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MYOCARDITIS WITH ST ELEVATION AND ELEVATED CARDIAC ENZYMES MISDIAGNOSED AS AN ST-ELEVATION MYOCARDIAL INFARCTION

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□ **Abstract—Background: Acute myocarditis can mimic ST-elevation myocardial infarction (STEMI). Quickly determining the correct diagnosis is critical given the "time is muscle" implication with a STEMI and the potential adverse effects associated with use of fibrinolytic therapy. Case Report: A 46-year-old man presented to a rural emergency department with chest pain, and an electrocardiogram (ECG) read as showing 0.1 mV of ST-segment elevation in leads III and aVF. His initial cardiac troponin T was 0.44 ng/mL. He received fibrinolytic therapy for presumed STEMI. Cardiac magnetic resonance imaging was later performed and showed epicardial delayed enhancement consistent with myocarditis. Upon further questioning, he acknowledged 3 days of stuttering chest discomfort and a recent upper respiratory infection, as well as similar chest pain in his wife. Conclusions: A systematic evaluation is essential for acute chest pain, including a focused history, identification of cardiac risk factors, and ECG interpretation. A history of recent viral illness, absence of cardiac risk factors, or ECG findings inconsistent with a single anatomic lesion would suggest a potential alternate diagnosis to STEMI. This case emphasizes the importance of a focused history in the initial evaluation of chest pain.** © **2012 Elsevier Inc.**

□ **Keywords—Chest pain; Myocarditis; Myocardial infarction; Electrocardiogram pattern; Electrocardiography; ST-segment elevation**

INTRODUCTION

Acute chest pain is the chief complaint for many clinical syndromes. A focused evaluation is important to differentiate an acute coronary syndrome (ACS) from other diagnoses. We report the case of a patient who presented with chest pain that was interpreted as an acute, inferior ST-segment elevation myocardial infarction (STEMI). The patient received fibrinolytic therapy before the diagnosis of myocarditis was made.

CASE REPORT

A 46-year-old man presented to a rural medical center with 10-out-of-10 substernal, heavy chest discomfort radiating to his left arm. His lone cardiac risk factor was a 60 pack-year smoking history. The initial electrocardiogram (ECG) (Figure 1) was read as showing 0.1 mV of ST-segment elevation in leads III and aVF. Aspirin, clopidogrel, and sublingual nitroglycerin were administered. Tenecteplase was administered 27 min after presentation as adverse weather precluded rapid transfer for primary percutaneous coronary intervention. The patient's chest discomfort resolved and a repeat ECG seemed to show resolution of the ST elevation. Serial cardiac troponin T values were 0.44, 0.48, and 0.49 ng/mL, respectively.

Coronary angiography performed 4 h after presentation showed moderate diffuse atherosclerosis, including a 50% mid-right coronary artery lesion. No intervention was done, and cardiac magnetic resonance imaging (MRI) was performed to further investigate the etiology of his chest pain and elevated troponin. The cardiac MRI revealed patchy areas of epicardial delayed

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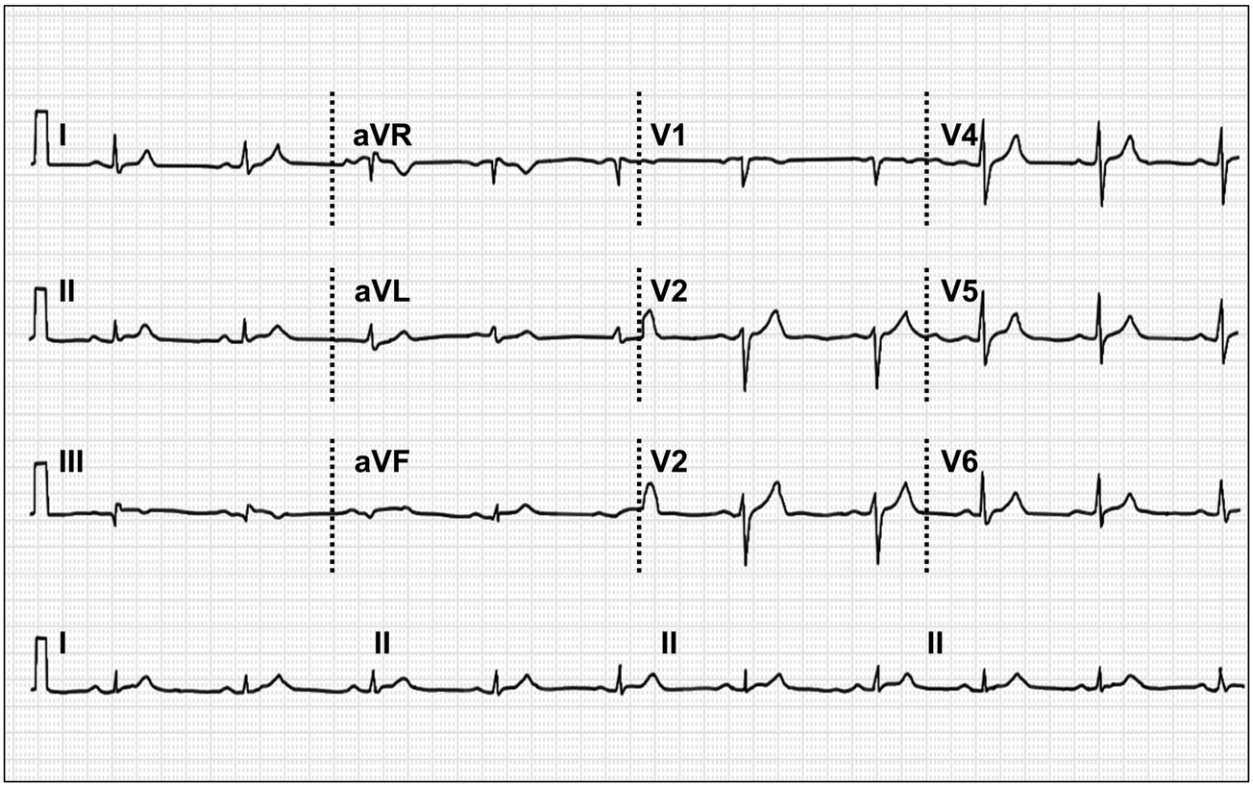

Figure 1. Initial electrocardiogram interpreted as 1 mm of ST elevation in leads III and aVF.

enhancement (Figure 2) consistent with myocarditis. There was no evidence of endocardial or transmural enhancement to suggest myocardial infarction.

Upon further questioning, the patient acknowledged stuttering chest discomfort for 3 days, a recent upper respiratory tract infection, and he noted that his wife had similar chest discomfort. He was diagnosed with myocarditis and moderate coronary artery atherosclerosis. He was discharged with plans for surveillance of his myocarditis and treatments to reduce his risk of a cardiac event.

DISCUSSION

This case illustrates several key features of the clinical presentation of acute myocarditis and its ability to mimic acute myocardial infarction. The clinical presentation of acute, viral myocarditis varies and may include a viral prodrome (fever, myalgias, respiratory or gastrointestinal symptoms), chest discomfort, heart failure, dysrhythmia, or sudden cardiac death (1). Both myocarditis and ACS can present with the constellation of chest discomfort, ECG changes, and elevated cardiac enzymes, making it challenging at times to differentiate the two. Important clinical hints to the presence of myocarditis can be either absent or missed.

Elevated cardiac troponin T levels can be seen in the absence of an ACS. Non-coronary causes include pulmonary embolism, acute heart failure, septic shock, cardiotoxic medications, renal failure, and recent cardiac procedures (2). Perimyocarditis, however, is one of the only situations other than acute myocardial infarction in which very high levels of cardiac troponin T have been observed (3). Most patients with biopsy-proven myocarditis have an elevated cardiac troponin T, whereas elevations can be modest or absent in over half of patients with clinically suspected myocarditis (3). The primary differential in a patient presenting with acute chest discomfort and a significantly elevated cardiac troponin T includes ACS, perimyocarditis, aortic dissection, and pulmonary embolism. Marked elevations are more common with ACS and myocarditis.

The most common ECG abnormalities in patients with myocarditis include ST-segment elevation (55%), T-wave inversion (27%), ST-segment depression (18%), and pathologic Q waves (18%) (4). The distribution of ECG abnormalities can be helpful as often the findings are diffuse rather than focal. Our patient had minimal ST-segment elevation on his initial ECG that seemed to resolve on the subsequent ECG after administration of nitroglycerin and tenecteplase. Differences in his second ECG may be attributable to resolution of viral-induced coronary vasospasm with nitroglycerin or lead placement differences. Although ST-segment changes evolve with myocarditis, this evolution usually does not occur over

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