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Translating the Translation

What Clinicians Should Know About the Fourth Universal Definition of Myocardial Infarction

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This past August, the American College of Cardiology, American Heart Association, European Society of Cardiology, and World Heart Federation released the long-awaited 2018 Fourth Universal Definition of Myocardial Infarction (MI) with the goal of clearing up confusion over how MI is clinically defined (1).

The expert consensus document recognizes that there are multiple stakeholders around the globe that rely on the accurate diagnosis of MI and non-MI troponin elevation, including patients, clinicians, hospitals/health systems, and insurers. "Tentative or final diagnosis is the basis for advice about further diagnostic testing, lifestyle changes, treatment, and prognosis for the patient," the authors write. "The aggregate of patients with a particular diagnosis is the basis for health care planning, and policy and resource allocation" (1).

One of the biggest clinical takeaways from the document is an emphasis on the critical concept of how to differentiate among type 1 MI, type 2 MI, and non-MI causes of troponin elevation (e.g., myocardial injury without infarction). All guidance stems from the clinical definition of MI, denoting "the presence of acute myocardial injury detected by abnormal cardiac biomarkers in the setting of evidence of acute myocardial ischemia" (1).

Given the increasing accountability for high-quality MI care, the impending introduction of high-sensitivity cardiac troponin assays, and the increasing number of medical conditions other than MI associated with elevated troponin levels, the recommendations and guidance included in the document are essential.

Translating the recommendations into the routine practice of health care delivery and documentation are the next challenge. In the United States, for example, clinician documentation not only influences payment of hospital claims, but may also unwittingly contribute to penalties, such as those associated with the Centers for Medicare and Medicaid Services Hospital Readmissions Reduction Program (2).

To that end, a separate group of physicians, clinical documentation specialists, coding experts, and health policy experts from the American College of Cardiology and American Heart Association have taken on the critical task of educating clinicians about the importance of accurately documenting MI and non-MI causes of troponin elevation in the medical record. This group has specifically worked with members of the 10th Revision of the International Statistical Classification of Diseases and Related Health Problems (ICD-10) and the Centers for Medicare and Medicaid Services to more closely align MI documentation and coding practices with clinical practice guidelines (3).

While these more formal efforts are underway, a group led by Abhinav Goyal, MD, MHS, FACC, have created the following "Top 10" list of items clinicians

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must remember when documenting the type of MI or non-MI causes of troponin elevation (4).

1. *Clinicians should avoid reflexively documenting a diagnosis of MI solely on the basis of elevated troponin levels.* "Arriving at a diagnosis of MI using the criteria set forth in this document requires the integration of clinical findings, patterns on the ECG, laboratory data, observations from imaging procedures, and on occasion pathological findings, all viewed in the context of the time horizon over which the suspected event unfolds," the authors note (1).
2. *Documentation of a type 1 MI (which includes acute ST-segment elevation myocardial infarction [STEMI] and non–ST-segment elevation myocardial infarction [NSTEMI]) requires evidence of acute coronary thrombus or plaque rupture/erosion by coronary angiography (1).* For patients with type 1 MIs (both STEMI and NSTEMI), there should be prompt initiation of antithrombotic and anti-ischemic therapy, along with coronary angiography and revascularization as dictated by the coronary anatomy. In contrast, a type 2 MI results from imbalance between myocardial oxygen supply and demand that is unrelated to acute coronary thrombosis or plaque rupture (1). In general, treatment of a type 2 MI should *not* include antithrombotic therapy or urgent coronary angiography. Instead, clinicians should focus on managing the underlying cause(s) of the demand-supply mismatch. As a result, a type 2 MI should always be documented as a secondary diagnosis in the discharge summary, as it stems from an alternate (primary) cause (2).
3. *The terms "STEMI" and "NSTEMI" should only be used when referring to a type 1 MI.* As of October 1, 2017, ICD-10 and the Centers for Medicare and Medicaid Services have implemented a new ICD-10 diagnosis code for type 2 MI (I21.A1), distinct from the ICD-10 diagnosis code for NSTEMI (I21.4) (2). As such, the term "type 2 NSTEMI" should no longer be used.
4. There are myriad cardiac and systemic etiologies that can result in myocardial injury without infarction where an ischemic mechanism of injury is not implicated, including advanced chronic kidney disease and the presence of heterophile antibodies to troponin assay reagents (5,6). As such, *when documenting conditions associated with troponin elevation without myocardial injury, the term "non-MI troponin elevation (due to an underlying cause)" rather than "myocardial injury without infarction" should be used.*
5. Given the number of potential mechanisms for an elevated troponin, *"non-MI troponin elevation due to heart failure," rather than type 2 MI, is the correct diagnosis for most patients with decompensated heart failure.*
6. *Patients with a significantly elevated blood pressure (hypertensive urgency or emergency) and an elevated troponin usually do not have a type 1 MI.* Furthermore, based on the new Universal Definition, a patient must have overt symptoms of acute MI or new ischemic electrocardiography (ECG) findings accompanying other conditions resulting in myocardial ischemia to be diagnosed with type 2 MI. In contrast, a diagnosis of "non-MI troponin elevation (due to an underlying cause)" may be more appropriate if ischemic symptoms or ECG changes are lacking.
7. *Elevated troponin levels in the setting of tachyarrhythmias (e.g., atrial fibrillation with a rapid ventricular response) may represent a type 2 MI or a non-MI troponin elevation.* Some patients with tachyarrhythmias develop diffuse ST-segment depression resembling myocardial ischemia that in fact represents repolarization changes (termed "cardiac memory"). As such, new "ischemic" ECG changes may not be a reliable differentiator between these 2 conditions (1). Given that several studies showing elevated troponin levels in the setting of tachyarrhythmias were not associated with evidence of underlying ischemia with further investigation, provocative testing (e.g., stress testing) may help to distinguish between these conditions (7–9).
8. *Troponin elevation in the noncardiac perioperative period and in patients with critical illness may represent a type 1 MI, a type 2 MI, or a non-MI troponin elevation* (1). To distinguish among these diagnoses, it is necessary to first assess whether the patient has symptoms of ischemia and/or new ischemic ECG changes. Although determining this may require additional testing, strong consideration should be given to deferral of testing to the outpatient setting after recovery from surgery or the presenting illness. When testing is deferred, clinicians should use their best judgment as to the diagnosis.
9. *It is permissible for clinicians to document uncertainty as to the elevated troponin's etiology early into the hospitalization, with subsequent clarification.* For example, it is perfectly reasonable at presentation to not know the cause of an elevated troponin in a patient with known coronary artery disease presenting with hypertensive emergency. However, if the patient undergoes coronary

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angiography and there is no evidence of coronary thrombus or plaque rupture, it should be documented that a type 1 MI has been "ruled out." Similarly, in a patient with preserved left ventricular systolic function, normal wall motion, left ventricular hypertrophy, and a largely unchanged, but elevated troponin level on serial testing (i.e., not a characteristic rise and fall), it should be documented that a type 2 MI has been "ruled out" and that non-MI troponin elevation due to hypertensive emergency and left ventricular hypertrophy is the most likely diagnosis.

10. *If possible, avoid using the term "MINOCA" (or myocardial infarction with nonobstructive coronary arteries) in clinical documentation.* Per the new Universal Definition, the term MINOCA is used to describe patients with an elevated troponin level presenting with symptoms of acute myocardial ischemia and/or new ischemic ECG changes, but without obstructive coronary artery disease (1). In this context, the term "MINOCA" is an anatomic descriptor and does not replace the necessity of documenting the pathophysiology of the condition, which drives therapeutic decisions, coding practices, outcomes from quality programs, and reimbursement. Additionally, using a term like MINOCA may increase the risk that that it will be misapplied to patients having a non-MI troponin elevation due to conditions such as decompensated heart failure, defibrillator shocks, and so on

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