Evaluation of Chest Pain and Acute Coronary Syndromes

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KEYWORDS

• Chest pain • Acute coronary syndrome • Troponin • Coronary CT angiography

KEY POINTS

- Chest pain is a common complaint in the emergency department, but less than 15% of patients are diagnosed with acute coronary syndrome.
- Risk stratification tools incorporating cardiac troponins have created new accelerated diagnostic pathways that are highly accurate and sensitive.
- Coronary computed tomography angiography can be used in low-risk to intermediate-risk patients for diagnostic testing with high accuracy.

INTRODUCTION

Chest pain currently represents the second most common chief complaint of patients presenting to emergency departments (EDs) in the United States, representing approximately 8 million visits.[1] Only 10% to 20% of patients are ultimately diagnosed with acute coronary syndrome (ACS), with only one-third with acute myocardial infarction (AMI).[1,2] Despite decades of research, 2% to 10% of ACS cases are still missed.[3,4] Although the tools for evaluating chest pain continue to evolve and improve, translating these new tests and imaging studies into an updated but meaningful and efficient clinical evaluation within the time and resource constraints faced by emergency care providers can be a complicated and often confusing task.

Myocardial Ischemia is a Spectrum

ACS encompasses both unstable angina and AMI.[5] In ACS, atherosclerotic plaque rupture and platelet-rich thrombus develop. Coronary blood flow is reduced, and myocardial ischemia occurs. The degree and duration of the oxygen supply-demand mismatch determines whether the patient develops reversible myocardial ischemia without necrosis (unstable angina) or myocardial ischemia with necrosis (myocardial infarction [MI]). More severe and prolonged obstruction increases the likelihood of infarction. The most recent definition of MI was

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published in 2012,[5] which defined this as myocardial cell death caused by prolonged ischemia. MI is determined by clinical features including electrocardiogram (ECG) and biomarker findings. There are 5 classifications of acute MI (Table 1), and clinicians should be aware of these. Usually, clinicians are focused on type I MI, but supply-demand mismatch or infarction in the absence of thrombotic occlusion (type 2) is also common.

RISK STRATIFICATION IN THE EMERGENCY DEPARTMENT

History

Recent systematic reviews of investigations indicate that history and physical examination are not helpful in symptomatic patients with acute chest symptoms. Swap and Nagurney[6] conducted a systematic review of chest pain characteristics from observational studies and found that certain characteristics increased or decreased the likelihood of ACS or AMI, but none are useful to disposition the patient. Fanaroff and colleagues[7] conducted a systematic review of the accuracy of the history, physical examination, ECG, and risk factors. Similarly, symptoms were not useful in isolation for risk stratification. In both studies, radiation to right arm or both arms was more specific than radiation to left arm (specificity, 96%; likelihood ratio (LR), 2.6 [95% confidence interval (CI), 1.8–3.7] vs specificity 69%; LR, 1.3 [95% CI, 1.2–1.4]). Other helpful historical factors included similar to prior ischemia or MI (specificity, 79%; LR, 2.2 [95% CI, 2.0–2.6]) and associated diaphoresis. There were mixed responses associated with nausea and vomiting. Response to nitroglycerin was also unhelpful, whereas descriptions of the pain, such as pleuritic, positional, or sharp, were only minimally helpful in decreasing the likelihood of ACS. In addition, these features, which are usually associated with lower probability of ACS, have only poor to fair interrater reliability.[8]

In recent years, pain scores have received a lot of attention; however, Edwards and colleagues[9] found that there was no difference in AMI or 30-day outcomes in patients with pain rated as severe (9 or 10 on 10-point scale) or not.

Table 1
Classification of acute myocardial infarction types

Type 1	Spontaneous MI related to ischemia caused by a primary coronary event such as plaque erosion and/or rupture, fissuring, or dissection
Type 2	MI secondary to ischemia caused by either increased oxygen demand or decreased supply such as caused by coronary artery spasm, coronary embolism, anemia, arrhythmias, hypertension, or hypotension
Type 3	Sudden unexpected cardiac death, including cardiac arrest, often with symptoms suggestive of myocardial ischemia, accompanied by new ST elevation, or new left bundle branch block, or evidence of fresh thrombus in a coronary artery by angiography and/or at autopsy, but death occurring before blood samples could be obtained, or at a time before the appearance of cardiac biomarkers in the blood
Type 4a	MI associated with percutaneous coronary intervention
Type 4b	MI associated with stent thrombosis by angiography or autopsy
Type 5	MI associated with coronary artery bypass grafting

Cardiac Risk Factors

Traditional cardiac risk factors such as hypertension, diabetes, hyperlipidemia, and tobacco use are often used to predict the long-term risk of coronary artery disease (CAD) and are included in models such as the Framingham Risk Score.[10] However, these are not useful for predicting ACS in symptomatic patients in the ED. Recent studies have found that up to 12% of patients with AMI had no cardiac risk factors.[11,12]

Prior Cardiac History

Patients with a prior normal stress test are at the same risk of 30-day adverse cardiovascular events as patients who have not previously undergone stress testing.[13,14] Stress testing does not assess whether nonobstructive plaque existing at the time of the test will subsequently rupture leading to ischemia. Thus, knowledge of a recent normal stress test may not help inform current ACS risk; patients with a prior normal stress test still had a 5% event rate at 30 days. In contrast, prior invasive coronary angiography results are useful for risk stratification of patients. Patients with no or minimal (<25%) stenosis have an excellent long-term prognosis, with 90% free from 1-vessel disease and greater than 98% free from MI nearly a decade later.[15,16] Thus, recent coronary angiography with normal or minimally diseased vessels makes the development of an ACS extremely unlikely and may be helpful during the current visit.