Electrocardiographic Patterns Mimicking ST Segment Elevation Myocardial Infarction

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KEYWORDS

• Electrocardiogram • ECG • ST segment elevation • STEMI • Mimic

KEY POINTS

- The 12-lead surface electrocardiogram (ECG) is inexpensive, portable, and transmittable; it remains the cornerstone of prompt diagnosis of and primary indication for the management of ST elevation myocardial infarction (STEMI).
- Although the ECG is reasonably reliable, it remains an imperfect diagnostic tool. Some patients do present with classic symptoms and findings; however, approximately 60% to 80% of patients with ST segment elevation on the presenting ECG are ultimately found to not be associated with STEMI.
- In certain difficult cases, a patient's ECG can resemble STEMI yet manifest ST segment elevation from a non–acute coronary syndrome entity, the so-called *STEMI mimics.* In other situations, the patient's ECG makes it difficult or impossible to determine whether STEMI is present, the so-called *STEMI confounders*; these *confounders* to STEMI diagnosis are also *mimickers* of AMI.
- The ultimate goal with both the STEMI mimics and the confounders is to maximize rapid, accurate diagnosis while avoiding delays in treatment of alternative causes of ST segment elevation.

INTRODUCTION

Cardiovascular disease is the leading cause of death worldwide. In fact, 7 million patients present annually to emergency departments in the United States with symptoms concerning for myocardial ischemia.[1] Prompt reperfusion in the setting of ST segment elevation myocardial infarction (STEMI) can dramatically reduce the associated mortality and morbidity. Unfortunately, the benefit of reperfusion therapies in acute myocardial infarction (AMI) decays quickly over time. Improvements in care systems, such as the door-to-balloon initiative and the American Heart Association's Mission Lifeline have altered the landscape of STEMI management over the past decade, minimizing delays to reperfusion and significantly improving outcomes. These systems require the rapid mobilization of large teams of practitioners and resources, involve not insignificant risk to patients, and rely heavily on the clinician to quickly and accurately determine whether an electrocardiographic finding represents acute closure of a coronary artery and related STEMI. The 12-lead surface electrocardiogram (ECG) is inexpensive, portable, and transmittable; it remains the cornerstone of the prompt diagnosis of and the primary indication for management of STEMI. Although reasonably reliable, the ECG remains an imperfect diagnostic tool. Some patients do present with classic symptoms and findings; however, approximately 60% to 80% of patients with ST segment elevation on the presenting ECG ultimately are found to not be associated with STEMI.[2,3] Refer to **Fig. 1** for a depiction of the

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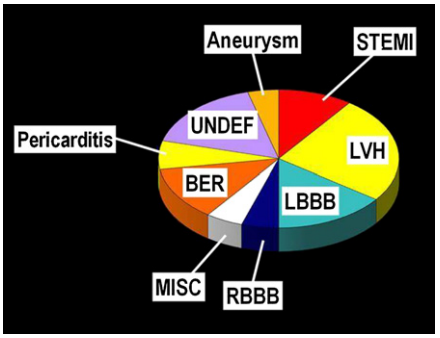

Fig. 1. Causes of ST segment elevation in adult patients with chest pain. BER, benign early repolarization; LBBB, left bundle branch block; LVH, left ventricular hypertrophy; MISC, miscellaneous; RBBB, right bundle branch block; UNDEF, undefined.

causes of ST segment elevation in adult patients with chest pain.[2,3]

In certain difficult cases, a patient's ECG can resemble STEMI, yet manifest ST segment elevation from a non–acute coronary syndrome (ACS) entity, the so-called *STEMI mimics*. In other situations, the patient's ECG makes it difficult or impossible to determine whether STEMI is present, the so-called *STEMI confounders*; these *confounders* to STEMI diagnosis are also *mimickers* of AMI.

All such cases can leave a practitioner wondering whether to initiate reperfusion therapy, either via administration of a fibrinolytic agent or activation of STEMI alert process, in essence, whether to expose patients to both the benefits and the risks of fibrinolysis or invasive coronary angiography. In some cases, the astute clinician can detect an alternative diagnosis masquerading as an STEMI. Failure to recognize these mimics can lead to inappropriate use of resources, exposure of patients to unnecessary risk, and increased rather than decreased morbidity and mortality. The ultimate goal with both the STEMI mimics and the confounders is to maximize rapid, accurate diagnosis while avoiding delays in the treatment of alternative causes of ST segment elevation. Because the risk of cerebral hemorrhage from fibrinolysis is not insignificant, careful consideration of the ECG, looking for the STEMI mimics, is required in patient-care situations in which primary percutaneous coronary intervention (PCI) is not an option. More importantly, fibrinolysis given in the setting of certain STEMI mimics, such as acute myopericarditis, is associated with high mortality.

Although each of the conditions discussed here is unique, a common issue that must not be overlooked is the interpretation of the ECG within the context of the patient's presentation; in other words, does the patient look like he or she is experiencing a STEMI? The STEMI mimics and confounders more often imitate the ECG findings of AMI than the clinical syndrome, so the patient *with* ST segment elevation but *without* a convincingly clinical picture of STEMI should prompt the provider to suspect a non-AMI presentation. At times, these diagnoses are very challenging, which will understandably impact the rapid application of reperfusion therapy and likely increase the door-to-therapy time.

STEMI MIMICKING PATTERNS

Myocarditis and Myopericarditis

Inflammation of the pericardium and heart muscle is a common cause of chest pain with ST segment elevation. Seventy-three percent of patients diagnosed with acute myopericarditis will have ST segment elevation on initial ECG.[4] Additionally, 44% of patients with chest pain and positive troponin but who do not have obstructive coronary disease by angiography demonstrate evidence of myocarditis by cardiac magnetic resonance imaging (MRI) using late gadolinium enhancement to reveal areas of myocardial necrosis in a noncoronary distribution (ie, midwall or subepicardial rather than subendocardial).[5]

Myocarditis affects patients of all ages and has a wide spectrum of clinical severity, ranging from incidental chest discomfort to fulminant heart failure with cardiogenic shock. The term *myocarditis* refers to an inflammatory process of the heart muscle (as reflected by the presence of biomarkers and ECG changes), whereas *pericarditis* refers to isolated inflammation of the lining around the heart. The pericardium is electrically silent, thus, when patients present with a clinical picture suggestive of pericarditis and demonstrate ST segment changes, the myocardium is also affected. Frequently, inflammation involves both components, hence, the term *myopericarditis* is used.

The cause is not frequently elucidated, but a viral cause is thought to be most common, with a minority of cases stemming from toxins or autoimmune processes. Patients may remember a prodrome of viral illness in the previous week or two. The pain is classically pleuritic in nature and changes in severity with position (sitting forward or lying back).

Biomarkers of myocardial necrosis, such as troponin and creatinine kinase, are positive in the setting of myocarditis, although they may be negative in the setting of pure pericarditis. The absolute value of troponin elevation is associated with the extent of myocardial cell injury but only very roughly correlates with clinical severity. A rub on cardiac auscultation is a highly specific but very