Benign Late T Wave Inversion Post CABG: Case Report of Myocardial Reperfusion Sign Appearing Late on ECG

Abstract:

The onset of new T wave inversion (TWI) on an Electrocardiogram (ECG) has always been considered an alarming sign of myocardial ischemia. Successful myocardial infarction reperfusion is indicated by the presence of early TWI after revascularization. We report a case of benign late precordial TWI post-coronary artery bypass grafting (CABG) with no evidence of myocardial ischemia.

Introduction:

The appearance of precordial TWI on ECG may represent anterior wall ischemia. However, it is not pathognomonic and may be due to several other nonischemic entities, one of which is reperfusion and restoration of blood flow through an occluded artery. Early TWI after revascularization has been extensively reported following post-fibrinolysis and primary percutaneous coronary intervention (PCI) for St segment elevation MI (STEMI) [2,3,4,5].

Herein we report a case of non-ST-segment elevation MI(NSTEMI) with triple vessel coronary artery disease (CAD) and preserved left ventricular function (LVF), who underwent successful revascularization via CABG, with no evidence of myocardial stunning post-reperfusion to present two months later with new nonischemic anterior wall TWI.

Case report:

A 44-year-old middle eastern male from Pakistan, medically free apart from dyslipidemia on a trial of diet presented with non-ST-segment elevation MI (NSTEMI). The patient's physical examination was unremarkable, laboratory work-up revealed the following parameters; Troponin I peeked to 8012 pg/mL, Creatine kinase (CK) 376 U/L, and additional investigations included normal Chest x-ray and Echocardiogram.

Coronary catheterization revealed triple vessel disease involving ostial to mid LAD 80% lesion, ostial 80% D1 lesion, ostial 80% OM1 lesion, and diffusely disease RCA up to the bifurcation of PDA/PLV branches, followed by CABG two weeks later-LIMA to LAD, SVG to D1, OM1 and PDA.

ECG on presentation was normal, pre and immediately post-CABG (figure 1A-B). Two months later, the patient represented with short-lived non-specific chest pain with new TWI in v1-v3, progressed to v4 on the second day (figure 2A-B) then persisted. No troponin leak, normal LV function with no regional wall motion abnormalities (RWMA) on Echocardiogram.

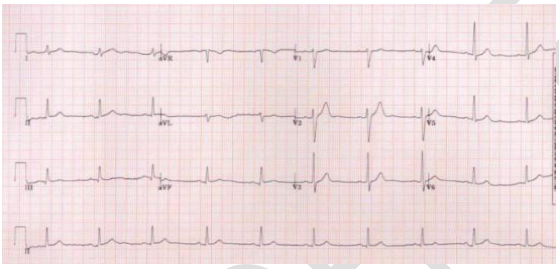


Figure 1A: ECG NSTEMI first presentation before CABG, normal sinus rhythm-normal ECG.

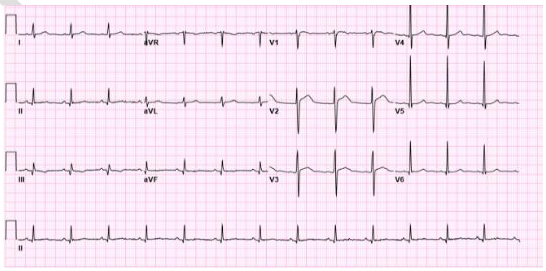


Figure 1B: ECG 6 days post-CABG NSR with non- specific T wave changes in lead III/AVF.

Furthermore, coronary CT angiogram (figure 3 A-B) was normal with patent grafts, eventually re assured and discharged home on Metoprolol 50 mg od, Aspirin, and high dose statin.

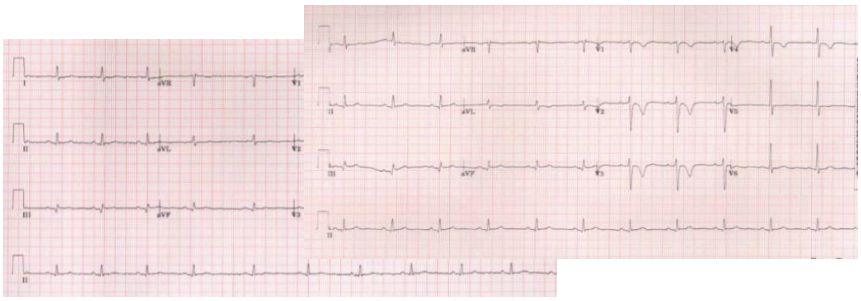
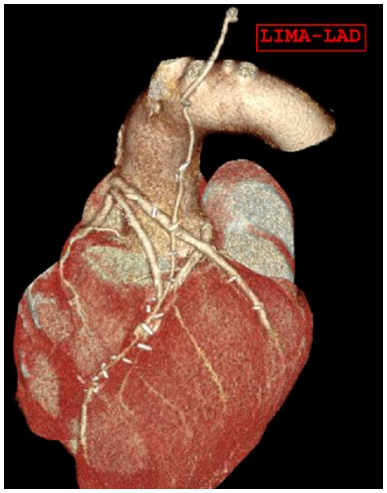


Figure 2B: ECG two months later after CABG, day 2 NSR, with deep TWI in V1 up to V4, and AVL. Flat TW in V5 and later in lead V6.

Figure 2A: ECG two months later after CABG reperfusion in LAD and first day NSR TWI diagonal area.



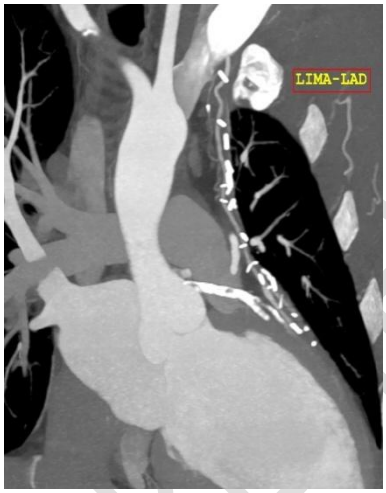


Figure 3A-B: (Figure 3A) is on the left and (Figure 3B) is on the right. Both figures are showing normal coronary CT angiogram with patent LIMA to LAD graft.

Discussion:

T wave inversion on Electrocardiogram represents altered ventricular repolarization and was first described by Jacobson and Schrire [(1)]. The differential diagnosis of precordial TWI is vast, ranging from life-threatening to entirely benign conditions including acute coronary syndrome (ACS), pulmonary embolism, central nervous system injury, to benign persistent juvenile TWI.

Early TWI has been considered a robust indicator of successful reperfusion, better prognosis, and improved LV function [(2,3,4,5)]. Reperfusion ECG changes develop within 4 hours, but no later than 24 hours in STEMI cases, including the normalization of ST-segment, TWI, idioventricular rhythm, and the deeper the negativity of the T waves the greater the effectiveness of myocardial reperfusion [(6)]. The presentation of our case was NSTEMI followed by CABG. Therefore, we investigated ECG changes related to CABG.

A conducted study regarding CABG-induced ECG changes suggested a marginal increase in TW alternans and improvement in QRS-T angle, while other ECG parameters like QRS amplitude, TW amplitude, and ST-segment deviation were not significantly influenced in the early postoperative period [(7)]. Newly developed prominent T waves with QT prolongation in the ECG post-CABG may indicate reperfusion injury and myocardial stunning [(8)].

The apparent presentation of our case does not post CABG related ECG changes, or

reperfusion injury and myocardial stunning, he had short-lived atypical chest pain, with no troponin leak, normal LV function with no regional wall motion abnormalities on Echocardiogram and normal CT coronary angiogram indicating patent grafts.

The presence of early TWI in anterior leads with STEMI is associated with patency of the left anterior descending artery (LAD), identical to the distribution of TWI in our case (TWI inV1- V4), the major graft is LIMA to LAD plus three other grafts to diagonal, OM, and PDA.

This relation was not found in other infarct-related arteries, probably because LAD feeds the vast majority of LV mass, the sensitivity of TWI to predict spontaneous reperfusion in anterior STEMI was 30.5% and specificity was 94.2% according to Ernesto Alexis. et al [(9)].

Another theory that may also be relevant in our case is cardiac memory. Cardiac memory (CM) refers to persistent T wave changes, after abnormal ventricular activation, such as left bundle branch block, ventricular tachycardia, ventricular pacing, and ventricular preexcitation. Initially described by Chatterjee, et al, and was named as "cardiac memory" by Rosenbaum, et al [(10)(11)]. The combination of positive T in AVL, positive or isoelectric T in lead-I, and maximal voltage of TWI in precordial leads > lead III – is 92% sensitive and 100% specific for cardiac memory [(12)]. Septal bounce or paradoxical septal motion post-CABG is a well-known phenomenon present in our case simulating left bundle branch bounce on Echocardiogram, which may cause abnormal ventricular activation but is not known to cause persistent T wave inversion.

These criteria may not apply to our case, there was no documented aberrant electrical conduction, and TWI was not present in lead III. Thus, CM is unlikely based on the established CM definition and septal bounce with altered ventricular repolarization may not suffice to consider CM. Therefore, we conclude to the best of our knowledge, precordial TWI two months post-CABG is an ECG sign of myocardial reperfusion presenting late.

Conclusion:

Anterior precordial T wave inversion can present as a late ECG sign of successful myocardial reperfusion in Non ST segment myocardial infarction after revascularization via CABG.

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