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The Universal Definition of Myocardial Infarction (UDMI)[1] classifies myocardial infarction

(MI) into 5 subtypes, of which type 1 and type 2 MI are the most common and relevant to

practicing clinicians. Type 1 MI is defined as MI caused by acute atherothrombotic mechanisms,

with type 2 MI defined as MI resulting from myocardial oxygen supply/demand mismatch

without acute atherothrombosis. The UDMI recognizes multiple potential causes of type 2 MI,

including "demand side" abnormalities such as tachyarrhythmia or severe hypertension, and

"supply side" issues such as severe anemia, hypoxemia or hypotension. Type 2 MI may occur

with or without obstructive coronary disease, with the threshold for type 2 MI lower in patients

with fixed obstructive CAD.

Type 2 MI is common and is associated with substantial risk for cardiac (and noncardiac)

death and major adverse cardiac events.[2] Research to date has been limited largely to

observational studies that have used varying definitions and adjudication criteria for type 2 MI,

focusing on prevalence, risk factors, and prognosis, with almost no data on treatment.[2] Recently,

an ICD-10 code was introduced for type 2 MI, and it is hoped that this will facilitate research

using administrative data.[3] However, we believe that the current definition for type 2 MI is too

phenotypically heterogeneous to permit adequate study or reliable coding by hospital

administrators.

Importantly, the UDMI includes under the umbrella of type 2 MI several acute coronary

processes that obstruct blood flow, including spontaneous coronary artery dissection (SCAD),

coronary embolism and coronary vasospasm. We believe that these specific diagnoses are a poor

fit in the category of type 2 MI: from both a pathophysiological and clinical perspective, they are

more closely aligned with type 1 MI. SCAD, coronary embolism, and vasospasm are acute

"supply side" obstructive processes that have clinical presentations and initial diagnostic and

management approaches that are similar to type 1 MI. They are usually spontaneous

presentations, without an obvious precipitating event. These conditions typically are initially

triaged as suspected acute coronary syndromes (ACS), treated with guideline-recommended

therapies for ACS, and evaluated with early coronary imaging. The diagnosis is usually made in

the cardiac catheterization laboratory, with subsequent treatment determined by findings from

coronary imaging. In contrast, most other etiologies causing type 2 MI, including severe

tachycardia and hypertension, anemia, and hypoxemia, are apparent at the time of clinical

presentation, and diagnosed based on clinical criteria, with coronary angiography delayed or

deferred.

Applying the same diagnosis of type 2 MI to such phenotypically distinct patients has

clear disadvantages for clinical management, and negatively impacts the quality of research into

type 2 MI. Reporting the epidemiology, outcomes and treatment responses of type 2 MI, as

currently defined, is of little value other than making sure that these diagnoses do not "muddy"

the interpretation of type 1 MI.   On the other hand, including patients without acute

atherothrombosis in the type 1 MI category also creates problems. Clinical trials and guideline

recommendations for management of ACS are only applicable to type 1 MI. For example,

applying therapies tested in atherothrombosis, such as parenteral anticoagulation and

intracoronary stenting, to patients with SCAD, may be harmful.[4] Evidenced-based therapies

exist for coronary vasospasm and are emerging for SCAD, with coronary embolism typically

managed empirically based on the source of embolism. However, such therapies are clearly

distinct from those used to treat acute atherothrombosis.

We propose consideration of a modest redefinition of type 1 and type 2 MI **(figure)**, with

type 1 MI defined by acute coronary obstruction or reduction in coronary blood flow rather than

by atherothrombosis. This would move SCAD, coronary embolism and coronary vasomotor abnormalities (including epicardial vasospasm and microvascular dysfunction) into the type 1 MI category. We further propose subclassifying type 1 MI based on the underlying pathophysiology, with type 1A MI being the typical atherothrombosis category and the other etiologies having separate subclassifications **(figure)**. Type 2 MI would be defined as MI due to acute supply/demand mismatch *without acute coronary obstruction.* We propose further subclassifying type 2 MI into those with or without obstructive CAD **(figure)**, as the subsequent management approaches differ substantially based on the presence of severe CAD. As an important corollary, modification of ICD codes to improve specificity would be an important step forward for research and quality improvement in patients with MI caused by factors other than atherothrombosis.

We believe that this redefinition would better align with modern approaches to diagnosis and management of the spectrum of patients with MI. It would facilitate research into specific diagnostic subcategories and identification of optimal treatment approaches. This designation would also eliminate the need for a separate classification scheme for myocardial infarction with no obstructive coronary disease (MINOCA).[5] Each of the categories of MINOCA would fit within the new MI definition construct. Also, the creation of subcategories (Type 1A, 1B, etc) would allow room for additional MI phenotypes, as new information on pathophysiology becomes available, without altering the fundamental structure of the classification.

Although some may view differences between the UDMI and our proposed revision as little more than administrative detail, we would argue that alignment of diagnosis with clinical presentation, pathophysiology, and diagnostic approach is an essential step to address current knowledge gaps. Moreover, accurate diagnosis has direct implications for quality reporting, as

evidence based standards exist only for MI due to atherothrombosis. Clinicians should be held accountable only for adhering to process and performance measures for those patients in whom the measures apply. Finally, and arguably most importantly, as we enter the precision medicine era, it is imperative that our diagnoses be as precise as possible.

Disclosures

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Figure Legend

Figure. Proposed revision to Universal Definition of Myocardial Infarction (UDMI). The proposal redefines type 1 and type 2 MI based on the presence or absence of acute coronary obstruction, with subclassifications based on underlying pathophysiology. This differs from the current UDMI by categorizing spontaneous coronary dissection, coronary embolism and vasospasm as type 1 MI, and subclassifying type 2 MI based on the presence or absence of fixed obstructive coronary disease.

Acute myocardial injury with signs and/or symptoms of ischemia
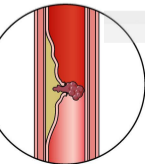
Type 1 Myocardial Infarction
MI with acute
coronary obstruction

Type 2 Myocardial Infarction
MI due to oxygen supply/demand mismatch
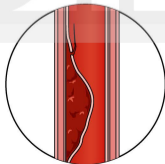without acute coronary obstruction
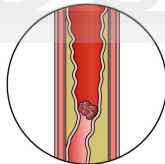
Type 1A
Plaque rupture/erosion
with thrombus
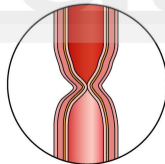
Type 1B
Spontaneous coronary
artery dissection

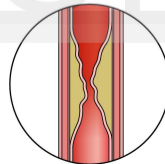**Type 1C**
Coronary
embolism

Type 1D
Vasospasm or
microvascular dysfunction

Type 2A
With fixed
obstructive CAD

Type 2B
Without fixed
obstructive CAD