Emergency Department Treatment of Acute Coronary Syndromes

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KEYWORDS

- Acute coronary syndrome
- Non-ST-segment elevation myocardial infarction
- ST-segment elevation myocardial infarction
- Non-ST-segment elevation acute coronary syndrome
- Unstable angina • Chest pain • Cardiac ischemia

Acute coronary syndrome (ACS) is a broad term encompassing a spectrum of acute myocardial ischemia and injury ranging from unstable angina (UA) and non–ST-segment elevation myocardial infarction (NSTEMI) to ST-segment elevation myocardial infarction (STEMI). ACS accounts for approximately 1.2 million hospital admissions in the United States annually.[1] The aging of the United States population, along with the national obesity epidemic and the associated increase in metabolic syndrome, means that the number of individuals at risk for ACS will continue to increase for the foreseeable future.[2] This article reviews the current evidence and guidelines for the treatment of patients along the continuum of ACS.

DEFINITIONS

ACS is a syndrome defined by the presence of symptoms, electrocardiographic (ECG) changes, and/or biochemical markers consistent with myocardial ischemia or injury. Typical symptoms include chest pain or pressure, but ACS can also manifest with symptoms such as shortness of breath, nausea, or malaise. ECG changes run the gamut from ST-segment elevations to subtle ST-segment depressions or T-wave inversions. Dynamic ECG changes—those that change or evolve over time—raise particular concern for ACS.[3] In the context of ischemic symptoms, biochemical evidence of myocardial necrosis defines acute myocardial infarction (MI), and the greater the elevation in cardiac biomarkers, the higher the risk of serious morbidity and mortality.

Primary ACS refers to a syndrome of acute myocardial ischemia initiated at the level of the coronary artery itself. The most common form of primary ACS is triggered by

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rupture of an atherosclerotic plaque, leading to intraluminal thrombus formation, and either complete or partial occlusion of a coronary artery. The process of coronary thrombus formation is complex. Activation of the coagulation cascade culminates in the production of fibrin, which catalyzes the polymerization of fibrinogen into a fibrin mesh. Platelets adhere and become activated via several receptor-mediated pathways (including the thrombin receptor). Activated platelets express fibrinogen receptors, allowing the aggregation of platelets into the thrombus via fibrin cross-links. Downstream myocardial ischemia and/or injury results directly from the thrombus occluding the coronary blood flow and/or from distal embolization of microthrombi. Less common forms of primary ACS include coronary artery spasm, coronary artery dissection, and coronary artery thromboembolism.

Secondary ACS refers to pathophysiology external to the coronary arteries that precipitates signs and symptoms of coronary ischemia or injury. Any condition that limits myocardial oxygen delivery—profound anemia, hypotension, or hypoxemia, for example—can produce a clinical picture indistinguishable from primary ACS, even in patients with normal coronary anatomy. Just as "supply-side" conditions can precipitate ACS, "demand-side" conditions such as uncontrolled hypertension or tachycardia can create myocardial energy imbalance and lead to ischemia or injury. It may actually be the case that secondary ACS is actually more common than primary ACS, particularly in patients who have underlying coronary artery disease. However, unlike primary ACS, for which management is focused on the coronary thrombus, the approach to secondary ACS is on balancing the supply-demand mismatch by treating the inciting condition, whether it be sepsis, hypovolemia, hypertensive crisis, or tachyarrhythmia.

ST-segment elevation MI (STEMI) is a diagnosis made solely via ECG (**Box 1**). Based on 2007 estimates, STEMI accounts for approximately one-third of all acute MIs.[4] STEMI criteria identifies a subset of ACS patients that benefit from rapid coronary reperfusion therapy.[5] As such, any patient presenting to the emergency department (ED) with symptoms concerning for STEMI should have an ECG done within 10 minutes of arrival.[6]

However, in considering the diagnosis of STEMI, it is also important to note that there are other conditions that may cause ST elevations on an ECG (**Table 1**).

It has been well established that early reperfusion of the infarct-related artery is associated with improved outcome in STEMI.[8] Delay to reperfusion is associated with

Box 1
STEMI criteria

American College of Cardiology/American Heart Association ST-Segment Elevation Myocardial Infarction (STEMI) Diagnosis Guidelines[7]
In a patient presenting with active chest pain, a 12-lead electrocardiogram showing:

1. ST-segment elevation $\geq$1 mm (0.1 mV) in 2 or more adjacent limb leads (from aVL to III, including aVR)
2. ST-segment elevation $\geq$1 mm (0.1 mV) in precordial leads V4 through V6
3. ST-segment elevation $\geq$2 mm (0.2 mV) in precordial leads V1 through V3
4. New left bundle-branch block

Therapy should not be delayed while awaiting results or cardiac biomarkers. Reciprocal depressions (ST depressions in the leads corresponding to the opposite side of the heart) make the diagnosis of STEMI more specific.