Myocardial Infarction with Normal Coronary Arteries: A Role for MRI?

To the Editor:

We recently cared for several patients with chest discomfort characteristic of coronary disease, electrocardiogram (ECG) changes, and increasing troponin concentrations. Coronary angiography revealed mild or no coronary artery disease (CAD), so we evaluated these patients with contrast-enhanced cardiac MRI. Our data suggest that these patients had myocardial infarction (MI).

With Institutional Review Board permission, we reviewed our angiography database for patients with normal coronary arteries or mild coronary artery disease during the period from January 2005 to November 1, 2006, to augment those cases we found clinically. Inclusion criteria included presentation with acute coronary syndrome, increased serum troponin T concentration (>0.01 μg/L), or mild or absent CAD by angiography and a contrast-enhanced cardiac MRI (CE-CMR) considered diagnostic for infarction. Patients with prior infarction, known CAD, heart failure, pulmonary embolism, or suspected pericarditis/myocarditis were excluded. Eight patients met these criteria. All angiogram results were reviewed by one of the investigators (V.M.).

CE-CMR studies performed on a GE Signa CVi system (GE Medical Systems) were reviewed by J.F.B., who had no knowledge of other clinical data. Regional wall motion was characterized as normal, hypokinetic, or akinetic. Delayed enhancement was described as involving the endomyocardial border and transmural as involving the epicardial border alone without transmural extension or a midseptal myocardial stripe. Only solitary areas of subendocardial enhancement were considered diagnostic for infarction.

Six of the 8 identified patients (75%) were women. Risk factors for CAD were present in all patients except one. Hypertension, present in 75%, was the most common risk factor. Troponin increases had a rising and falling pattern, with a mean peak increase of 1.10 μg/L (range, 0.34–2.13 μg/L). Patterns of delayed hyperenhancement involved only the subendocardium (Fig. 1). Five of the patients had enhancement involving ≤50% of the myocardial wall; the remainder had at least 50% hyperenhancement. The inferior and lateral walls were involved in 6 patients (75%); the apical wall only was involved in 1 patient and anterior wall only in 1 patient. Mean ejection fraction, determined by MRI, was 60% (range, 40%–74%). Vasospasm was found in 2 of 3 patients who underwent coronary vasospasm study. All patients were treated for MI, and all were alive at a mean of 26 months (range, 6–59 months) later.

A subset of patients with clinical findings characteristic of acute MI have no or mild CAD. These patients meet the definition for acute MI, with increases in troponin combined with ischemic symptoms, pathologic Q waves, and/or ischemic ECG changes (1). In the absence of the finding of occlusive epicardial CAD, however, there is reluctance to diagnose infarction in these patients because troponin increases, although specific for cardiac damage, can occur for many reasons.

Ischemia and even myocyte injury can occur in the absence of overt epicardial CAD. It is possible that coronary occlusion resolved before angiography, but we also acknowledge that coronary angiography is

References


Robert M. Bossarte1
Mary Jean Brown2*
Robert L. Jones3

1 Department of Community Medicine
School of Medicine
West Virginia University
Morgantown, WV

2 Lead Poisoning Prevention Branch
Division of Emergency and Environmental Health Services
National Center for Environmental Health
Centers for Disease Control and Prevention
Atlanta, GA

3 Inorganic Toxicology and Radionuclide Laboratories
Division of Laboratory Services
National Center for Environmental Health
Centers for Disease Control and Prevention
Atlanta, GA

* Address correspondence to this author at: Lead Poisoning Prevention Branch, Centers for Disease Control and Prevention, 4770 Buford Highway, NE (MS-F40), Atlanta, GA 30341. Fax 770-488-3635; e-mail mjb5@cdc.gov.

DOI: 10.1373/clinchem.2006.082404
not a perfect tool (2). Epicardial CAD or alterations in coronary vasomotion that changed the severity of epicardial coronary lesions could have been missed or underestimated. We believe that mechanisms such as these were the most likely etiologies for the troponin increases in our patients.

Although troponin increases can occur in response to moderate to severe pulmonary embolism with acute right heart overload, heart failure, and myocarditis, these were not present clinically in our patients. CE-CMR is an accurate method for detecting MI and has high sensitivity and excellent spatial resolution (3). The delayed hyperenhancement seen in our patients is not specific for infarction, but we observed solitary subendocardial locations typical for MIs (4), and we were able in all cases to rule out other diseases that present this way. We cannot exclude myocarditis, but the changes seen on cardiac MRI were not typical for this disorder (5), which usually shows one or several foci of contrast enhancement, most often in the left epicardial ventricular free wall. We are unaware of documented cases of myocarditis involving a solitary area of the subendocardium. Our data are similar to a small series recently reported (2) in that most patients were women and most of the abnormalities were in the inferior wall.

In these patients presenting symptoms typical of ischemia accompanied by ECG changes and biomarkers classic for MI, the MRI images were also diagnostic. Perhaps MRI detected old insults, but our data suggest that when patients have a classic presentation, one needs to be cautious in relying solely on angiography for definitive information.

Grant/funding support: None declared.

Financial disclosures: Dr. Jaffe is a consultant to Dade-Behring and Beckman-Coulter, and he receives research support from them. He is or has been a consultant to most of the major diagnostic companies. The authors have no other disclosures to declare.

References


