

Serial determination of troponin concentrations in the diagnosis of acute myocardial infarction

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Abstract

Contemporary guidelines for the definition of acute myocardial infarction recommend the presence of an increase or decrease in troponin concentration in patients presenting to the hospital with chest pain and electrocardiogram changes. Here we report the case of a 49-year-old woman with recurring chest pain, nausea, and diaphoresis with initially undetectable troponin concentrations that subsequently increased. This case demonstrates the importance of serial determination of troponin concentrations and changing biomarker patterns in the management of patients presenting with possible cardiac ischemic injury.

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Case report

In April 2008, a 49-year-old woman presented to the emergency department after three episodes of chest pain, the first of which had occurred 6 h before presentation. The chest pain was described as a sensation of tightness associated with shortness of breath, which awoke her during the night. The second and third episodes were accompanied by mild nausea and diaphoresis. The patient was free of pain at the time of presentation. Her personal medical history included essential hypertension, under medical control. She had been told that her cholesterol was high but, given her high high-density lipoprotein concentrations, she did not require treatment. She had recently returned from a long vacation during which she and her spouse had driven across the country.

The patient's electrocardiogram (ECG) in the emergency department showed normal sinus rhythm with minor anterior T-wave abnormalities. Blood tests were unremarkable, including a cardiac troponin T (cTnT) value less than 0.01 ng/mL. Chest X-ray showed clear lungs, normal heart size, and a tortuous aorta. The D-dimer concentration was mildly increased.

The patient was placed in the observation room. At approximately 4 h after presentation, her ECG was unchanged, but her cTnT concentration had increased to 0.02 ng/mL. At 6 h after the patient's admission, the third cTnT value was 0.03 ng/mL, indicative of a pattern of increasing values. Because of this increasing pattern, the patient was admitted from the emergency department to a cardiology service. Aspirin had been given in the emergency department, but, because of the modest cTnT concentrations, the

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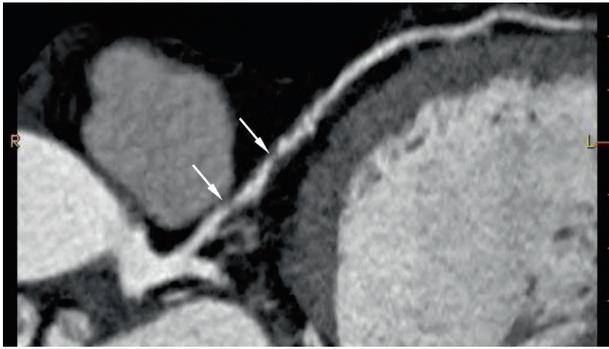


Figure 1. Computed tomography contrast-enhanced coronary angiogram of the left anterior descending artery. Arrows indicate noncalcified lesions.

patient was not treated with heparin or a GpIIb/IIIa inhibitor. She did, however, receive a β -blocker. A computed tomography (CT) angiogram was obtained to evaluate for possible pulmonary embolism and to assess her coronary arteries noninvasively. Computed tomography excluded pulmonary emboli, but revealed discrete noncalcified lesions in her proximal and mid left anterior descending (LAD) coronary artery, estimated at 75% each (Figure 1). The distal LAD was normal. Shortly thereafter, the patient developed severe chest pain, diaphoresis, and nausea. The ECG showed changes suggestive of an acute antero-septal myocardial infarction, so she was taken expeditiously to the cardiac catheterization laboratory. Coronary angiogram revealed that the proximal LAD was 100% occluded and the mid LAD had a discrete 70% obstruction lesion; the distal segment was of normal size. The proximal circumflex artery was 30% obstructed by a single discrete lesion, and the proximal right coronary artery was 20% obstructed by a single discrete lesion. Each of the two LAD lesions was treated with a bare metal stent. The patient continued to take aspirin 325 mg daily and was discharged from hospital 3 days later, receiving clopidogrel 75 mg, lisinopril 5 mg, metoprolol 200 mg, and simvastatin 40 mg, all daily.

Comment

This case report emphasizes the importance of a pattern of increasing cTn values. The concentrations of cTn currently measurable with contemporary assays are far greater than those we now consider to be normal values [1], therefore increases in cTn are usually important as a marker of a cardiac abnormality [2]. There can be analytic false-positives, but they are uncommon and rarely manifest a changing pattern of values. Thus an increasing pattern should prompt additional investigation.

In this patient, because of the antecedent history of a long car journey and a mildly increased D-dimer value, the diagnosis of pulmonary embolism was

considered. Pulmonary embolism can cause increases in cTn, but, when they occur, they usually mark quite large pulmonary emboli. In this case, the patient appeared very stable, so a large pulmonary embolism was unlikely. However, given the ability of CT angiography to evaluate both the pulmonary circulation and the coronary arteries, the decision was made to obtain a CT angiogram, which excluded pulmonary embolism but documented significant coronary artery disease. This is perhaps not unexpected, because coronary artery disease is common in our society. However, we now understand that many other diseases can also be associated with increases in cTn concentrations. In this context, myocarditis appears to be a common mimicker of coronary artery disease [3] in patients who present acutely. In addition, toxic metabolic insults such as carbon monoxide poisoning can cause cardiac injury. Critically ill patients [4,5] often have a pattern of increasing values of cTn. Some of these increases may be the result of occult coronary artery disease with or without supply–demand abnormalities, but they can also be result from hypo- or hypertension, or medications such as catecholamines that are used to treat these patients. Regardless of the etiology, increases in troponin concentration appear to be highly prognostic, in both the short and longer terms [4,5].

On occasion, no etiology is apparent, and that reflects the fact that there are causes for cardiac injury of which we are unaware or for which we lack knowledge as to their assessment. A word of caution is necessary, however. At times, we overestimate the accuracy of some of our tests. We [6] and others have reported a pattern of acute myocardial infarction (AMI) that was revealed by magnetic resonance imaging in patients, mostly women, with what appeared to be normal coronary arteries on coronary angiography. Therefore, in our view, an apparently normal coronary angiogram does not always exclude AMI, especially in women.

This case report illustrates the importance, as recommended by the recent guidelines for the diagnosis of AMI [7], of serial determination of troponin concentrations, and the significance of a changing pattern of troponin concentrations, in the management of patients presenting with possible cardiac disease. ■

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