorough review of previous ECG and operative reports (1). Reports have described higher rates of "false-positive" activation of the cardiac catheterization lab (2), an increased burden of comorbid illness, and worse clinical outcomes among patients with previous CABG. In fact, previous CABG has been reported to be an independent risk factor for adverse clinical outcomes in patients with STEMI (3–5).

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ABBREVIATIONS AND ACRONYMS

CABG = coronary artery bypass graft

ECG = electrocardiogram

IRA = infarct-related artery

MACE = major adverse cardiac outcomes

MI = myocardial infarction

PCI = percutaneous coronary intervention

STEMI = ST-segment elevation myocardial infarction

SVG = saphenous vein graft

TIMI = Thrombosis In Myocardial Infarction

Patients with previous CABG compose a small percentage of all patients who present with STEMI, and this subset of patients has not been well characterized. In STEMI patients with previous CABG undergoing primary balloon angioplasty, the in-hospital and 6-month mortality was increased compared with that of patients without previous CABG, especially when the culprit was a saphenous vein graft (SVG) (6). Bare-metal stent placement was subsequently demonstrated to be superior to balloon angioplasty, with respect to major adverse cardiac outcomes (MACE), in the treatment of obstructed venous bypass grafts (7). Drug-eluting stents were then shown to be superior to bare-metal stents in the treatment of SVG lesions (8). Previous reports, including post-hoc analyses of randomized clinical trials, have described lower rates of primary PCI and/or inferior clinical outcomes after STEMI in patients with previous CABG, especially those with a SVG culprit lesion (9–13).

Based on available data, STEMI in the setting of previous CABG or STEMI with the presence of culprit lesion in a bypass graft may confer an increased rate of morbidity and mortality. However, the literature remains limited and is confounded by lower rates of intervention among those with previous CABG. In addition, significant advancements have been made in stent design, adjunctive pharmacology, and development of regional STEMI systems. Therefore, our goal was to determine the contemporary outcome of patients with previous CABG who presented with STEMI.

METHODS

The Minneapolis Heart Institute at Abbott Northwestern Hospital in Minneapolis, Minnesota, is a tertiary cardiovascular center with established referral relationships with community hospitals throughout Minnesota and western Wisconsin. In 2003, a regional system for the management of STEMI, the "Level 1" MI program, was initiated with a standardized protocol for transfer of STEMI patients for primary or pharmacoinvasive PCI from 31 community hospitals up to 210 miles (338 km) from the PCI center. The detailed design and results of the Level 1 MI program have been previously reported (14–16). All patients with STEMI or new left bundle branch block within 24 h of symptom onset are included in the Level 1 MI program. No patients were excluded from the protocol unless a cardiology physician felt that reperfusion therapy was inappropriate (e.g., advanced dementia or widely metastatic cancer). All patients, including those with advanced age, out-of-hospital cardiac arrest, and cardiogenic shock were included in the data analysis (15). All patients included in the database received emergent coronary angiography unless they expired during transport to the cardiac catheterization laboratory. Institutional review board approval was obtained, including waiver of consent for in-hospital data collection, data analysis, and follow-up up to 5 years.

Using a comprehensive prospective database from 3,542 consecutive patients treated as part of the Level 1 MI regional STEMI program from March 4, 2003 through April 22, 2012, we examined demographic information; cardiovascular risk factors; cardiac medications at presentation; clinical condition at presentation; time to treatment; angiographic findings; cardiac biomarkers during hospitalization; and short- and long-term outcomes, which included mortality (in-hospital, 30-day, 1-year, and 5-year) and 30-day cardiac readmission, reinfarction or recurrent ischemia, stroke, or combined MACE.

Patients with previous CABG were further stratified to 3 groups by culprit lesion based on ECG and angiographic findings: 1) SVG culprit; 2) native vessel culprit; or 3) no clear culprit. Patients with no culprit artery identified were stratified by the presence or absence of positive cardiac biomarkers (**Figure 1**).

Descriptive statistics were reported as mean ± SD; categorical variables as number and percentage with characteristic. Where continuous variables had skewed distributions (length of stay, door-to-balloon times), median (25th and 75th percentiles) were reported. Continuous variables were assessed using analysis of variance or Student *t*-test, and categorical variables using chi-square or Fisher exact tests. Kruskal-Wallis rank tests were used to compare continuous variables with skewed distribution. A p value of <0.05 was considered statistically significant and p values are 2-sided whenever possible. All analyses were performed with Stata (version 11.2, StataCorp, College Station, Texas).

RESULTS

Of 3,542 consecutive patients with STEMI, 249 (7%) had a history of previous CABG. These patients were older, more likely male, with increased prevalence of previous MI, PCI, and some cardiovascular risk factors, including diabetes mellitus, but with lower current smoking rates (**Table 1**). The incidence of anterior MI was higher in patients without previous CABG (35.5% vs. 23.6%; $p < 0.001$), which is likely due to the presence of left internal mammary artery grafts. There was

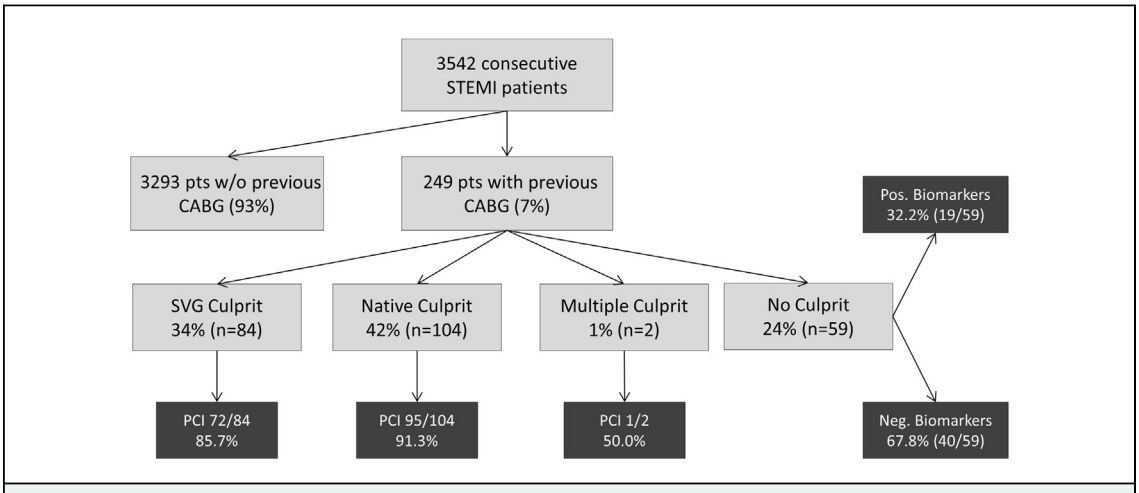

FIGURE 1 Study Flow Diagram

Stratification of patients from the Level 1 MI project database for the current study. The absolute number and percentage are included for each group. CABG = coronary artery bypass graft; Neg = negative; PCI = percutaneous coronary intervention; Pos = positive; Pts = patients; STEMI = ST-segment elevation myocardial infarction; SVG = saphenous vein graft.

no difference in pre-PCI cardiogenic shock, out of hospital cardiac arrest, or Killip class at the time of presentation. Use of cardiac medications, including aspirin, beta-blockers, angiotensin-converting enzyme inhibitors, and 3-hydroxy-3-methylglutaryl coenzyme A reductase inhibitors (statins), was higher among patients with previous CABG (Table 1).

Time to intervention was similar in patients with and without previous CABG (Table 2). Patients without previous CABG were more likely to demonstrate decreased flow (TIMI [Thrombolysis In Myocardial Infarction] flow grade 0 or 1) in the culprit artery, but they were noted to have equivalent post-procedural flow. Primary PCI was performed on 67.5% of the patients in the previous CABG group, as compared to 79.8% of those without previous CABG ($p < 0.001$). The likelihood of finding "no clear culprit" was higher in patients with previous CABG (23.7% vs. 15.4%, $p = 0.001$).

Among patients with previous CABG versus those without, mean peak creatine kinase-myocardial band level was significantly lower (38 vs. 69, $p < 0.001$), and the left ventricular ejection fraction measured during hospitalization was lower (45.2% vs. 47.3%, $p = 0.02$), likely reflecting the higher prevalence of previous MI. There was no significant difference in median length of stay or in-hospital death (Table 2). MACE at 30 days (including stroke, recurrent infarction, recurrent ischemia, or death) and rates of hospital readmission at 30 days for cardiac causes were equivalent in the 2 groups. At 1 year, no significant

TABLE 1 Characteristics of All Patients Split by History of Previous CABG

	Previous CABG (n = 249)	No Previous CABG (n = 3,303)	p Value
Age, yrs	69.4 ± 12.0	62.1 ± 14.3	<0.001
Male	203 (81.5)	2,325 (70.6)	<0.001
Hypertension	207 (83.1)	1,859 (56.7)	<0.001
Dyslipidemia	216 (87.5)	1,679 (52.4)	<0.001
Diabetes	88 (35.5)	519 (15.8)	<0.001
History of CAD	249 (100)	810 (24.6)	<0.001
Family history of CAD	98 (42.1)	1354 (43.1)	0.76
History of smoking	146 (59.4)	1,996 (61.0)	0.60
Current smoker*	47 (32.2)	1,233 (61.8)	<0.001
Previous MI	161 (66.0)	533 (16.2)	<0.001
Previous PCI	136 (55.5)	593 (18.0)	<0.001
History of stroke	10 (4.1)	80 (2.5)	0.13
Cardiogenic shock pre-PCI	20 (8.0)	279 (8.5)	0.81
Cardiac arrest pre-PCI	15 (6.0)	296 (9.0)	0.11
Anterior MI	57 (23.6)	1,155 (35.5)	<0.001
Killip class			1.00
1	217 (87.2)	2,868 (87.2)	
2-4	32 (12.9)	423 (12.9)	
Admission medications†	142	1,839	
Aspirin	111 (78.2)	667 (36.3)	<0.001
Beta-blocker	98 (69.0)	499 (27.2)	<0.001
ACE-inhibitor	70 (49.3)	399 (21.7)	<0.001
Statin	95 (66.9)	517 (28.1)	<0.001
Antiplatelet	29 (20.4)	133 (7.2)	<0.001

Values are mean ± SD, n (%), or n. *Percentage of current smokers based on the number of patients with a positive history of smoking. †Admission medications only available for patients presenting after April 1, 2009.

ACE = angiotensin-converting enzyme; CABG = coronary artery bypass graft; CAD = coronary artery disease; MI = myocardial infarction; PCI = percutaneous coronary intervention

TABLE 2 Angiographic and Clinical Outcomes of All Patients Split by History of Previous CABG

	Previous CABG (n = 249)	No Previous CABG (n = 3,303)	p Value
No clear culprit artery	59 (23.7)	507 (15.7)	0.001
TIMI flow grade pre-PCI			0.029
0/1	96 (39.8)	1,519 (47.1)	
2/3	145 (60.2)	1,707 (52.9)	
TIMI flow grade post-PCI			0.19
0/1	8 (3.3)	66 (2.1)	
2/3	233 (96.7)	3,158 (98.0)	
Peak CK	397 (159–1,103)	726 (241–1,664)	<0.001
Peak CK-MB	38 (9–105)	69 (19–170)	<0.001
Ejection fraction, %	45.2 ± 14.4	47.3 ± 13.3	0.021
Primary PCI performed	168 (67.5)	2,628 (79.8)	<0.001
Length of stay, days	3 (2–4)	3 (2–4)	0.61
Door to balloon,* min	98 (76–122)	95 (74–125)	0.39
Death in-hospital	12 (4.8)	170 (5.2)	0.82
Outcomes at 30 days			
Cardiac readmission†‡	4 (1.7)	72 (2.3)	0.82
Stroke†‡	2 (0.8)	26 (0.8)	1.00
Reinfarction/reischemia†‡	3 (1.3)	41 (1.3)	1.00
Death	12 (4.8)	194 (5.9)	0.49
MACE	17 (6.8)	242 (7.4)	0.76
Outcomes at 1 year			
Cardiac readmission‡	21 (8.9)	205 (6.6)	0.17
Stroke†‡	5 (2.1)	34 (1.1)	0.19
Reinfarction/reischemia‡	10 (4.2)	87 (2.8)	0.20
Death	27 (10.8)	301 (9.1)	0.36
MACE	39 (15.7)	403 (12.2)	0.12
Death at 5 years	62 (24.9)	466 (14.2)	<0.001

Values are n (%), median (interquartile range), or mean ± SD. *Door-to-balloon time represents the total time from presentation at a referral emergency department or hospital to the first device at the PCI hospital, including time for transport to Abbott Northwestern Hospital. Patients who expired prior to coronary angiography are included in the database for calculation of mortality, but they are not assigned a door to balloon time. †Fisher exact test used to assess statistical significance. ‡Percentage based on those patients alive at discharge.

CK = creatine kinase; CK-MB = creatine kinase-myocardial band; MACE = major adverse cardiac events (death, stroke, recurrent ischemia, or infarction); TIMI = Thrombolysis In Myocardial Infarction.

mortality differences were observed (p = 0.36), but there was an increased rate of death at 5 years among those with CABG (24.9% vs. 14.2%, p < 0.001).

RESULTS BASED ON CULPRIT LESION. Patients with CABG as stratified by culprit lesion (SVG, native vessel, no culprit), are compared in Table 3. Among patients with previous CABG, the culprit lesion was identified as a SVG in 84 patients (34%) and as a native vessel in 104 (42%). No clear culprit was identified in 59 patients (24%) and 2 patients (1%) were found to have native and SVG coculprit lesions. Among patients with no clear culprit artery on coronary angiography, 19 of 59 (32.2%) had elevated biomarkers. The 2 patients with multiple culprits were excluded from further analysis. The left internal mammary artery was not identified as the culprit artery in any case.

Patients with a native vessel culprit were less likely to report a history of smoking but were more likely to be current smokers and had lower rates of treatment with beta-blockers or angiotensin-converting enzyme inhibitors at the time of admission. Patients with a SVG culprit lesion, as compared to those with a native vessel culprit, had increased rates of pre-PCI shock (14.3% vs. 4.8%, p = 0.038). Patients without clear culprit lesions were more likely to present with anterior MI by ECG.

The angiographic and clinical outcomes of the patients with CABG, as stratified by culprit lesions, are displayed in Table 4. Door-to-balloon times are comparable in the 3 groups. As expected, patients without a clear culprit had normal TIMI flow and did not undergo PCI. In contrast with previous reports, there was no difference in the rate of intervention between SVG culprit (85.7%) and native vessel culprit (91.4%) groups (p = 0.22). Mortality in patients with SVG culprit was numerically, but not statistically, different from those with native vessel or no culprit (in-hospital mortality: 8.3% vs. 3.9% vs. 1.7%, p = 0.19; 1-year mortality: 14.3% vs. 9.6% vs. 6.8%, p = 0.35). There were no differences in length of stay, readmission for cardiac causes at 30 days, or MACE at 30 days.

DISCUSSION

This report represents the largest cohort of patients with previous CABG undergoing primary PCI for STEMI. Notably, 7% of the current study population had previous CABG, which represents an increased prevalence when compared with previous reports (2.2% to 5.3%). In patients with previous CABG, the culprit artery was more frequently a native vessel (42%) than a SVG (34%), and these patients had a greater burden of medical comorbidities. The left internal mammary artery graft was never identified as a culprit vessel, which may explain the lower incidence of anterior MI and is a further testament to the durability of this graft. In this prospectively collected registry involving a large number of patients, we did not identify increased rates of mortality at 30 days or 1 year after STEMI in those with previous CABG. This observation contrasts with previous reports and may represent the influence of the regional STEMI system using a standardized protocol with aggressive evidence-based therapy, including timely PCI. Mortality in the subgroup of patients with a SVG culprit vessel was numerically, but not statistically, higher when compared with that of patients with a native vessel culprit or no clear culprit.

Previous studies have suggested increased mortality after STEMI in those with previous CABG, especially among those with a SVG culprit vessel, and this greater mortality may reflect a combination of inadequate adjunctive medical therapy, older stent technology, and lower rates of reperfusion or PCI among those with previous CABG. In the report of Stone et al. (6) from the PAMI-2 (Second Primary Angioplasty in Myocardial Infarction Study) (1993 to 1995), a significantly lower rate of balloon angioplasty and post-procedural TIMI flow was achieved in 58 patients with previous CABG. In-hospital death was increased among those with previous CABG and those with SVG culprit lesions. Notably, coronary stenting and dual antiplatelet medications were not standard at the time. Similarly, Brodie et al. (17) report inferior short- and long-term outcomes of 57 patients with SVG culprit lesions treated with percutaneous transluminal coronary angioplasty and early coronary stents from 1984 to 2003. As was the case in the previous study, significantly fewer patients with SVG culprit lesions were noted to have post-procedural TIMI flow grade 3. These patients were reported to have significantly increased in-hospital and long-term mortality when compared with all patients with a native vessel culprit (including 50 patients with prior bypass).

Only 2 contemporary reports have been published involving randomized clinical trials of patients with previous CABG and STEMI treated with PCI. The first is a post-hoc analysis of the APEX-AMI (Assessment of Pexelizumab in Acute Myocardial Infarction) trial in which 128 study participants (2.2%) had undergone previous CABG (10). In patients with previous CABG, successful reperfusion was less likely, and 90-day mortality was significantly greater. The 90-day mortality rate among patients with a SVG infarct-related artery (IRA) (19.0%) was significantly increased after multivariate analysis (hazard ratio: 3.3) when compared with native IRA in patients with previous CABG (5.7%, $p = 0.031$) or a native IRA in patients without previous bypass (4.6%, $p < 0.01$). The outcomes of patients with a native IRA, regardless of bypass status, are similar to our experience. Among those with a SVG culprit, we report lower rates of morality, despite an excess of shock on presentation, and statistically noninferior clinical outcomes. The excess mortality reported from APEX-AMI may represent type I error given the limited sample size or may be related to the decreased rates of post-procedural TIMI grade 3 flow in infarct-related bypass grafts due to lower rates of primary PCI. The second contemporary report is a post-hoc analysis of the HORIZONS-AMI (Harmonizing Outcomes With Revascularization and Stents in Acute Myocardial Infarction) trial, which found increased MACE at 3 years among 105 patients with previous bypass, regardless of IRA (6). This result was driven by significantly higher rates of target vessel revascularization and stroke. A trend toward increased mortality did not reach statistical significance. These findings are confounded by longer door-to-balloon times, lower rates of referral to primary PCI, and inferior post-PCI TIMI flow grades among those with previous CABG. After multivariate analysis, neither previous CABG nor SVG culprit was associated with increased 3-year mortality or MACE.

In our study, the rate of primary PCI among patients with previous CABG was lower, albeit to a smaller degree, when patients with no clear culprit artery are censored (88.4% vs. 95.4%, $p < 0.001$). There were, however, no significant differences in post-PCI TIMI flow grades between the 2 groups, as was described in previous studies, suggesting that clinical outcomes may be equalized if comparable reperfusion is achieved with current therapies.

TABLE 3 Characteristics of Patients With Previous CABG Split by Culprit Lesion

	Culprit SVG (n = 84)	Native Culprit (n = 104)	No Culprit (n = 59)	p Value
Age, yrs	69.2 ± 11.7	69.4 ± 12.0	69.1 ± 12.5	0.99
Male	70 (83.3)	86 (82.7)	46 (78.0)	0.68
Hypertension	71 (84.5)	80 (76.9)	54 (91.5)	0.052
Dyslipidemia	71 (84.5)	93 (90.3)	51 (87.9)	0.49
Diabetes	30 (35.7)	32 (31.1)	25 (42.4)	0.35
Family history of CAD	32 (41.0)	38 (38.8)	27 (49.1)	0.45
History of smoking	53 (63.1)‡	49 (48.0)‡	43 (74.1)‡	0.004
Current smoker*	17 (32.1)	18 (36.7)	11 (25.6)	0.52
Previous MI	52 (62.7)	69 (67.7)	40 (70.2)	0.62
Previous PCI	44 (52.4)	57 (56.4)	34 (58.6)	0.74
History of stroke†	3 (3.6)	4 (3.9)	3 (5.2)	0.84
Cardiogenic shock pre-PCI	12 (14.3)‡	5 (4.8)‡	3 (5.1)‡	0.038
Cardiac arrest† pre-PCI	4 (4.8)	7 (6.7)	4 (6.8)	0.84
Anterior MI	13 (15.7)‡	23 (22.6)‡	21 (38.2)‡	0.009
Killip class				0.17
0/1	69 (82.1)	95 (91.4)	51 (86.4)	
2/4	15 (17.9)	9 (8.7)	8 (13.6)	
Admission medications§	43	69	30	
Aspirin	36 (83.7)	49 (71.0)	26 (86.7)	0.13
Beta-blocker	33 (76.7)‡	40 (58.0)‡	25 (83.3)‡	0.018
ACE inhibitor	21 (48.8)‡	28 (40.6)‡	21 (70.0)‡	0.027
Statin	30 (69.8)	41 (59.4)	24 (80.0)	0.12
Antiplatelet	8 (18.6)	13 (18.8)	8 (26.7)	0.63

Values are mean ± SD, n (%), or n. *Percentage of current smokers based on the number of patients with a positive history of smoking. †Fisher exact test used to assess statistical significance. ‡Superscripts that are the same do not significantly differ ($p > 0.05$). §Admission medications only available for patients presenting after April 1, 2009.

SVG = saphenous vein graft, other abbreviations as in Table 1.

TABLE 4 Angiographic and Clinical Outcomes of Patients With Previous CABG Split by Culprit Lesion

	Culprit SVG (n = 84)	Native Culprit (n = 104)	No Culprit (n = 59)	p Value
TIMI flow grade pre-PCI*				<0.001
0/1	47 (56.6)†	49 (48.0)†	0 (0)†	
2/3, (%)	36 (43.4)	53 (52.0)	54 (100)	
TIMI flow grade post-PCI*				0.061
0/1	6 (7.2)	2 (2.0)	0 (0)	
2/3	77 (92.8)	100 (98.0)	54 (100)	
Peak CK	571 (216, 1,480)†	573 (260, 1,337)†	139 (79, 275)†	<0.001
Peak CK-MB	46 (20, 113)†	65 (18, 139)†	7 (7, 15)†	<0.001
Ejection fraction, %	44.8 ± 13.7	44.8 ± 14.9	46.8 ± 14.9	0.65
Primary PCI performed	72 (85.7)†	95 (91.4)†	0 (0)†	<0.001
Length of stay, days	3 (2, 5)	3 (2, 4)	2 (1, 5)	0.23
Door to balloon,‡ min	95 (75, 119)	98 (74, 119)	106 (83, 129)	0.40
Death in-hospital*	7 (8.3)	4 (3.9)	1 (1.7)	0.19
Outcomes at 30 days				
Cardiac readmission*§	1 (1.3)	3 (3.0)	1 (1.7)	0.85
Stroke*§	1 (1.3)	0 (0)	1 (1.7)	0.33
Reinfarction/reischemia*§	2 (2.6)	1 (1.0)	0 (0)	0.61
Death*	7 (8.3)	4 (3.9)	1 (1.7)	0.19
MACE*	10 (11.9)	5 (4.8)	2 (3.4)	0.11
Outcomes at 1 year				
Cardiac readmission*§	10 (13.0)	9 (9.0)	2 (3.5)	0.18
Stroke*§	2 (2.6)	2 (2.0)	1 (1.7)	1.00
Reinfarction/reischemia*§	3 (3.9)	7 (7.0)	0 (0)	0.099
Death*	12 (14.3)	10 (9.6)	4 (6.8)	0.35
MACE*	8 (10.4)	14 (14.0)	4 (6.9)	0.40
Death at 5 years*	21 (25.0)	27 (26.0)	12 (20.3)	0.71

Values are n (%), median (IQR), or mean ± SD. *Fisher exact test used to assess statistical significance. †Superscripts that are the same do not statistically differ. ‡Door-to-balloon time represents the total time from presentation at a referral emergency department or hospital to the first device at the PCI hospital, including time for transport to Abbott Northwestern Hospital. Patients who expired prior to coronary angiography are included in the database for calculation of mortality, but they are not assigned a door-to-balloon time. §Percentage based on those patients alive at discharge.

Abbreviations as in Tables 1 and 2.

Excess 5-year mortality may merely reflect other risk factors including greater age, lower left ventricular ejection fraction, and more extensive coronary artery disease of the CABG group.

The strengths of this study include a large sample size: the 249 patients with previous CABG represent the largest prospective series to date. Although this cohort is nonrandomized and drawn from a prospective registry, the design allows for provision of evidence-based therapy throughout the study period (2003 to 2012) and ensures that treatment reflects current clinical practice. The Minneapolis Heart Institute's Level 1 MI program is designed to evolve in parallel with changes in contemporary standards of care, including preferential use of newer-generation drug-eluting stents and bivalirudin during coronary intervention, which may improve outcomes in patients with advanced disease or multiple medical comorbidities. Perhaps more importantly, the time to reperfusion (door-to-balloon time) and adjunctive medical therapy are similar because standardized protocols have been used in the regional STEMI system, a strategy that has demonstrated efficacy in improving the clinical outcomes of women and elderly patients (18). In addition to interventional cardiologists, we believe that this data has broad applicability to the emergency medicine, internal medicine, or general cardiology physicians treating a patient with previous CABG who presents with acute chest pain and ST-segment elevation.

When patients with previous CABG are stratified by culprit artery, several interesting trends emerge. There is an increased prevalence of cardiogenic shock at hospital admission among those with a SVG culprit artery. Interestingly, when compared with patients with a native vessel culprit, there are no corresponding differences in baseline ejection fraction, rates of reperfusion, post-PCI flow, infarct size, or length of stay. There is also a trend toward increased mortality at 30 days and 1 year among patients with an SVG culprit that did not reach clinical significance. As noted, Welsh et al. (9) reported a similar finding in their analysis of the APEX-AMI data. The relatively small numbers of patients, per culprit artery, limit the statistical power with respect to subgroup analysis, and it is possible that this trend toward increased mortality is real and reflects the increased rates of shock on arrival. In patients with previous CABG and no clear culprit identified on angiography, approximately one-third were noted to have positive cardiac enzymes. The remaining two-thirds included patients with left bundle branch block, prior MI, and other ECG mimics, characteristics similar to those described in a previous report (2). Whereas one must maintain a high index of suspicion for acute ischemia patients with previous bypass, the absence of cardiac enzyme elevation in 16% of patients with previous CABG highlights the value of obtaining an old ECG for comparison and, potentially, reviewing the case with a consulting cardiologist prior to catheterization lab activation. The removal of a "new" left bundle branch block as an ECG criterion for catheterization lab activation in the most recent American College of Cardiology Foundation/American Heart Association guidelines for the management of STEMI may also decrease the risk of false activation in patients with previous CABG. Nevertheless, one-third of patients with previous CABG and no clear culprit had elevated biomarkers and the 30-day, 1-year, and 5-year mortality was similar to patients with a SVG or native vessel culprit, highlighting the significant comorbid illness in this population.

STUDY LIMITATIONS. Although prospective, this study is nonrandomized; however, it is unlikely a randomized trial will ever be conducted. The regional STEMI program database captures only patients referred for urgent angiography and therefore possibly includes a referral bias. It remains a large sample, and our previous reports suggest very low rates of eligible but untreated patients. Finally, the power to analyze differences in clinical outcome may be limited by the small sample size available.

CONCLUSIONS

In a large, prospective STEMI database, there was no excess 1-year mortality, MACE, or rehospitalization among patients with previous CABG versus patients without previous bypass surgery. Mortality at 5 years is increased among patients with previous bypass. Although adverse clinical outcomes, including death, were numerically increased in those with a SVG culprit, this finding did not reach statistical significance. This study demonstrates that contemporary 1-year clinical outcomes of STEMI patients with previous CABG are equivalent to those of an unselected STEMI population with use of a standardized protocol and early reperfusion with primary PCI.

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