

Delayed Cath-Lab Activation for STEMI Due to Erroneous Computer Electrocardiogram Interpretation: A Note of Caution for Emergency Physicians

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A 67-year-old gentleman experienced retrosternal burning pain radiating to both elbows associated with shortness of breath and sweating while he was hunting. The pain was initially intermittent but later became constant that prompted him to come to the emergency room (ER) for evaluation. He had no prior history of chest pain or cardiac disease. He has a medical history of gastro-esophageal reflux disease with different symptoms from the current presentation. He never smoked, does not drink alcohol or use illicit drugs and has no family history of premature coronary artery disease. His physical examination in the ER was unremarkable and he was vitally stable. His electrocardiogram (ECG) was read by the computer as sinus rhythm, possible left atrial enlargement and ST-segment depression (STD), consider subendocardial injury (Figure 1). There was a 1 mm ST-segment elevation (STE) in leads I, aVL with reciprocal STD in leads II, III, aVF suggestive of high lateral ST-segment elevation myocardial infarction (STEMI). This was not recorded by the computer ECG which has delayed cath-lab activation 45 minutes from onset of initial ECG. After evaluating the ECG by an astute emergency physician, the cardiologist was contacted for a working diagnosis of high lateral STEMI and the cath-lab was activated. Coronary angiography revealed total occlusion of the 1st diagonal branch and 99% occlusion of mid-LAD which prompted drug eluting stent deployment in both vessels followed by TIMI III flow. Peak troponin I value was 50 mg/mL (normal 0.02-0.03 mg/mL). Repeat ECG 12 hours later showed the development of pathological Q waves in leads I and aVL. Myocardial perfusion scan showed scar in the affected territory. He had no recurrence of chest

pain, normal left ventricular function and was discharged home on anti-ischemic medications (Figure 2).

Cardiovascular disease remains the number-one cause of mortality in the United States [1] with approximately 500000 STEMI diagnosed per year [2]. Every minute of delay from symptom onset to revascularization is accompanied by an increased mortality [3-5] and hence the American College of Cardiology/American Heart Association had recommended a door-to-balloon time (DBT) < 90 minutes when percutaneous coronary intervention is the mode of revascularization. As part of strategy of reducing the DBT, prehospital ECG potentially reduces it by up to 30 minutes [6-8] which leads to a 7.4% decrease in 1-year mortality [7,8]. Computer ECG interpretation of STEMI has been a major source for STEMI alerts and subsequent cath-lab activation. One concern has been that the computer occasionally over-reads STEMI leading to false positive STEMI alarms. Only up to 10% prehospital false activation rate is generally acceptable [9] and this modest rate of false positive misdiagnoses is expected to maximize the diagnostic sensitivity [10], but frequent false alarms will lead to unnecessary cath-lab activation and subsequently increase healthcare costs. Conversely, a more important concern is a false negative STEMI interpretation by computer ECG as it may harm the patient. In the study by Bhalla and colleagues [11], 42/100 STEMI were not identified by computer ECG which had a sensitivity of 58% and Massel and colleagues found the Marquette 12SL system to have a sensitivity of 61.5% when evaluating patients with STE for eligibility for thrombolytic therapy [12]. On the other hand, Ducas and colleagues assessed the accuracy of prehospital ECG interpretation by nonphysician emergency medical services, and found that this method had an excellent sensitivity of 99.6% in detecting STEMI [13].

Despite the significant improvement in technology of the 12-lead ECG equipment, the sensitivity of the ECG in detecting STEMI is still not optimal. It was also found that ECGs read as normal by the computer did not provoke further checking. An interesting study showed that false negative clinical STEMI diagnosis by physicians is related to several factors including the height of STE (odds of accurate diagnosis were 14% greater per millimeter of maximal STE), the

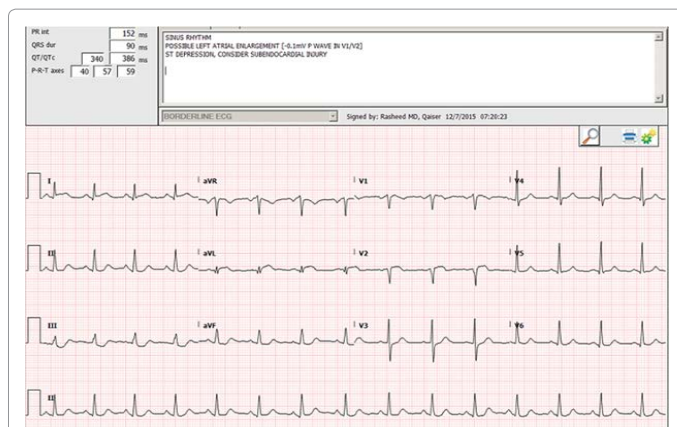


Figure 1: 12 lead ECG showing approximately 1mm ST-segment elevation in leads I and aVL with reciprocal ST-segment depression in the inferior leads due to high lateral STEMI. Notice the subtle ST-segment elevation in V1 and V2. Magnified leads revealed ST-segment elevation of 1.2 mm in leads I and aVL thereby meeting criteria for STEMI and ST-segment elevation in leads V1 and V2 0.6 and 1.4 mm, respectively, thereby not meeting criteria for STEMI according to defining guidelines.

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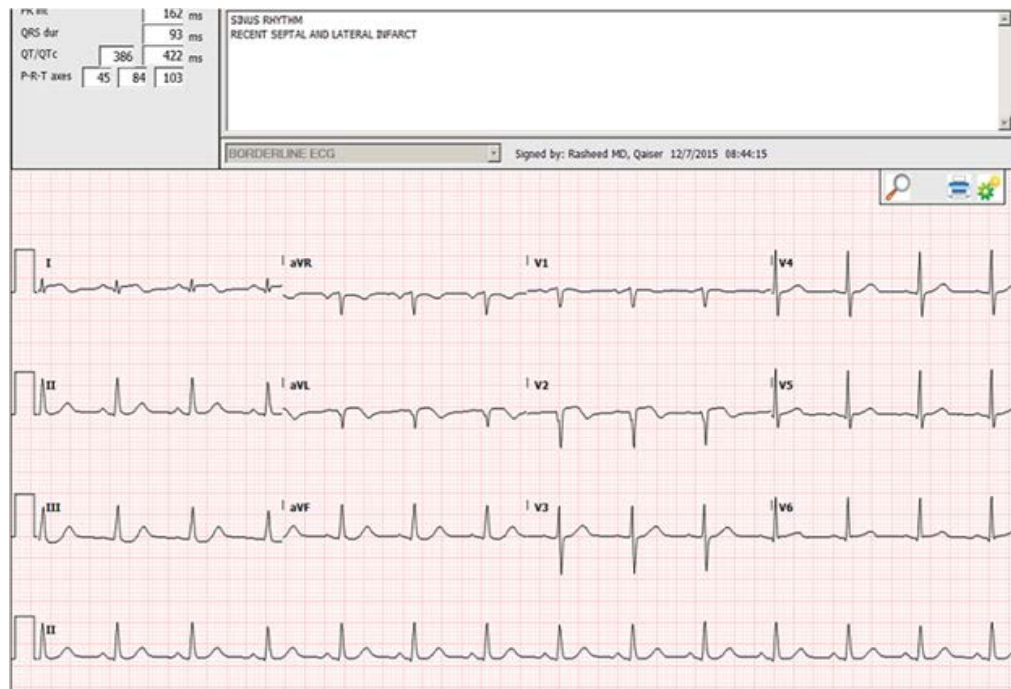


Figure 2: Follow up ECG 12 hours later showing pathological Q waves in leads I and aVL.

number of leads with STE (odds of accurate diagnosis 42% greater per lead with diagnostic STE) and the territory of STE (easier identified if in inferior leads) [10]. In the presented case, the modest STE in leads I and aVL of 1 mm and the unusual location of high lateral STEMI may be a cause for delayed diagnosis. Although there was STE in leads V1 and V2, it did not meet criteria for STEMI on admission ECG according to defining criteria. In conclusion, we aim to emphasize that the sensitivity of computer ECG in diagnosing STEMI is not optimal and nothing surpasses the personal interpretation of the ECG in patients presenting with chest pain. This is especially important for those working in the pre-hospital environment as well as emergency physicians. Paramedics should obtain appropriate training in the pre-hospital identification of STEMI independently and not to depend solely on computer interpretation.

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