

“And, when you want something, all the universe conspires in helping you achieve it” Paulo Coelho, *The Alchemist*

Case report

Getaw worku*

Department of Emergency Medicine, NYMC, Valhalla, NY

Received: Jan 31, 2020; **Accepted:** Feb 28, 2020; **Published:** Mar 04, 2020

***Corresponding author:** Getaw worku, Department of Emergency Medicine, NYMC, Valhalla, NY

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Abstract

Electrocardiogram (ECG) is instrumental in identifying acutemyocardial infarction (AMI). ST segment elevation in a region of specificcoronary vessels in the setting of acute coronary syndrome (ACS) is the hallmark of AMI. Reciprocal ST depression and T wave inversion (TWI) are well-recognized changes that could accompany ST segment elevations. Reciprocal changes may represent the only early manifestation of AMI and may be present in specific leads such as lead aVL as early and sensitive markers of AMI. Abnormalities in ST segment and T wave can result from non ACS causes, but early recognition and appropriateintervention is of paramount importance for those patient with ACS. Wereport a case of a 55 year old male with acute IWMI with early reciprocaland dynamic ECG changes with literature review.

Key words: T wave inversion, MI and Reciprocal change

Introduction

Electrocardiogram (ECG) is instrumental in identifying acute myocardial infarction (AMI). ST segment elevation in a region of specific coronary vessels in the setting of Acute Coronary Syndrome (ACS) is the hallmark of AMI [1-3]. Reciprocal ST depression and T wave inversion (TWI) are well-recognized changes that could accompany ST segment elevations [4-7]. Reciprocal changes may represent the only early manifestation of AMI and maybe present in specific leads such as lead aVL as early and sensitive markers of AMI [8]. Abnormalities in ST segment and T wave can result from non ACS causes, but early recognition and appropriate intervention is of paramount importance for those patient with ACS. We report a case of a 55 year old male with acute inferior wall myocardial infarction (IWMI) with early reciprocal and dynamic ECG changes with literature review.

Case report

This is the case of a 55-year-old male with history of hyperlipidemia, hypertension, and coronary artery disease (CAD) with stents who presented with chest pain. He first noticedchest pain 5 days before presentation. His symptomthen lasted for 30 minutes and improved with sublingual nitroglycerin. He was seen, treated, observed and discharged to follow up with his doctor at another facility. On the day of his presentation to our facility, his pain wasdescribed as sharp, left-sided, radiates to hisbackand 6/10 in severity. The pain later progressed to 10/10 pain with diaphoresis. His symptom started while walking, but the patient denied shortness of breath, vomiting, cough or fever. At presentation his vital signs included a temperature of 98.3 degrees, blood pressure of 147/86 mmHg, heart rate of 70 beats per minute, respiratory rate of 16 breaths per minute and oxygen saturation of 99% on room air. The patient was a former smoker and takes Crestor 40mg, Atenolol 25mg and ASA 81mg daily. He has extensive

family history of CAD with myocardial infarction in both parents and he had myocardial infarction 4 years ago that led to placement of 3 stents. The remainder of review of the systems was unremarkable. His initial electrocardiogram showed isolated TWI in lead aVL (Figure 1A) which was new compared to his old ECG (Figure 1B). His physical

to PCI-related delay from first medical consult to PCI and with consultation with the Cardiology team, tPA was started as per protocol. Adjuvant treatment for STEMI was also initiated. Repeat vital signs were BP 159/83, HR 81, RR 20, O₂ 99%. ECG after tPA showed resolution of both TWI in lead aVL and ST elevations in inferior leads and his symptoms improved. Patient was then transferred out for PCI. Cardiac catheterization revealed total occlusion of the proximal RCA. The patient underwent successful intervention and did well after the procedure.

Figure 1: Dynamic ECG changes.

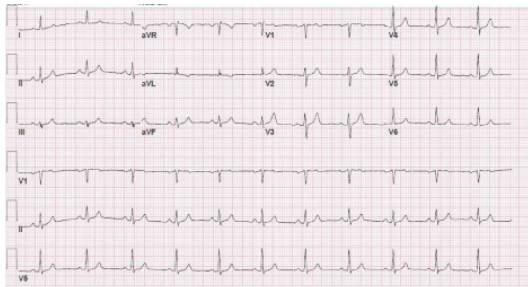


Figure 1A: Normal T wave in lead aVL (old ECG, black arrow)

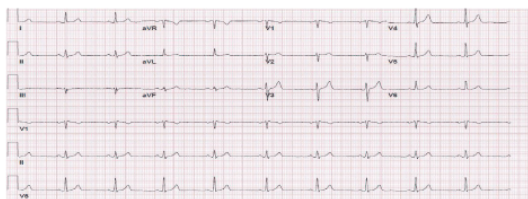


Figure 1B: Isolated T wave inversion in lead aVL (green arrow)

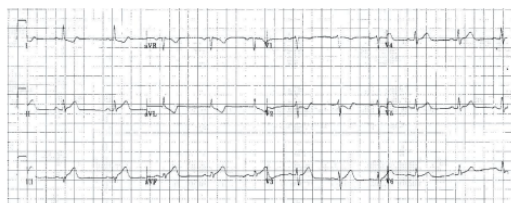


Figure 1C: ST elevation in inferior leads (30min later after presentation, red arrows)

examination revealed moderate distress from pain and diaphoresis. He had no murmurs on cardiac exam and his lungs were clear to auscultation. The rest of the physical examination was unremarkable. The patient was started on 325mg Aspirin, 0.4mg sublingual nitroglycerin and oxygen. He was placed on continuous cardiac and pulse oximetry monitor. During his stay, his pain was not resolved by oral Nitroglycerin or Morphine 4mg IVP. Nitroglycerin drip with 10mcg/min was initiated to control his symptoms. Repeat ECG showed Hyperacute T waves in inferior leads with reciprocal ST depression in leads I and aVL followed by ST segment elevation in inferior leads (Figure 1C). The right-sided ECG did not show right ventricular involvement. Due

Discussion

ST and T wave changes have gained popularity and much attention has been given to these changes in recent years. ST depression in lead aVL has been postulated to predict poor outcome after AMI. Its role in other conditions such as ACS has not been well studied. T-wave changes may signify benign or pathologic conditions [4]. T wave inversion and ST segment depressions may simply represent reciprocal changes or ischemic changes in the presence of CAD. Reciprocal changes may represent remote ischemia secondary to vasospasm in non-infarct coronary vessel especially in patients with multi-vessel CAD or are due to depolarization changes in area of myocardial infarct opposite the reciprocal lead [9-15]. ST depression and T wave inversions are the result of myocardial ischemia except in leads V1 and V2, which may represent posterior myocardial infarction. ST depression may represent reciprocal change for AMI in areas opposite the site of the myocardial injury due to electric field effect or are considered to be due to a second area of ischemia [11]. Many factors such as the size, the location of the infarct as well as the presence of LAD lesion influence reciprocal changes. Lead aVL positioned over the superior and / or high lateral aspect of the inferior wall making it truly reciprocal to the inferior wall. It is also less influenced by the infarct size.

T wave inversion in lead aVL has also been associated with left anterior descending (LAD) artery lesion [16,17]. Changes in this specific ECG lead may represent significant coronary artery disease (CAD) in LAD segment or it represents early reciprocal change for acute inferior wall MI (IWMI) in the appropriate clinical presentation.

In addition, TWI in lead aVL may represent mid LAD lesion and in the appropriate setting ischemia in the LAD distribution. Farhan et al. and our group showed that had T-wave inversions in lead aVL signify mild LAD lesion.

T-wave changes in lead aVL have not been emphasized as significant and are not well recognized across all specialties. As reported by Farhan the corresponding ECG changes were read as normal by the referring physicians. This patient's LAD was not patent.

Moreover, 25-53% of patients with IWMI have right ventricular involvement. These cases carry higher morbidity and mortality than IWMI that has favorable outcome in general. The value of ST depression in lead aVL in identifying involvement of the right ventricle has been studied. Turhan et al. have shown that the presence of more than 1mm ST-depression in lead aVL has a sensitivity of 87% and a positive predictive value (PPV) of 90% in diagnosing right ventricular involvement in acute IWMI [18]. We did perform right-sided ECG, but we did not detect right ventricular involvement.

Shah et al. have shown that these reciprocal changes have prognostic significance [19,20]. Their data indicate that patients with inferior wall infarction who have associated precordial S-T segment depression have greater global and regional left ventricular dysfunction presumably due to associated ischemia or infarction in areas remote from the inferior wall and they have relatively high in-hospital mortality and morbidity rates. Early non-invasive detection of this high risk subset may permit the testing of aggressive modes of therapy designed to limit the extent of myocardial ischemic damage with resultant decrease in mortality and morbidity. Akhras et al. also showed that patients after MI who underwent stress testing and those who have reciprocal ST segment depression occurring at the time of acute myocardial infarction may identify patients with severe coronary disease [21]. It appeared that the presence of this reciprocal change as reliable as an early postinfarction exercise test in predicting the underlying coronary anatomy. In the absence of reciprocal ST change at the time of acute myocardial infarction, however, it remains important to perform early exercise testing to exclude additional coronary artery disease. Their conclusion was reciprocal ST depression should be considered indicative of severe multivessel coronary arterial disease and a risk factor for subsequent cardiac events. Specialist referral for angiography is advised. When there is no reciprocal change early exercise testing provides valuable additional information concerning underlying coronary arterial disease and should be performed routinely.

Given our past studies with the subject and emphasis

in staff education the TWI in lead aVL was recognized and serial ECG performed which led to the identification of the dynamic changes. The patient was appropriately treated early on and transferred for higher level of care.

Conclusion

There is accumulating evidence that changes in lead aVL can have significance for patients. They may represent early inferior wall MI or indicate significant LAD lesion. The presence and dynamic changes of this specific ECG finding should not be considered non-specific and the diagnosis should not be missed as it can lead to significant morbidity and mortality. Physician training should emphasize changes in lead aVL and their significance.

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