

Multiple Culprit Arteries in Patients With ST Segment Elevation Myocardial Infarction Referred for Primary Percutaneous Coronary Intervention

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In most cases of acute ST-segment elevation myocardial infarction, only 1 epicardial artery contains an occluding thrombus, commonly referred to as the “culprit” artery. Rarely, however, patients present with >1 acutely thrombosed coronary artery (i.e., “multiple culprits”). The investigators present their experience with 18 patients presenting with ST-segment elevation myocardial infarctions and angiographically documented multiple culprit arteries, provide a detailed review of an additional 29 patients previously reported, and summarize baseline characteristics, pertinent electrocardiographic and angiographic findings, laboratory values, and clinical outcomes for all 47 patients. In this case series, most patients were men (85%) with histories of tobacco use (49%). Although nearly 1/3 of the patients had isolated inferior ST-segment elevation on initial 12-lead electrocardiography, 50% of them had simultaneous thrombotic occlusions of the right coronary and the left anterior descending coronary arteries documented on coronary angiography. These patients were hemodynamically unstable on presentation, with >1/3 in cardiogenic shock. In most cases, no other potential predisposing factors were identified. In conclusion, patients with multiple culprit arteries in the setting of ST-segment elevation myocardial infarctions represent a unique population with high rates of cardiogenic shock and no clear cause. © 2009 Elsevier Inc. All rights reserved. (Am J Cardiol 2009;104:619–623)

We present our own case series of 18 patients with ST-segment elevation myocardial infarctions (STEMIs) who were referred for primary percutaneous coronary intervention (PCI) in whom multiple culprit arteries were identified angiographically. In addition, we provide a detailed review of an additional 29 patients previously reported, report summary data for all 47 patients, and discuss possible causative factors.

Methods

With investigational review board approval, we searched the primary PCI database at the University of Texas Southwestern Medical Center and the University of Virginia for all patients presenting with STEMIs referred for primary PCI who had evidence of ST-segment elevation on admission 12-lead electrocardiography and >1 acutely occluded coronary artery documented on coronary angiography. All angiograms were reviewed by an interventional cardiologist (E.C.K.), and the angiographic results were overread by 2 additional reviewers (P.M.P. and M.K.). Only patients whose angiograms revealed de novo angiographic evidence of intraluminal filling defects consistent with acute thrombi

and for whom there was consensus among the reviewers were included. The previously described and widely accepted definition of angiographic thrombus was used to discriminate between cases¹: (1) intraluminal filling defect, defined as an abrupt vessel cutoff with persistence of contrast seen in multiple angiographic views, and (2) an occluded vessel with evidence of a convex margin that stains with contrast and persists for several cardiac cycles. Patients with chronic total occlusions were excluded. Patients with evidence of acute or subacute stent thromboses were also excluded, because the discontinuation of dual-antiplatelet therapy with aspirin and clopidogrel, inadequately deployed intracoronary stents, and individual unresponsiveness to the antiplatelet effects of aspirin are well-accepted risk factors for this occurrence² and do not reflect the unique patient with a STEMI with multiple de novo coronary occlusions.

We collected demographic and clinical information, including medical history, long-term medications, laboratory values, 12-lead electrocardiographic results, angiographic findings, periprocedural complications, and in-hospital clinical outcomes, for all patients. These data were obtained from the electronic medical records and the cardiac catheterization database.

We searched previously published cases of de novo multiple culprit arteries in patients with STEMIs. We reviewed the Medline database for reports published from 1988 to 2008. The following key words were used in the search: “thrombosis,” “thrombus,” “thromboses,” “multiple,” “myocardial infarction,” “acute myocardial infarction,” “ST segment elevation myocardial infarction,” “occlusion,” “simultaneous occlusion,” “multivessel,” “culprit,” and “infarct-related artery.”

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Table 1

Characteristics of 18 patients with ST-segment elevation myocardial infarctions and multiple culprit arteries in our original case series

Age	Gender	Race	LDL (mg/dl)	HDL (mg/dl)	Previous CAD	Previous MI	Previous PCI	β Blockers	Statins
32	M	Hispanic	96	25	0	0	0	0	0
43	M	White	176	72	+	+	+	0	0
48	M	White	126	31	0	0	0	0	0
49	M	White	192	39	0	0	0	+	0
49	M	Hispanic	127	40	0	0	0	0	0
50	M	White	NA	NA	0	0	0	0	+
54	M	Hispanic	174	38	0	0	0	0	0
57	M	White	131	38	0	0	0	0	0
57	M	Indian	85	33	0	0	0	0	0
64	M	White	88	34	+	+	+	+	+
65	M	White	130	38	0	0	0	0	0
66	M	Black	181	43	+	+	+	0	+
67	M	White	113	45	0	0	0	0	0
71	M	White	117	28	+	+	+	+	+
73	M	White	105	35	+	+	+	+	0
85	M	Black	87	36	+	+	+	+	+
49	F	Hispanic	142	NA	0	0	0	0	0
70	F	White	84	60	0	0	0	0	0

CAD = coronary artery disease; ECG = electrocardiographic; HDL = high-density lipoprotein; HIV = human immunodeficiency virus; LAD = left anterior descending coronary artery; LC = left circumflex coronary artery; LDL = low-density lipoprotein; MI = myocardial infarction; NA = not available; RCA = right coronary artery.

The reports reviewed were limited to those written in English. Only cases that documented angiographic evidence of filling defects consistent with intraluminal thrombus were included; cases reported as secondary to transient coronary artery vasospasm that resolved with nitrates alone were not included. Data from autopsy series were excluded unless coronary angiography was performed in life and angiographic appearance of thrombus was reported. Reports of acute or subacute stent thromboses were excluded for the reasons stated previously.

Results

From December 1, 2000, to March 31, 2006, 184 consecutive patients underwent primary PCI for STEMI at the University of Texas Southwestern Medical Center. Of these 184 patients, 9 (4.8%) had angiographic evidence of multiple culprits. From January 1, 2004, to December 31, 2007, 527 consecutive patients underwent primary PCI at the University of Virginia. Of these, 9 (1.7%) had multiple culprits. Therefore, our original case series consisted of 18 of 711 patients with STEMIs (2.5%) with multiple culprit arteries. Our review of the published research revealed an additional 29 patients with multiple culprit arteries,^{3–26} bringing our total case series to 47 patients. Detailed information pertaining to the 18 patients in our original case series is listed in Table 1. Baseline comparative patient characteristics (Table 2), laboratory values (Table 3), 12-lead electrocardiographic and angiographic information (Table 4), and procedural and in-hospital clinical outcomes (Table 5) for all 47 patients are listed in Tables 2 to 5.

Most patients in our original series were Caucasian (61%), were male (89%), and had histories of current to-

bacco use (56%). Nearly 1/3 of patients were taking β blockers, statins, and aspirin before admission (Table 2). A high percentage of patients in published reports were current smokers, and nearly 1/3 were Japanese (Table 2). Laboratory values revealed poorly controlled total, low-density lipoprotein, and high-density lipoprotein cholesterol in our original series and elevated triglyceride levels in patients described in published reports (Table 3). Overall, the extent of myocardial damage was evident by the markedly elevated peak troponin and creatine kinase levels. None of the patients in our series (original or published reports) had histories of rheumatic heart disease, infective endocarditis, nonbacterial thrombotic endocarditis, chest wall trauma, mural thrombi, or prosthetic heart valves, all of which are associated with coronary artery embolization.²⁷ Of the 47 patients, 2 were noted to be in atrial fibrillation on admission (1 from a published report⁵ and 1 from our original case series). Echocardiography performed after the procedures in these 2 patients, however, did not reveal evidence of mural thrombi.

In our original series, 50% of patients had simultaneous occlusions of the right coronary and left circumflex coronary arteries: 62% of the patients previously reported had occlusions of the right coronary and the left anterior descending coronary arteries (Table 4). The most frequent 12-lead electrocardiographic finding in our original series was ST-segment elevation in the inferior leads; in previously published reports, ST-segment elevation in the inferior and anterior leads was the most frequent finding (Table 4).

Patients with multiple culprit arteries in our original series presented with a high incidence of clinical and hemodynamic instability (Table 5): 28% were in cardiogenic

Table 1
(continued)

Aspirin	Clopidogrel	ECG Location of ST Elevation	ECG Reciprocal Depression	Initial Troponin	Peak Troponin	Culprit Coronary Artery	Ventricular Arrhythmias	Cardiogenic Shock	Potential Cause
0	0	Anterior	0	62	259	LAD/LC	+	+	Cocaine
0	0	Anterior/Inferior	+	2	18	RCA/LC	0	0	0
0	0	Inferior	+	0.1	44	RCA/LC	0	0	0
0	0	Inferior	+	9	43	RCA/LAD	0	0	Cancer
0	0	Inferior	0	0.1	208	RCA/LC	0	0	0
0	0	Anterior/lateral	0	2	232	LAD/LC	0	0	HIV
0	0	Anterior	0	2	240	RCA/LAD	0	0	0
0	0	Anterior/inferior/posterior/lateral	+	7	98	RCA/LAD	+	+	0
0	0	Anterior	+	0.1	262	LAD/LC	0	0	0
+	+	Inferior	+	150	150	RCA/LC	+	+	Cancer
0	0	Anterior	+	0.1	500	LAD/LC	0	+	HIV
+	0	Inferior/posterior	+	0.4	362	RCA/LC	0	0	0
0	0	Anterior	0	45	59	RCA/LAD	0	0	0
+	0	Inferior	0	0.1	32	RCA/LAD	0	0	0
+	0	Inferior	+	0.6	112	RCA/LC	+	+	0
+	0	Inferior/posterior	+	5	254	RCA/LC	0	0	0
0	0	Inferior/posterior	0	0.1	25	RCA/LC	0	0	0
0	0	Inferior	0	36	36	RCA/LC	0	0	Cancer

shock, 22% had life-threatening ventricular arrhythmias, and 22% required intra-aortic balloon pumps. Although the rates of life-threatening arrhythmias were very consistent with those previously reported, the rates of cardiogenic shock and intra-aortic balloon pump placement in our original series were lower (Table 5). Most patients underwent PCI, and a small number of patients from the previously published reports were treated concomitantly with intra-coronary infusions of thrombolytic therapy. Despite their unstable presentations and large extents of myocardial damage, nearly all patients survived to hospital discharge, with few major in-hospital adverse events.

Discussion

Autopsy series in patients who died from acute myocardial infarctions report that thrombotic occlusion of >1 major epicardial coronary artery is not rare, occurring in up to 50% of patients.²⁸ In our original case series, we found 18 patients with multiple culprit arteries among 711 patients (2.5%) who underwent emergent primary PCI. This discrepancy is likely due to selection bias: patients with STEMIs with multiple culprit arteries may be more likely to present with sudden cardiac death, not surviving long enough to undergo angiography. Our findings emphasize the clinical severity associated with this condition: approximately 1/3 of patients presented in cardiogenic shock, and nearly 1/4 of patients had life-threatening arrhythmias or required intra-aortic balloon pumps.

Although the presence of multiple complex plaques (but not thrombotic occlusions) in patients with STEMIs has been reported,¹ our patients were unique in that > 1 artery

was angiographically documented to be acutely thrombosed. Moreover, whereas the arteries responsible for the STEMIs were clearly identified in 98% of patients with complex plaques in a previously published report,¹ no single coronary artery could be deemed the culprit artery in our patients.

Although the causative factors involved in the acute and simultaneous thrombosis of multiple coronary arteries are unclear, possible contributing factors include (1) heightened inflammatory response and catecholamine surge caused by the acute occlusion of 1 vessel, resulting in a second coronary arterial occlusion; (2) hemodynamic instability and hypotension due to the occlusion of 1 coronary artery, resulting in blood stasis and acute occlusion in another artery with a severe underlying lesion; (3) prolonged coronary vasospasm (due to Prinzmetal's [variant] angina or in the context of cocaine use)²⁹; (4) hypercoagulable states due to malignancy and thrombocytosis³⁰; and (5) coronary embolism.²⁷ In our total case series of 47 patients, 19 (40%) had co-morbidities that were potential contributing factors, including a history of cancer,^{16,24} human immunodeficiency virus, cocaine use,²¹ coronary artery vasospasm,^{11,22,26} platelet abnormalities,^{4,10,13,15} atrial fibrillation,⁵ and hyperhomocysteinemia.¹⁸ In 60% of the patients, however, no other possible contributing factors were identified.

Our study had limitations. First, it was a retrospective analysis of patients referred for primary PCI; it is possible that selection bias may have influenced who was, and who was not, referred for angiography. Second, because of the inherent nature of a case series, ours was descriptive and limited by the lack of a control group. Third, although all potential causative factors available were reported, it is

Table 2
Summary of baseline patient characteristics

Characteristic	Original Series (n = 18)	Previously Reported (n = 29)	Total Case Series (n = 47)
Gender			
Male	16 (89%)	24 (83%)	40 (85%)
Female	2 (11%)	5 (17%)	7 (15%)
Age (years)	57 ± 13	54 ± 15	54 ± 14
Race/ethnicity			
White	11 (61%)	1 (3%)	12 (26%)
Black	2 (11%)	NR	2 (4%)
Hispanic	4 (22%)	1 (3%)	5 (11%)
Japanese	0	9 (31%)	9 (19%)
Chinese	0	1 (3%)	1 (2%)
Indian	1 (6%)	1 (3%)	2 (4%)
NR	0	16 (55%)	16 (34%)
Hypertension	9 (50%)	6 (21%)	15 (32%)
Diabetes mellitus	3 (17%)	7 (24%)	10 (21%)
Tobacco use (current)	10 (56%)	13 (45%)	23 (49%)
Hypercholesterolemia	6 (33%)	2 (7%)	8 (17%)
Family history of CAD before age 50 years	7 (39%)	5 (17%)	12 (26%)
History of CAD	6 (33%)	1 (3%)	7 (15%)
Previous MI	6 (33%)	1 (3%)	7 (15%)
Previous PCI	6 (33%)	1 (3%)	7 (15%)
Previous congestive heart failure	1 (6%)	0	1 (2%)
Cocaine use (current)	1 (6%)	1 (3%)	2 (4%)
Medications			
β blockers	5 (28%)	NR	5 (11%)
ACE inhibitors	2 (11%)	NR	2 (4%)
Nitrates	2 (11%)	NR	2 (4%)
Statins	5 (28%)	NR	5 (11%)
Aspirin	5 (28%)	NR	5 (11%)
Clopidogrel	1 (6%)	NR	1 (2%)
Insulin	1 (6%)	NR	1 (2%)

Data are expressed as mean ± SD or as number (percentage).

ACE = angiotensin-converting enzyme; NR = not reported. Other abbreviations as in Table 1.

Table 3
Laboratory values

Laboratory	Original Series	Previously Reported	Total Case Series
Total cholesterol (mg/dl)	179 ± 42	140 ± 80	177 ± 47
LDL cholesterol (mg/dl)	126 ± 36	75 ± 68	115 ± 39
HDL cholesterol (mg/dl)	38 ± 12	36 ± 8	37 ± 11
Triglycerides (mg/dl)	127 ± 128	174 ± 381	141 ± 189
White blood cell count (×10 ³ /mm ³)	10 ± 5	14.5 ± 4	12 ± 5
Glucose (mg/dl)	134 ± 83	NR	
Creatinine (mg/dl)	1.1 ± 0.2	NR	
Creatine kinase (IU/L)	NR	3,780 ± 4,365	
Creatine kinase-MB (%)	NR	274 ± 488	
Initial troponin (μg/L)	2 ± 38	NR	
Peak troponin (μg/L)	131 ± 136	NR	

Data are expressed as mean ± SD.

Abbreviations as in Tables 1 and 2.

possible that the workup was incomplete for other causes. Fourth, most patients did not undergo subsequent invasive testing for the presence of coronary vasospasm.

Table 4
Electrocardiographic and angiographic findings

Finding	Original Series (n = 18)	Previously Reported (n = 29)	Total Case Series (n = 47)
Electrocardiographic findings			
Location of ST elevation			
Inferior	7 (39%)	7 (24%)	14 (30%)
Anterior	5 (28%)	1 (3%)	6 (13%)
Lateral	0	1 (3%)	1 (2%)
Inferior, anterior	1 (6%)	8 (28%)	9 (19%)
Inferior, lateral	0	3 (10%)	3 (6%)
Anterior, lateral	1 (6%)	6 (21%)	7 (15%)
Inferior, posterior	3 (17%)	0	3 (6%)
Anterior, inferior, lateral	0	1 (3%)	1 (2%)
Anterior, inferior, posterior, lateral	1 (6%)	0	1 (2%)
Hyperacute T waves	0	1 (3%)	1 (2%)
Coronary angiographic findings			
Acutely occluded arteries			
RCA and LC	9 (50%)	4 (14%)	13 (28%)
RCA and LAD	5 (28%)	18 (62%)	23 (49%)
LAD and LC	4 (22%)	3 (10%)	7 (15%)
Left main and RCA	0	1 (3%)	1 (2%)
Left main and ramus	0	1 (3%)	1 (2%)
RCA and ramus	0	1 (3%)	1 (2%)
RCA, LAD, and LC	0	1 (3%)	1 (2%)

Abbreviations as in Table 1.

Table 5
Periprocedural and in-hospital clinical outcomes

Characteristic	Original Series (n = 18)	Previously Reported (n = 29)	Total Case Series (n = 47)
Clinical severity on presentation			
Cardiogenic shock	5 (28%)	12 (41%)	17 (36%)
Ventricular arrhythmias	4 (22%)	7 (24%)	11 (23%)
Procedural details			
Intra-aortic balloon pump	4 (22%)	11 (38%)	15 (32%)
Balloon angioplasty ± stenting	16 (89%)	19 (66%)	35 (74%)
Platelet glycoprotein IIb/ IIIa inhibitor used	12 (67%)	11 (38%)	23 (49%)
Intracoronary infusion of thrombolytic agent	0	6 (21%)	6 (13%)
Thrombectomy	0	3 (10%)	3 (6%)
In-hospital clinical outcomes			
Recurrent myocardial infarction	0	1 (3%)	1 (2%)
New congestive heart failure	1 (6%)	2 (7%)	3 (6%)
Bleeding	1 (6%)	0	1 (2%)
Stroke	1 (6%)	0	1 (2%)
Death	0	1 (3%)	1 (2%)

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