Vasospasm and Type 2 AMI?

In 2007, an attempt was made to define types of acute myocardial infarctions. A type 1 was defined as a spontaneous event due to a plaque rupture, causing symptoms and signs and a rising or falling pattern of cardiac troponin (cTn). Other spontaneous events based on the same diagnostic criteria but associated with vasospasm, endothelial dysfunction, dissection, or supply–demand imbalance were termed type 2 acute myocardial infarctions. There have been many approaches in defining type 2 acute myocardial infarction. One that is problematic is using an isolated elevation of cTn to define acute myocardial infarction, because there are many circumstances in which elevations occur due to structural heart disease, renal dysfunction, sleep-disturbed breathing, drug toxicities, and the like. This problem is more complex in patients who manifest more subtle presentations, such as older, diabetic, or post-operative patients in whom symptoms may be ambiguous, and electrocardiographic changes may not be overt. This distinction will be more important with high-sensitive cTn (hs-cTn) assays because type 2 acute myocardial infarctions cause less cTn release, and thus, type 2 acute myocardial infarctions will increase more than type 1 events.

Into this realm comes the present manuscript by Matsue et al in this issue of The American Journal of Medicine. They categorized patients with angiographically defined vasospastic angina into those with and without cTn elevations. They suggest those with troponin elevations have type 2 acute myocardial infarction. That is in part reasonable because they have eliminated patients with renal failure and some of the other comorbidities associated with cTn elevations, but the absence of a changing pattern of values is still problematic. A similar construct has been suggested after noncardiac surgery. This approach has the potential to lead to misdiagnoses, over-testing, and over-treatment. Thus, their diagnosis of type 2 acute myocardial infarction is not totally secure. Also, a variety of cTn assays were used. One would expect that the more sensitive the assay, the more common the elevations. With high-sensitivity assays coming to the US soon, even more elevations will be found.

Despite these issues, this report advances the field by showing that those individuals with elevated cTn do worse; adding to information indicating that an elevated cTn is an independent risk factor for adverse outcomes.

One might know this from the clinical characteristics of the patients because those with cTn elevations were sicker. One then could ask whether cTn was helpful clinically because one might argue that knowing a given patient is at an even greater risk than the increased risk marked by the clinical parameters might not be incrementally important.

How might variant angina cause cardiac injury? First, it should be clear that one can have coronary artery disease with variant angina. Because the cross-sectional area of a tube is related to the square of the radius, it takes only modest vasoconstriction to change an insignificant lesion into one that is severe. In addition, marked vasoconstriction due to variant angina causes procoagulant activity and perhaps, thrombotic occlusion. Finally, with more sensitive cTn assays, transient occlusion alone may be sufficient to cause cardiac injury. Studies of patients with stable angina indicate that individuals with more vulnerable-appearing lesions have elevated hs-cTn values. This could be because these patients have intermittent periods of ischemia severe enough to cause cardiac injury.

Whether these mechanisms can be extended to patients with endothelial dysfunction as suggested is less clear. Although many patients do not have culprit lesions at the time of angiography, because endothelial dysfunction is common in patients with coronary artery disease, it is seductive to suggest when one sees plaque alone that vasoconstriction is responsible. However, the timing of the angiogram is key. In addition, because plaque rupture is the way in which coronary artery disease progresses, it may be found with either type 1 or type 2 myocardial infarction.

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The task for operationalizing the criteria for type 2 acute myocardial infarction is ongoing. This paper adds some potentially important information to that discussion.

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References


