Biomarkers in Acute Cardiac Disease

In their state-of-the-art paper on biomarkers in acute cardiac disease (1), Dr. Jaffe and colleagues list creatine kinase-MB (CK-MB) as a “potentially outdated marker.” However, CK-MB has a specific utility in the diagnosis of reinfarction (2), and it cannot be replaced by the cardiac troponins for this purpose. By following up the time course of rise and fall of CK-MB, an interruption in the progressive decline in the level of the biomarker (to levels below upper reference limit) can be detected (2–4). Re-elevations in CK-MB by more than 50%, can be used to diagnose re-infarctions as early as 18 h after the index event (2). Both cardiac troponin T (cTnT) and cardiac troponin I (cTnI) on the other hand are continuously released from degenerating contractile apparatus in necrotic cardiomyocytes and may show persistent elevations, 7 to 10 days in the case of cTnI and up to 10 to 14 days in the case of cTnT, after the index event (2). The protracted time course of kinetic release of cTnI and cTnT limit their ability to diagnose reinfarction even several days after the index ST-segment elevation myocardial infarction (STEMI) because the cardiac troponin levels will still be on the rise during this period as a result of their normal kinetics, and it is not possible to be sure whether the rise is due to a re-infarction or not. It is because of this important difference in the kinetics between CK-MB, which shows a rapid rise and fall, and the troponins, the American College of Cardiology/American Heart Association Practice guidelines for STEMI specifically state that CK-MB is superior for diagnosing reinfarction (2). This is very relevant as recurrent chest pain is a common complaint of patients admitted for myocardial ischemia and CK-MB plays a vital role in the further evaluation of this complaint.

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down slope at all and those criteria are likely very insensitive. We now know that reinfarction is easily detected with troponin by the presence of a rising pattern of values (8). In the vast majority of patients, changes in troponin, presuming sensitive assays and recommended cut off values, occur rapidly, obviating the delay in the occasional patient in whom decision making might depend on the values (9). Because troponin is more sensitive, it should provide a clearer signal on the down slope than CKMB and several groups are considering a recommendation of a 20% to 25% change in that situation. Those criteria too will likely be insensitive but will prevent false-positive diagnoses as well. Once one starts to rely on troponin values, the ease with which they can be used becomes easily appreciated and their use becomes part of good common sense. Hanging onto the past adds costs and retards the ability of clinicians to learn how easily many of these issues can be resolved.

The issue Dr. Fye highlights is that we need to be smarter about how we respond to elevations of troponin both acutely and longer term. Troponin elevations should never trump common sense, but we must also be open to the possibility that at times, especially in a busy world, we may need to allow our common sense to evolve as the science of our discipline improves and to acknowledge a responsibility to work up patients with abnormal troponin values as outpatients when appropriate rather than ignore such elevations. All of that would be good common sense.

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