Acute Myocardial Infarction: What's in a Name?

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The development of newer-generation troponin assays has led to remarkable progress in the rapid diagnosis or exclusion of acute myocardial infarction (AMI) (1). With increasing use of higher-sensitivity assays, it has also become evident that troponin level elevations are prevalent in several clinical situations in the absence of an acute coronary syndrome (ACS) (2). Experts have developed a universal classification system to categorize AMI on the basis of differing pathophysiologic mechanisms (3).

Type 1 or spontaneous myocardial infarction (T1MI, or ACS in common parlance) is secondary to acute atherosclerotic plaque instability in the coronary bed with intraluminal thrombosis, decreased myocardial blood flow, downstream platelet embolization, and myocardial necrosis. In contrast, type 2 myocardial infarction (T2MI) occurs in the absence of ACS from an imbalance between myocardial oxygen supply and demand. However, an important but overlooked corollary to the definition of T2MI is that there is currently no separate International Classification of Diseases (ICD) diagnostic code for this important and increasingly prevalent clinical entity in the ICD-9 (currently active in the United States) and ICD-10 (implemented in several countries around the world) manuals. This situation poses important clinical, epidemiologic, and economic ramifications that deserve careful consideration.

Although clinical differentiation between T1MI and T2MI may be challenging, their definitions in the universal classification system are logical and discrete and the implications of the 2 diagnoses differ. Despite knowing that an elevated troponin level portends a worse long-term prognosis, we lack evidence to guide management of T2MI. This makes it particularly challenging for the clinician, who is often left to grapple with accurately interpreting an elevated result of a highly sensitive troponin assay, which is frequently performed outside of the appropriate clinical context in which this assay was originally studied. Despite these challenges, T2MI is evolving into a distinct clinical entity and is associated with higher mortality rates than T1MI in several series, therefore meriting focused clinical attention (4–6).

The evolution of the higher-sensitivity troponin assays has also ushered in a shift in the epidemiologic trends of ACS. Dr. Eugene Braunwald, the principal architect of the classification of unstable angina, recently suggested that it may be time for a requiem for the term “unstable angina” (7). Data from Medicare patient claims provide “real-life” evidence to mirror Dr. Braunwald’s observation. Using ICD-9 diagnostic codes, we have shown that claims for unstable angina (code 411) among Medicare beneficiaries have declined rapidly in the past decade, whereas those for AMI (code 410) have remained fairly steady (8). This trend probably reflects the increased sensitivity of contemporary troponin assays for detecting minor degrees of myonecrosis, making the syndrome of unstable angina (the absence of detectable myonecrosis) increasingly obsolete.

The elephant in the room is the potential for significant economic consequences from the lack of a diagnostic code for T2MI. On the basis of the now widely accepted universal definition, all cases of T2MI can technically be coded as AMI (code 410) by clinicians and coders attempting to capture the appropriate severity of the patient’s illness. In the context of the growing recognition of T2MI as a clinical entity (4), rising sensitivity of modern troponin assays, and absence of a competing diagnostic code, it seems inevitable that the future will bring an uptick in the use of code 410 because of more frequent coding for T2MI (9). In fact, it can be easily conceived how higher reimbursement rates resulting from the use of code 410 could serve as an unintentional economic motivation to health care entities to be more liberal with its use. The fiscal consequences of such behavior, with a resultant increase in cost of hospitalizations, may be far from trivial. The risk in such a scenario may be highest for aging and economically vulnerable populations (such as Medicare patients), a matter that the Centers for Medicare & Medicaid Services may note.

Several additional sequelae could be anticipated. The use of code 410 at hospital discharge for patients with T2MI could also confuse clinical management if it leads to automatic triggers for pharmacologic management that is typically reserved for patients with ACS (for example, antiplatelet therapies, β-blockers, neurohormonal blockade, and statins). This kind of response is especially plausible in the contemporary era of heightened scrutiny by regulatory bodies overseeing hospitals and clinicians, with a very real downstream threat to reimbursement based on fulfillment of key quality metrics. Although speculative, this “knee-jerk” response could lead to “overtreatment” of patients with pharmacologic ACS strategies despite knowing that a substantial proportion of patients with T2MI may not have evidence for coronary artery disease (5). The downstream effects of a diagnosis of “AMI” in the absence of ACS could be detrimental to the individual patient for future insurability and employability if the previously mentioned subtleties and nuances of the definition of T2MI are not well-appreciated and documented by the concerned parties. Finally, researchers’ ability to study claims data to evaluate future trends in disease epidemiology (8), health care indicators, and economics could be confounded in the absence of a distinct diagnostic code.

In conclusion, the medical community has not yet paid adequate attention to the possible unintended consequences of the more widespread use of sensitive troponin assays and the evolution of the clinical concept of T2MI. Without question, we need well-designed, prospective studies to help clinicians determine the best strategies to manage patients with T2MI.
Meanwhile, the creation of a distinct diagnostic code for T2MI, also supported by experts in the field (4), would be a proactive step in the interest of patient-centeredness and appropriate stewardship of financial resources and may, in fact, help us better understand this entity from an epidemiologic standpoint. Our failure to act on this matter could lead to unfavorable financial and health care consequences and make the epidemiology of “spontaneous AMI” murky for future generations to decipher.

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Disclosures: The author has disclosed no conflicts of interest.

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References
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Drafting of the article: G.R. Shroff.
Critical revision of the article for important intellectual content: G.R. Shroff.
Final approval of the article: G.R. Shroff.