

Problems with myocardial infarction definitions

(DECEMBER 2013)

TO THE EDITOR: In the December 2013 *Cleveland Clinic Journal of Medicine*, Tehrani and Seto provide a review of the updated definitions of myocardial infarction (MI).¹ A key concept incorporated into the structured definitions is that cardiac biomarkers must be interpreted in a clinical context.² This in turn helps better align the laboratory and clinical findings with the pathophysiologic processes.

However, there is another dimension to the definitions that is sometimes overlooked and requires careful attention: translation of the definitions into codes and comparable databases. Accurate and consistent coding according to the International Statistical Classification of Diseases, ninth edition (ICD-9), and the ICD-10 is critically vital to the appropriate analysis of data, research, quality measurement, and reimbursement of services related to MI. Unfortunately, there is no straightforward translation of the definitions into ICD-9 codes, and the challenge is further confounded when it comes to ICD-10, which will be implemented in October 2014.

The ICD-10-CM Index to Diseases does not yet recognize this nomenclature. ST-elevation MI is the default for the unspecified term “acute MI.” Non-ST-elevation MI requires more explicit documentation and is classified based on whether it occurs during or after a variety of procedures. Type 2 MI is particularly challenging because of the several possible ways to code the condition—for example, as acute subendocardial MI (I21.4), demand ischemia (I24.8), or acute MI, unspecified (I21.9). Coding guidelines are assumed to standardize the approach to coding these conditions, but there is no guarantee that comparability of the data will endure biases of code assignment. Although extreme precision in disease capture by coding may not exist, other clinical conditions have better correlations with coding classifications, such as stages of chronic kidney disease

ranging from stage 1 through end-stage renal disease (N18.1 through N18.6). Furthermore, ICD-10 codes are insufficient to clearly distinguish the type of acute MI.³

While the concept of acute MI applies when the stated date of onset is less than 8 weeks in ICD-9,⁴ it changes to 4 weeks in ICD-10. “Acute” can reference an initial or a subsequent MI in ICD-10, but it does not define the time frame of the MI.⁵ This is different than in ICD-9, where the concept of “subsequent” refers to a “subsequent episode of care.”

On the surface, these variations may not seem significant. However, the discriminatory efforts to better define a patient’s clinical condition using the new definitions may get diluted by the challenges of the coding process. The implications on comparability of quality metrics and reporting are not to be underestimated and need to be assessed on a national level.

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IN REPLY: We thank Dr. Antonios for his comments regarding the current shortcomings of the ICD-9 and ICD-10 coding systems in describing the acute MI types as defined in the universal definition. We share his concern that accurate and consistent coding of MIs may be difficult when the definition

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of MI changes over a short period of time. Such changes create a disconnect not only between our clinical terminology and coding systems, but also potentially between our conventional sense of a “heart attack” as an acute coronary syndrome or a clinically significant infarction rather than a small troponin elevation from demand ischemia. This has consequences not only for quality measures and reporting, but also for clinical research trials and clinical care. This is exemplified by reports of recent trials that were possibly prematurely discontinued, as the use of troponin thresholds may conflate large MIs with clinically insignificant ones.¹

Recently, the Society for Cardiovascular Angiography and Interventions published a new definition of “clinically relevant” MI after revascularization.² Rather than relying on troponins, which are elevated in as many as 24.3% of uncomplicated percutaneous coronary interventions and in 42% to 82% of uncomplicated coronary artery bypass grafting procedures (based on the 2007 universal definition), they point to extensive literature documenting that only patients with elevated creatine kinase MB more than 10 times the upper limit of normal after revascularization have a worsened prognosis. We favor this clinically relevant MI definition for post-revascularization MI. We also favor the use of creatine kinase MB as a less sensitive but more specific confirmatory marker for acute coronary syndromes (type 1) or clinically significant supply-demand (type 2) MI, when the symptoms or electrocardiographic signs are nondiagnostic, as they often are.³ However, until there is a consensus around a single definition, clinicians are effectively walking around a Tower of Babel and must take care to be specific when documenting an MI.

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Myasthenia gravis

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TO THE EDITOR: Dr. Li and colleagues provide a well-written article about what is generally believed regarding myasthenia gravis (MG). However, like most reviews, it perpetuates the myths surrounding current medical practice, resulting in delays in diagnosis, treatment initiation, and insurance approval and reimbursement, and therefore increased morbidity and mortality. Stricter statistical and editorial review is needed and what is known and unknown clearly stated. Patients, in particular those of us who are physicians ourselves, recognize that this is no academic quibble.

Myasthenia gravis was a clinical diagnosis until blood tests began to pick up antibodies. If the blood tests have to be positive to diagnose MG, then everyone diagnosed with MG will have positive blood tests. If the muscle studies have to show particular abnormalities to diagnose MG, then everyone diagnosed with MG will have those abnormalities. It makes doctors more comfortable to have these evidences of their understanding verified, but it does not help any of the patients who do not meet the testing criteria but have the clinical findings.

We know, in contrast to what was thought a number of years ago, that there are “seronegative” patients with clinical evidence of myasthenia who are antibody-positive. For those who are MuSK-positive, their problem is now well described, and although it affects a different part of the neuromuscular

junction, it remains under the MG umbrella. We also know there are other antibodies, for which we have no commercial tests, in patients with symptoms of MG who respond to treatment for autoimmune problems. This article is relatively dismissive of the clinical validity of those antibodies, and certainly a degree of skepticism is a good thing as long as the patients remain diagnosed and treated.

It is of more than academic interest that these misconceptions and prejudices be recognized. At the very least, editorial boards should insist that statistics in papers reflect the diagnostic skills of the authors. If over 95% of an author's diagnosed patients are seropositive, then one can suspect there is heavy reliance on blood studies for diagnosis, and rejection of those who do not meet those criteria. The statistics should read "over 95% of patients *we diagnose* with MG have positive blood studies" rather than "over 95% of patients with MG have positive blood studies." Dismissing a significant portion of a patient population will also affect treatment statistics, which then should read that "for those who meet this criteria, __% will respond to..."

If patients meet clinical criteria for the diagnosis of MG and a large percentage do not have positive serology, then more research needs to be done into their particular autoimmune problems, and better testing may become commercially viable. Recognizing the problem will lead to better clinical diagnosis and treatment, and strict diagnostic criteria would permit their inclusion in studies. For many of us this would create a more open and questioning atmosphere as to our un-

derstanding of the spectrum of autoimmune myasthenia and the ability and willingness to diagnose and treat "seronegative" autoimmune myasthenia when we see it.

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IN REPLY: We appreciate Dr. Keiter's comments. We agree that myasthenia gravis, like most medical disorders, rests on clinical diagnosis. We have patients we treat for myasthenia gravis in the absence of the typical serological confirmation. A very few of these patients with restricted oculobulbar symptoms may also have normal single-fiber EMG studies. In this situation, the decision to treat an individual for myasthenia gravis must rest on the physician's clinical judgment, but also on the patient's understanding that the condition does not have the diagnostic support often seen. The decision to treat with medications that have potential severe side effects requires the patient's understanding of the context in which the diagnosis is being made and the specific treatment is being suggested.

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