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The late presenting STEMI: How ECG scores can be used to estimate event time

Zak Loring, MD,[a,*] Jenny K. Cohen, MD,[a] Juliet Morgan, MD,[a] Joseph Yang, MD,[b] Jeffrey Zimmet, MD, PhD[b,c]

[a] *Department of Medicine, University of California San Francisco, San Francisco, CA*
[b] *Division of Cardiology, Department of Medicine, University of California San Francisco, San Francisco, CA*
[c] *Division of Cardiology, Department of Medicine, San Francisco Veterans Affairs Medical Center, San Francisco, CA*

Abstract

Introduction: Patients presenting with ST elevation myocardial infarction (STEMI) derive significant benefit from timely percutaneous coronary intervention (PCI). Electrocardiogram (ECG) scoring systems can complement history to estimate the infarction age and inform clinical decision-making.
Case: A 76 year old man presented with eight days of dyspnea on exertion and chest pain. Triage ECG showed ST elevation and Q waves in the anterior leads. The patient was taken for coronary angiography and found to have a 100% occlusion of the left anterior descending artery.
Discussion: Determining timing of acute coronary syndromes is challenging. ECG indices aid clinical history by quantifying infarction acuity.

Keywords: STEMI; ECG scores; Clinical decision-making

Introduction

Timely reperfusion by primary percutaneous coronary intervention (PCI) can reduce the risk of mortality and complications in patients presenting with ST elevation myocardial infarction (STEMI) [1]. However, patients who present to care late into their infarction may have less salvageable myocardium and thus derive less benefit from PCI [2]. Determining whether PCI should be attempted in patients diagnosed with STEMI long after initial symptom onset is currently determined by the clinical judgment. Cases in which symptoms are ambiguous or atypical, or are stuttering in nature, present a particular dilemma. We suggest that electrocardiogram (ECG) scoring systems may help inform that decision.

ECG scoring systems exist which provide an estimate of the acuity of the infarction [3], size of the ischemic myocardium at risk [4], and the amount of myocardium that has already infarcted [5]. These scores can complement patient history and help determine how far a STEMI has progressed. We present a case of a 76 year-old man presenting with several days of symptoms found to have a STEMI on his triage ECG and discuss how ECG scores assessed the acuity of his presentation.

Case description

A 76 year-old male veteran with a history of diabetes mellitus, hypertension, and a 40-pack year smoking history, presented to the emergency department with eight days of shortness of breath. A week prior to presentation, he reported experiencing shortness of breath limiting his ability to walk, requiring frequent stops to catch his breath. He also noted new orthopnea and paroxysmal nocturnal dyspnea. One day prior to presentation, he began experiencing left-sided chest pain, which he thought was secondary to sleeping on a fold-out couch. On the morning of admission, he was dyspneic at rest prompting him to seek care.

On admission, he was afebrile, normotensive, tachycardic with a heart rate in the 130s, and with an oxygen saturation of 93% on 2 L nasal cannula. The patient's physical exam was notable for an elevated jugular venous pressure to approximately 15 cm H_2O, bilateral rales at the lung bases, and absence of lower extremity edema. Triage ECG showed sinus tachycardia at 114 bpm with ST-segment elevation in V2–V3, Q waves in V1–V3, and T-wave inversion in aVL (Fig. 1). Initial troponin was 1.05 μg/L and down trended to 1 μg/L on repeat assessment 6 h later. Chest radiograph revealed a small pleural effusion, pulmonary congestion, and bilateral vascular cephalization. The patient was given 325 mg of aspirin and sublingual nitroglycerin, and started on a heparin drip after receiving a bolus. He reported an improvement in his chest discomfort.

* Corresponding author at: University of California San Francisco, 505 Parnassus Ave, San Francisco, CA 94143.
E-mail address: zak.loring@ucsf.edu

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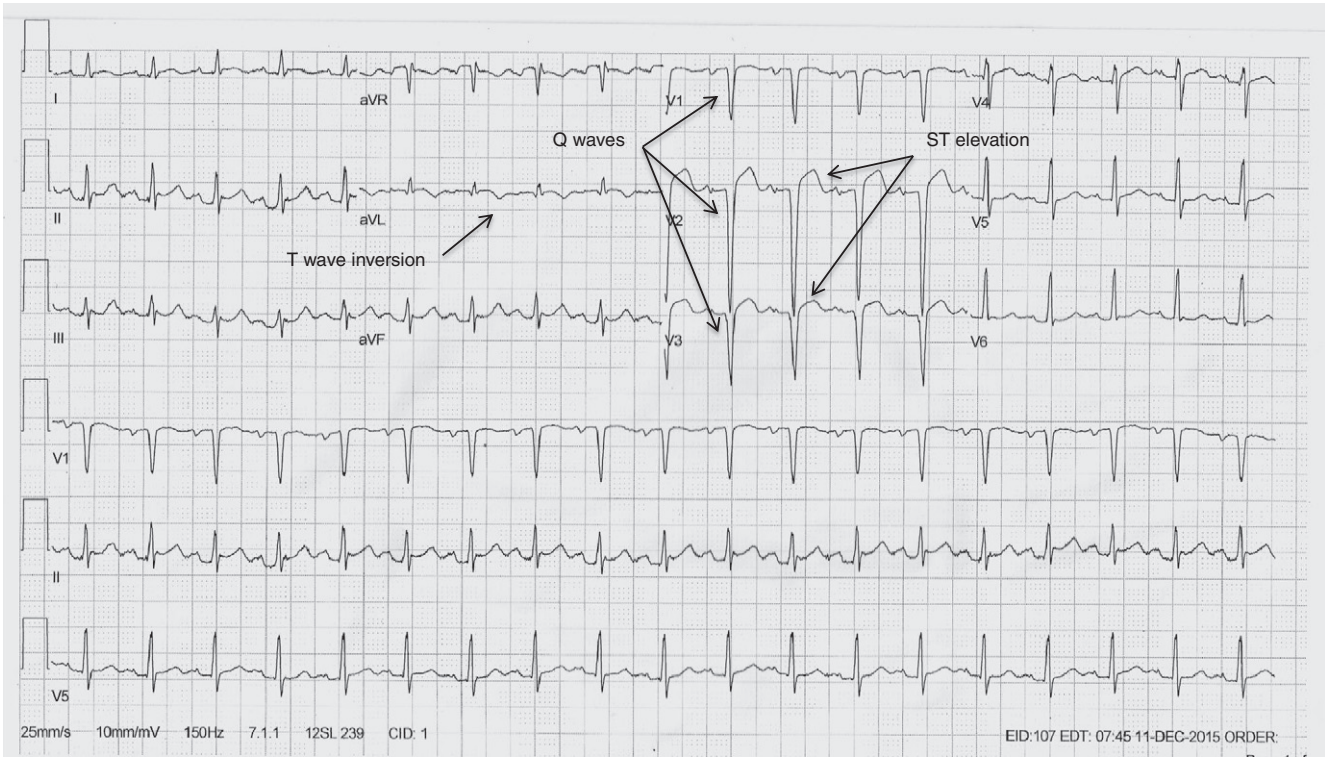

Fig. 1. Presentation ECG: ECG at time of presentation demonstrating ST elevations in V2–V3, Q waves in V1–V3, and T-wave inversions in aVL.

Cardiology was consulted emergently for evaluation of potential STEMI. After reviewing the presenting and repeat ECGs, eliciting a focused history and physical exam the case was reviewed with the interventional cardiologist who expressed concern regarding the delay between symptom onset and presentation as well as the presence of deep Q waves on the triage ECG. The patient was admitted to the coronary care unit for continued monitoring, blood pressure control on a nitroglycerin infusion, anticoagulation with a heparin infusion, and diuresis. At that time, he was unable to lie flat without significant respiratory distress due to pulmonary edema.

He was stabilized medically and taken for coronary angiography on the evening of admission. Cardiac catheterization showed 100% occlusion of mid left anterior descending (LAD) of uncertain chronicity, 70% stenosis of the left circumflex, and 50% stenosis of right coronary artery (RCA, Fig. 2). Despite attempts with wire escalation, the LAD occlusion could not be easily crossed. The nitroglycerin infusion was continued, heparin was stopped, and cardiothoracic surgery was consulted for consideration for coronary artery bypass grafting. Follow-up transthoracic echocardiogram showed an ejection fraction of 35%–40% with anterior wall hypokinesis and apical akinesis. Delayed-enhancement magnetic resonance imaging (MRI) showed transmural delayed enhancement consistent with nonviable scar in the mid anterior, mid anterolateral and apical anterior left ventricle consistent with an LAD infarction (Fig. 3). There was subendocardial enhancement (<50%) in the mid anteroseptal and apical septal walls. There was no MRI evidence of inferior wall infarction.

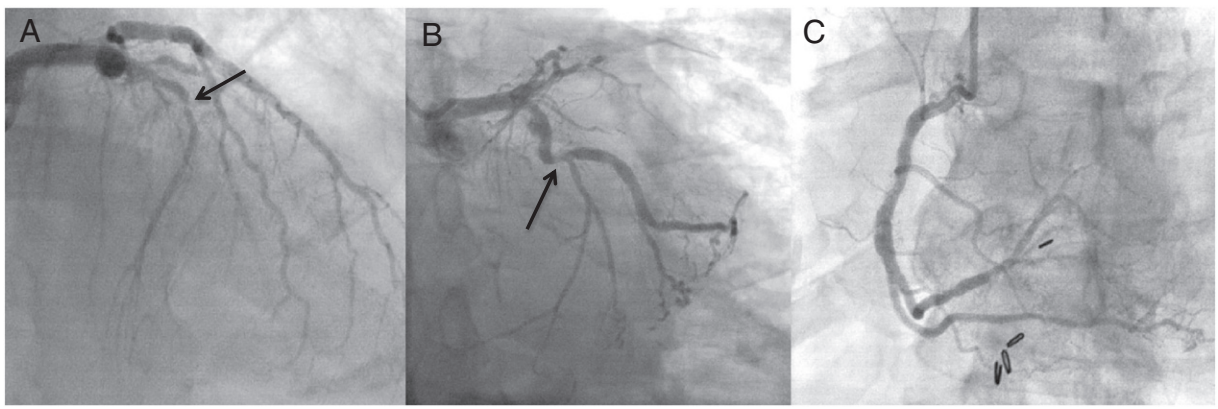

Fig. 2. Coronary angiogram: A: Right anterior oblique (RAO)/cranial view showing 100% occlusion of mid LAD after the origin of the first septal perforator (arrow); B: Anterior–posterior (AP)/caudal view showing 70% stenosis of left circumflex (arrow); C: LAO/cranial view showing serial 40%–50% stenosis of right coronary artery.