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LETTER TO THE EDITOR

Angiographic Correlates in Type 1 and 2 MI by the Universal Definition

In the 1980s, the angiographic morphology of culprit lesions in unstable angina and acute myocardial infarction (AMI) was described distinct from that seen in stable angina (1,2). These lesions were originally designated as "type 2 eccentric" and later on as "complex lesions." They were found in ~70% of patients with an acute coronary syndrome and <20% in stable angina patients. These lesions were thought to represent plaque rupture and/or thrombus formation (3).

Over the years, both the terminology and diagnostic criteria of acute coronary syndrome have evolved. Recently, a new universal definition of myocardial infarction was proposed based on the pathophysiology of AMI, which was defined as an increase and decrease in troponin in the presence of clinical evidence of ischemia (4). Of the types of AMI defined in this communication, type 1 myocardial infarction (MI) included patients with evidence of plaque rupture or erosion in an epicardial coronary vessel, whereas type 2 MI was AMI precipitated by a primary imbalance in the myocardial blood supply to myocardial oxygen demand ratio. Although morphological and angiographic findings in AMI have been previously reported, the angiographic findings in these 2 distinct subsets of AMI have not. We hypothesized that in type 1 MI, the angiographic findings should mirror previous studies (1,2) with a predominance of complex lesions in both ST-segment elevation myocardial infarction (STEMI) and non–ST-segment elevation myocardial infarction (NSTEMI), whereas in type 2 MI, culprit vessels and complex lesions would be infrequent.

The population was part of a prospective evaluation of patients presenting to a large community hospital with elevated troponin I levels. The clinical findings were previously published in part by Javed et al. (5). Type 1 MI required clinical ischemia and a presumed primary coronary event such as plaque rupture/erosion based on presentation, without a secondary clinical condition associated with a supply/demand mismatch such as a tachyarrhythmia, severe hypertension, and hypotension. Type 2 MI required a primary documented disturbance in supply/demand. In 10 of 31 cases, an angiogram was necessary to confirm a type 2 MI. These patients presented with typical ischemic chest pain either secondary to uncontrolled hypertension or presumed coronary spasm from illicit drug use and had normal or nonobstructive coronary artery disease on angiography.

The present analysis included all patients with an in-hospital angiogram and a diagnosis of either type 1 or 2 MI. Two angiographers, blinded to clinical history, reviewed all angiograms for the following: 1) number of diseased vessels; 2) the presence of an identifiable culprit lesion and vessel; and 3) culprit lesion morphology (complex vs. simple: simple if no complex features were identified). A culprit lesion was considered as the only significant lesion on angiography (usually >70% diameter stenosis). In multivessel disease, the culprit was a significant lesion in a vessel that corresponded to the new electrocardiographic changes or wall motion abnormalities. If not localizable to a single site, no culprit was assigned in multivessel disease. Patients with nonobstructive disease (<50% diameter stenosis) or vessels without luminal narrowing were also designated as an MI without a culprit lesion/vessel. The coronary morphology of the culprit lesion was qualitatively analyzed in orthogonal views. A *complex* lesion was defined as an acute or recent total occlusion (dye stasis at the site) or a patent vessel with a significant lesion that was usually eccentric with either overhanging edges, abrupt shoulders, ulcerations, and/or filling defects at or distal to the lesion indicating intracoronary thrombus.

A total of 224 patients were identified: 193 with type 1 and 31 with type 2 MI. Baseline demographic data are contained in Table 1. There were no significant differences other than a higher incidence of chronic kidney disease in the type 2 group and higher troponin levels in type 1. In type 1 MI patients, 73 (37.8%) had STEMI, whereas 120 (62.2%) had a diagnosis of NSTEMI (Fig. 1).

Nearly all type 1 STEMI patients (95%) had an identifiable culprit lesion. Nonobstructive/normal or 3-vessel disease was infrequent (1.4%). In type 1 NSTEMI patients, an identifiable

Table 1. Baseline Characteristics

	Type 1 (n = 193)	Type 2 (n = 31)	p Value
Age, yrs	63 ± 13	67 ± 13	0.13
Men	129 (66.8)	18 (58.1)	0.31
Peak TnI, ng/ml	5.57 (0.05–822.6)	1.00 (0.05–35.18)	<0.001
STEMI	44.38 (0.23–822.6)		
NSTEMI	1.44 (0.05–337.65)		
Diabetes mellitus	82 (42.9)	18 (58.1)	0.14
Dyslipidemia	137 (71.0)	21 (67.7)	0.53
Hypertension	151 (78.2)	27 (87.1)	0.4
Smoker	91 (47.2)	11 (35.5)	0.17
Illicit drug use	20 (10.4)	5 (16.1)	0.42
Chronic kidney disease	32 (16.6)	11 (35.5)	0.02
Obese (BMI >30 kg/m^2)	58 (30.1)	5 (16.1)	0.09
Family history of premature CHD	40 (20.7)	3 (9.7)	0.13
Previous coronary disease	59 (30.6)	14 (45.2)	0.14
Cardiomyopathy	17 (8.8)	6 (19.4)	0.09

Values are mean ± SD, n (%), or median (range).
BMI = body mass index; CHD = coronary heart disease; NSTEMI = non–ST-segment elevation myocardial infarction; STEMI = ST-segment elevation myocardial infarction; TnI = cardiac troponin I.

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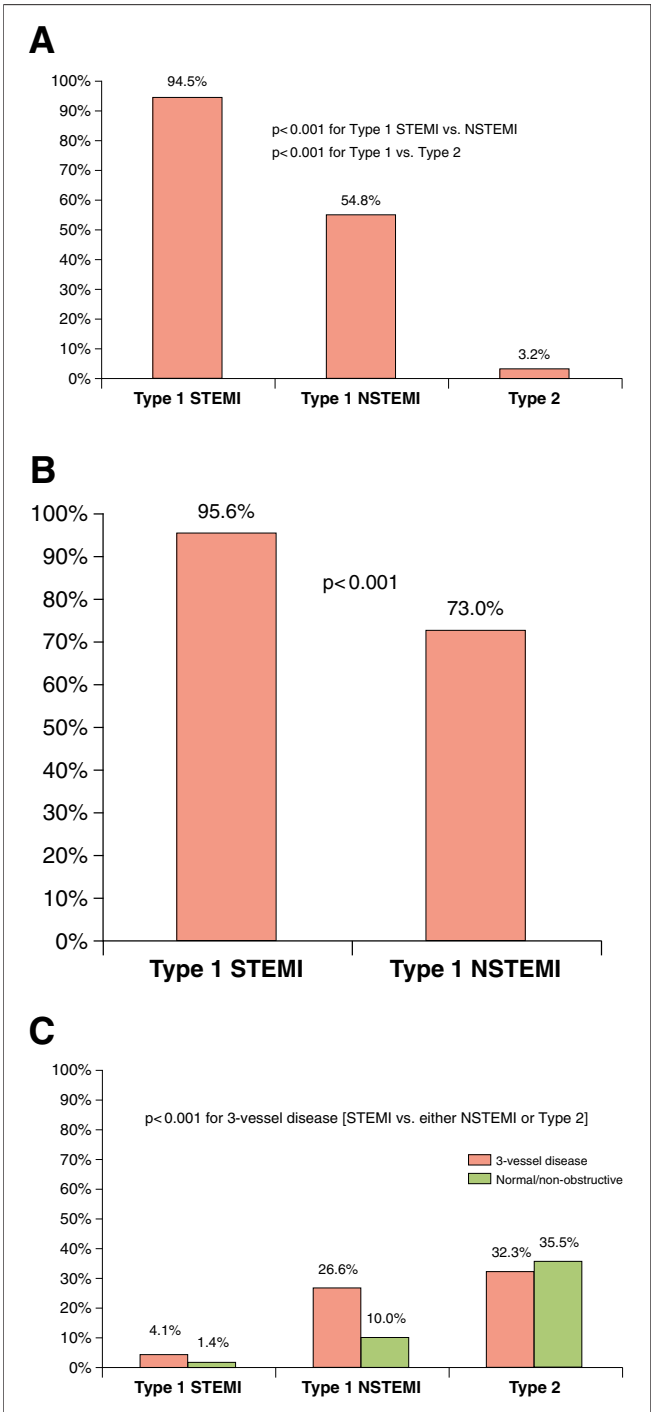

Figure 1. Results

(A) The percentage of an identifiable culprit lesion for type 1 STEMI, type 1 NSTEMI, and type 2 MI. **(B)** The percentage of complex lesion morphology in Type 1 STEMI and NSTEMI with an identifiable culprit lesion. **(C)** The percentage of patients with normal/nonobstructive and 3-vessel disease among type 1 STEMI, type 1 NSTEMI, and type 2 MI. (No statistical analysis was performed for normal/nonobstructive category as angiography was required for type 2 diagnosis in 10 of 31 with normal/nonobstructive coronary artery disease.) MI = myocardial infarction; NSTEMI = non–ST-segment elevation myocardial infarction; STEMI = ST-segment elevation myocardial infarction.

culprit lesion was found in 56%, and the majority (73%) were complex. Nonobstructive/normal or 3-vessel disease was present in 10% and 27%, respectively. A culprit lesion could be identified in 43% patients with 3-vessel disease.

Of the 31 type 2 MI patients, only 1 had an identifiable culprit vessel, and nonobstructive/normal or 3-vessel disease was found in 36% and 32%, respectively. In summary, these data suggest that culprit lesion morphology and other easily identifiable angiographic parameters contribute to our understanding of AMI pathogenesis by the universal definition. These findings support the differentiation of type 1 from type 2 MI.

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REFERENCES

1. Ambrose JA, Winters SL, Stern A, et al. Angiographic morphology and the pathogenesis of unstable angina. J Am Coll Cardiol 1985;5:609–16.
2. Ambrose JA, Winters SL, Arora RR, et al. Coronary angiographic morphology in myocardial infarction: a link between the pathogenesis of unstable angina and myocardial infarction. J Am Coll Cardiol 1985;6:1233–8.
3. Levin DC, Fallon JT. Significance of the angiographic morphology of localized coronary stenoses: histopathologic correlations. Circulation 1982;66:316–20.
4. Thygesen K, Alpert JS, White HD, et al. Universal definition of myocardial infarction. Circulation 2007;116:2634–53.
5. Javed U, Aftab W, Ambrose JA, et al. Frequency of elevated troponin I and diagnosis of acute myocardial infarction. Am J Cardiol 2009;104:9–13.