Identifying and classifying myocardial infarctions

Long ago, an acute myocardial infarction (MI) was diagnosed by a combination of patient history, electrocardiographic (ECG) findings, elevation of aspartate aminotransferase and creatine kinase (CK), and the pattern of lactate dehydrogenase isozymes. The erythrocyte sedimentation rate was useful in distinguishing prolonged angina from an MI, and even the presence or absence of leukocytosis was sometimes a determining diagnostic test. The recognition that CK-MB was fairly specific for myocardial injury was a major step forward in the diagnosis of what would later be called the acute coronary syndrome.

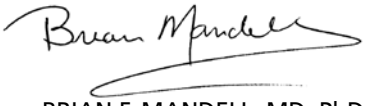
But way back then, although the patient (and the physician) were often diaphoretic, the acute diagnosis was of limited significance to acute management. We rushed to put the patient to bed rest, started the lidocaine drip at the first sign of a few premature ventricular contractions, slapped on the oxygen prongs, got serial electrocardiograms to watch for conduction blocks—and a few forward thinkers began heparin drips.

As therapeutic options became more interventional, the need for rapid diagnostic tests and better biomarkers of prognosis became more critical. ST elevations took on new meaning, but the major diagnostic advance was the incorporation of cardiac troponin into our diagnostic algorithm.

On page 159 in this issue of the *Journal*, Drs. Shaun Senter and Gary Francis discuss the power of these tests in the diagnosis of acute MI. They are not perfect tests. Acute pericarditis can still present diagnostic challenges, with sometimes confusing ECG findings, and almost a third of patients have elevated troponins (Bainey KR, Bhatt DL, *Mayo Clin Proc* 2009; 84:5–6; Imazio M, et al, *J Am Coll Cardiol* 2003; 42:2144–2148). Troponins may occasionally be elevated in acute severe heart failure and aortic dissection. In my practice, elevation of troponins may be difficult to interpret in the setting of chronic inflammatory muscle disease; it is not always easy to distinguish whether the leakage of these biomarkers is from injured regenerating skeletal muscle or from cardiac muscle.

When treating patients with a possible acute coronary syndrome, prompt diagnosis and intervention are often warranted, but the risks of using thrombolytic therapy inappropriately in the setting of pericarditis or an acute intracranial process with ECG changes are substantial.

Senter and Francis review for us the latest "precise definition" of acute myocardial infarction and provide a commentary on the utility of different diagnostic tests. They also highlight the value of using different diagnostic modalities to obtain the information we need for prognostication and treatment decisions.



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