Chest Pain Evaluation in the Emergency Department

Andrew J. Foy, MD[a], Lisa Filippone, MD[b,*]

KEYWORDS

• Chest pain • Myocardial infarction • Acute coronary syndrome

KEY POINTS

- Chest pain is a common chief complaint in the emergency department; approximately 9% of patients with chest pain are diagnosed with an acute coronary syndrome (ACS).
- ACS presents as a range of clinical conditions including unstable angina, non–ST-segment elevation myocardial infarction (MI), ST-segment elevation MI, and sudden cardiac death.
- The patient history and electrocardiogram are very important for early recognition of ACS.
- Type I MI is owing to coronary artery thrombosis, whereas type II MI represents myocardial injury from a variety of underlying processes not related to intracoronary thrombosis.
- Advanced noninvasive testing in patients who rule out for ACS is low yield from a diagnostic standpoint and has not been found to improve patient outcomes.

INTRODUCTION

Chest pain accounts for 5.5 million or approximately 9% of all non–injury-related emergency department (ED) visits for adults in the United States each year.[1] The development of catheter-based therapies and regional care networks allowing for rapid transfer of patients to centers with advanced treatment options has contributed to declining acute coronary syndrome (ACS) case fatality rates. Data from the Nationwide Inpatient Sample shows the overall case fatality in the United States for ST-segment elevation myocardial infarction (STEMI) declined 3% between 1993 and 2009.[2] A community study of patients hospitalized with incident myocardial infarction (MI) in Olmsted County, Minnesota, found the age- and sex-adjusted hazard ratio of death within 30 days for an MI occurring in 2006 compared with 1987 was 0.44.[3]

Despite improvements in the acute care of patients with ACS, it accounts for only a small percentage (9%) of all ED visits for chest pain[4] and there is now concern that current diagnostic strategies could be contributing to the overtreatment of obstructive

[a] Division of Cardiology, Heart and Vascular Institute, Milton S. Hershey Medical Center, Penn State University, 500 University Drive, Hershey, PA 17033, USA; [b] Department of Emergency Medicine, Cooper Medical School of Rowan University, One Cooper Plaza, Camden, NJ 08103, USA
* Corresponding author.
E-mail address: filippone-lisa@cooperhealth.edu

Med Clin N Am 99 (2015) 835–847
http://dx.doi.org/10.1016/j.mcna.2015.02.010 **medical.theclinics.com**
0025-7125/15/$ – see front matter © 2015 Elsevier Inc. All rights reserved.

coronary artery disease (CAD) in patients presenting with chest pain without evidence of ACS. From 1999 through 2008, the use of advanced medical imaging in patients with chest pain increased 368%.[1] This was largely undertaken in an effort to avoid missing ACS. However, although this strategy has indeed led to the increased detection and treatment of obstructive CAD, it has not translated into a reduction in cardiac events. A cross-sectional, population-based sample of Medicare patients from 1993 to 2001 found that overall hospitalizations for acute MI (AMI) remained flat at 8.7 per 1000 patients despite significant increases in imaging and revascularization rates.[5]

This article discusses the evaluation of patients presenting to the ED with chest pain and suspected ACS. It emphasizes important points of the pathophysiology, patient history, and physical examination, as well as clinical findings related to ACS. It will also discuss recommendations regarding ordering cardiac troponin (cTn) and the use of advanced noninvasive testing in this patient population.

PATHOPHYSIOLOGY

The term 'acute coronary syndrome' refers to any condition that is brought on by a sudden reduction in blood flow to the heart. It represents a spectrum of clinical conditions including unstable angina, non–ST-segment elevation MI (NSTEMI), and STEMI. Unstable angina is characterized by an unstable pattern of ischemic cardiac discomfort without increased cTn levels or ST-segment elevation on the electrocardiogram (ECG). It may or may not present with horizontal or down-sloping ST depression or dynamic T-wave changes on the ECG. NSTEMI is characterized by increased cTn levels. It too may or may not present with evidence of ischemia on the ECG. STEMI is characterized by ST-segment elevation on the ECG owing to transmural (full-thickness) necrosis of the myocardial segment(s) involved. cTn may be negative in the early stage of STEMI because cell lysis and release of intracellular components into the bloodstream do not occur immediately upon myocardial cell death.

AMI is defined by a 20% increase and/or decrease in cTn (I or T), the preferred myocardial biomarker, with 1 value exceeding the 99th percentile upper reference limit (URL) of a normal population.[6] MI can result from a variety of clinical conditions and the etiology of AMI is important to differentiate because it significantly affects patient management. The Third Universal Definition of MI specifies 5 types (**Table 1**).[6] For the purposes of patient evaluation and management, it is best to classify MI into 2 broad categories: (1) MI owing to acute coronary artery thrombosis or (2) MI not owing to acute coronary artery thrombosis, the causes of which are extensive (**Box 1**). The backbone of treatment for patients in the first category is administration of antiplatelet and anticoagulation therapy with or without percutaneous coronary intervention (PCI), which is intended to stabilize the process of acute thrombosis and restore coronary flow. Treatment for patients in the second category is directed at treating the underlying condition responsible for causing oxygen supply/demand imbalance or other myocardial injury (volume resuscitation, presser support, and antibiotics for patients in septic shock).

Coronary artery thrombosis is caused usually by either atherosclerotic plaque rupture or plaque erosion. In each case, there is a loss of integrity in the protective covering over an atherosclerotic plaque, formed by a single layer of endothelial cells, thus exposing the thrombogenic components of the necrotic core to the bloodstream.[7] The resulting intracoronary thrombus is composed of platelet aggregates layered with fibrin, red blood cells, and acute inflammatory cells.[7] Plaque rupture or erosion often occurs at sites without significant luminal stenosis.

One study of 3747 patients in the National Heart, Lung, and Blood Institute Dynamic Registry undergoing PCI found that 216 (5.8%) required additional PCI of the

Download English Version:

https://daneshyari.com/en/article/3792691

Download Persian Version:

https://daneshyari.com/article/3792691

Daneshyari.com