Acute Coronary Syndrome Clinical Presentations and Diagnostic Approaches in the Emergency Department

Maame Yaa A.B. Yiadom, MD, MPH

KEYWORDS

• Acute coronary syndrome • Chest pain
• Cardiac biomarkers • Cardiac ischemia

A 47-year-old woman with a history of gastroesophageal reflux disease, hypertension, and hyperlipidemia arrives in the emergency department complaining of shortness of breath for 4 hours. It began abruptly and is accompanied by nausea and vomiting. She has had these symptoms before, but they resolved with Maalox and were never this uncomfortable. Her vital signs are normal, she is given a lidocaine and Maalox suspension and zofran, which alleviates her discomfort. Her electrocardiogram, basic laboratory studies, and troponin sent on arrival are normal. Two hours later she looks and feels well. She is discharged home with a diagnosis of noncardiac chest pain and reflux disease exacerbation. Seven hours later, she returns to the emergency department poorly responsive with an electrocardiogram diagnostic of a ST-elevation myocardial infarction. In the coronary catheterization laboratory she is found to have a fully occlusive lesion in her left anterior descending artery.

Many discussions of cardiac ischemia start with chest pain. It is a chief complaint that captures attention because it is one of the most common emergency department (ED) patient presenting complaints, and is associated with potential life-threatening diagnoses, such as acute coronary syndrome (ACS), pulmonary embolism, and aortic dissection. However, the identification of cardiac ischemia, as is captured within the spectrum of ACS, requires one to cast a broader net. There are now years of research showing that nonclassic ACS symptoms (eg, shortness of breath, fatigue, and nausea)

This author has nothing to disclose.
Department of Emergency Medicine, The Cooper Heart Institute, Robert Wood Johnson Medical School, Cooper University Hospital, 1 Cooper Plaza, Camden, NJ 08103, USA
E-mail address: myiadom@gmail.com

Emerg Med Clin N Am 29 (2011) 689–697
doi:10.1016/j.emc.2011.08.006 **emed.theclinics.com**
0733-8627/11/$ – see front matter

are common and do not reflect less severe disease.[1–4] These are the patients in whom a diagnosis of ACS is likely to be missed,[5] and the biggest medicolegal risk as a specialty.[6,7] There were 124 million United States ED visits in 2010.[8] It is challenging to identify the 16% that will have ACS, particularly when the symptoms are more subtle.[9]

ACS CONTINUUM

ACS is a specific physiology that results in myocardial injury where a thrombus forms on an acutely ruptured acute coronary artery wall to heal the defect. If this clot obstructs the artery's lumen, it can diminish blood flow to tissues beyond the lesion. Diminished flow leads myocardial ischemia, or the process of cells starving for oxygen and other nutrients delivered by the blood.[10] When this process leads to cells starving to death, myocardial infarction occurs and myocardial cell components are released into the blood. The experience of injury from this physiology is represented by three clinical entities: (1) ST-elevation myocardial infarction (STEMI), (2) non-ST elevation myocardial infarction (NSTEMI), and (3) unstable angina (UA). Acute myocardial infarction (AMI) is a subset of ACS that includes both STEMI and NSTEMI.

STEMI is the most urgent and severe condition within ACS. It represents a complete occlusion of a coronary artery leading to full-thickness myocardial infarction, and its diagnosis is based solely on the presence of ST segment elevation on the electrocardiogram (ECG) that meets specific STEMI criteria.[11,12] NSTEMI represents myocardial cell loss and can be identified either by ECG changes or a positive biomarker evaluation, which typically consists of two samples of cardiac enzymes where one is drawn on arrival and the other hours later.[13] The diagnosis of UA is a clinical diagnosis made based on the report of symptom quality and duration. It represents a patient with known coronary disease (CAD) causing a coronary flow limitation who reports a recent history of symptoms that are accelerating or induced with less activity. Also included in this category are patients without a history of angina or CAD who are presenting with a report of symptoms concerning for ischemia without evidence of myocardial cell infarction. These patients are at risk for AMI and can evolve to have a NSTEMI or STEMI. The diagnosis of UA can only be confirmed in the setting of normal or unchanged ECGs, and a negative biomarker evaluation. Until this occurs NSTEMI should be included as the potential diagnosis.[14]

Cardiac biomarkers, typically troponin and creatine kinase-MB (CKMB) levels, serve as tests for myocardial injury. When ACS is suspected, signs of acute myocardial injury are assumed to be from ischemia-caused infarction, or primary (Type I) ACS.[15] However, this is not always a direct relationship. Myocardial injury may be the consequence of another medical condition stressing the heart. This is often referred to as "demand ischemia" (Type II ACS). In addition, other myocardial injury mechanisms, such as direct trauma and surgical manipulation, can elevate serum troponin levels. False elevations can occur because of poor serum troponin clearance, as often seen in end-stage renal disease patients.

Early treatment of ACS is the essential element in reducing morbidity and mortality. Because cardiac biomarker results have become available within the time frame of an ED evaluation, the burden of diagnosis, treatment, and mobilization of definitive therapy is a focus of emergency medical care. One approach to reducing the frequency of missed AMI is to test patients more liberally. However, this poses two problems. First, any test's negative predictive value and positive predictive value are dependent on the prevalence of disease in the test population. If those without concerning symptoms receive the test, its usefulness is reduced.[16] Second, this