

ST-Segment Elevation in Inferior Leads Associated with Acute LAD Stent Thrombosis

Ersin Ibisoglu*, Haci Murat Gunes, Filiz Kizilirmak Yilmaz, Gultekin Gunhan Demir, Ibrahim Oguz Karaca, Beytullah Cakal, Mehmet Onur Omaygenc, Ekrem Guler, Fatih Erkam Olgun, Umeyir Savur, Filiz Celebi, Deniz Dilan Naki and Tayyar Gokdeniz

Department of Cardiology, Faculty of Medicine, Istanbul Medipol University, Istanbul, Turkey

Abstract

A 45 year-old man was admitted to emergency service with new onset chest pain for 30 minutes. Electrocardiogram (ECG) showed ST-segment elevation in D1, AVL and V1-V6 leads and coronary angiography (CAG) detected proximal total occlusion of left anterior descending artery (LAD). Primer percutaneous intervention (PCI) was performed. Following intervention, ECG was normalized. Twenty minutes after PCI chest pain re-emerged and ECG showed ST-segment elevation in the D2, D3 and AVF leads. The patient was immediately transferred to catheterization laboratory and CAG showed acute LAD stent thrombosis. The culprit was successfully treated and the patient was discharged after three days with no complaint.

Keywords: Acute coronary syndrome; Electrocardiogram; Coronary angiography

Introduction

ST-segment elevation is associated with several conditions including acute myocardial infarction, myocarditis, pericarditis, early repolarization, electrolyte imbalance. Electrocardiogram (ECG) findings in acute coronary syndromes (ACS) are predictive for the responsible artery and lesions [1]. In patients with ST-segment elevation in the anterior precordial leads especially indicates left anterior descending (LAD) coronary artery occlusion and elevation in the inferior and/or lateral precordial leads especially indicates left circumflex artery (LCX) and/or right coronary artery (RCA) occlusion [2,3]. However ST-segment elevation in the inferior leads has also been described especially at the distal part of LAD occlusion [4,5]. In our case, we detected ST-segment elevation in the inferior leads associated with acute LAD stent thrombosis, following primer percutaneous coronary intervention (PCI) of LAD proximal occlusion which had presented with anterior myocardial infarction.

Case Report

A 45 year-old male patient, heavy smoker, was admitted to emergency service with chest pain starting for 30 minutes. On the first physical examination, his blood pressure was 160/80 mmHg, pulse 82 bpm and normal heart and lung sounds were present. The initial standard 12-lead ECG showed ST-segment elevation in leads D1, AVL, V1-V6 and ST-segment depression in leads D2, D3, AVF. Unfractionated heparin (10000 U), clopidogrel (600 mg) oral loading dose and oral aspirin (300 mg) were administered, and then patient was transferred to catheterization laboratory for coronary angiography (CAG). Proximal occlusion of LAD was detected. Primer PCI was performed to the proximal LAD occlusion by implantation of overlapped 2,75'23 and 2,75'38 everolimus-eluting stents (Xiencepro, AbottVascular, SantaClara, California). Postdilatation was done with 3,0'15 balloon (NC Trek, AbottVascular, SantaClara, California) (Figures 1-3). Chest pain disappeared and the patient was transferred to the coronary intensive care unit (CICU). ECG was normalized. After 20 minutes, chest pain re-emerged, ECG was taken immediately and ST-segment elevation in leads D2, D3, AVF, V5-V6 was detected (Figures 4 and 5). The patient was transferred to the catheterization laboratory again. CAG revealed stent thrombosis starting from the proximal part of the stent with TIMI 3 flow. RCA was not different

from first CAG. After administration of ticagrelor (180 mg) oral loading dose and aggrastat infusion, in-stent thrombosis was crossed and dilated with 3,0'15 balloon (NC Trek, AbottVascular, SantaClara, California) (Figures 6-8). PCI was performed successfully and the patient was transferred to the CICU again. The patient was hospitalized for three days in CICU and was discharged with no complaint.

Discussion

ST-segment elevation related with LAD occlusion usually presents in leads V1-V4 [1]. Occasionally, when occlusion is in the distal part of the artery or in the branches that supply inferior wall, ST-segment elevation occurs in leads D2, D3, AVF, V5-V6 [4,5]. In this patient, we detected thrombosis in the proximal part of the LAD that did not preclude TIMI 3 coronary flow. Embolism of distal LAD or collateral branches may cause ST-segment elevation in inferior leads. Similar cases are rare in the literature. There is a study group of 42 cases with LAD occlusion and ST elevation in inferior leads that Sapin et al. [6] studied and a case of LIMA-LAD bypass grafting occlusion presented with acute inferior myocardial infarction [7].

Another cause of ST-segment elevation in inferior leads seen in acute LAD stent thrombosis following primer PCI of LAD occlusion can be explained by electrical events. The extracellular electrical potential is positive in the normal myocardial tissue (Figure 9). The extracellular positive electrical potential in the anterior wall region of the myocardium tends to be negative, due to injury from acute anterior myocardial infarction (Figure 10). Stent thrombosis in the early period after primer PCI of LAD causes rapidly impaired perfusion of anterior myocardial tissue, causing the negative electrical potential of the tissue under the injured front wall to become more pronounced. Since the

***Corresponding author:** Ersin Ibisoglu, Department of Cardiology, Faculty of Medicine, Istanbul Medipol University, Istanbul, Turkey, Tel: +905322438586; Fax: +902124607070; E-mail: e_ibisoglu@hotmail.com

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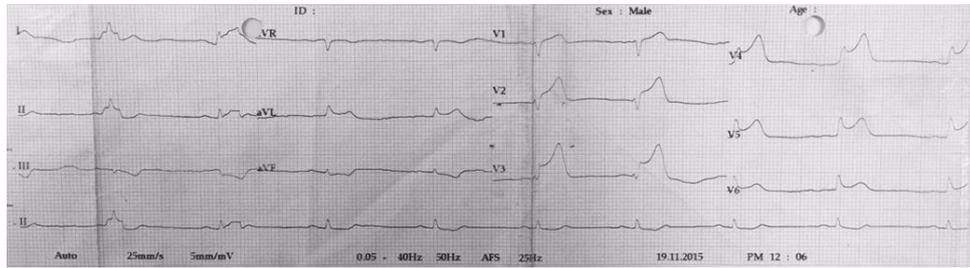


Figure 1: ECG of anterior myocardial infarction.

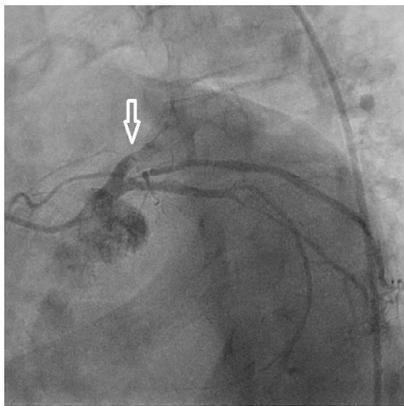


Figure 2: Image of LAD proximal occlusion.

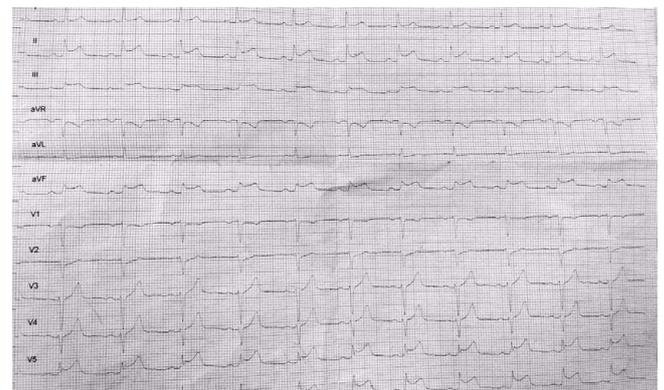


Figure 5: ECG of inferior myocardial infarction.

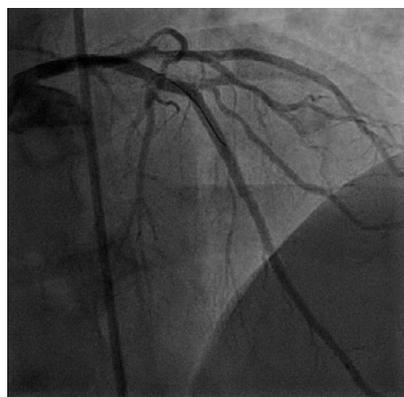


Figure 3: Successful revascularization.

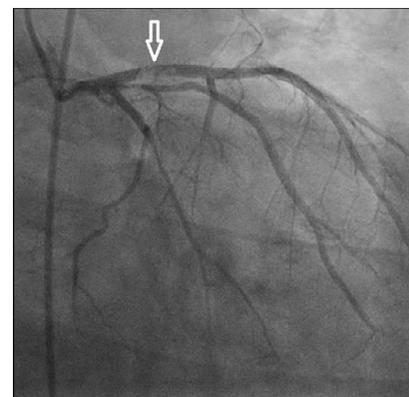


Figure 6: Image of acute LAD thrombosis.

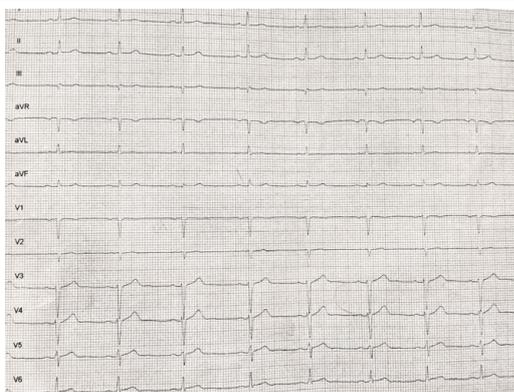


Figure 4: ECG of normal sinus rhythm.



Figure 7: Image of RCA.

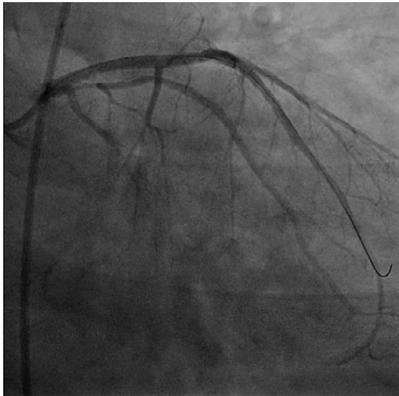


Figure 8: Successful revascularization.

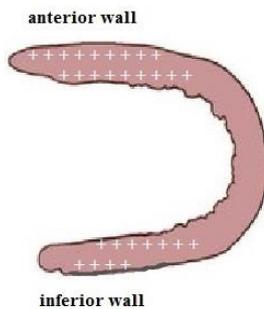


Figure 9: LV normal myocardial potential.

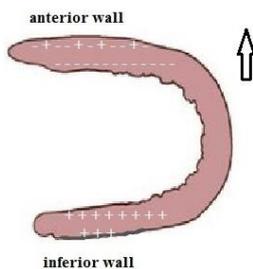


Figure 10: Potential of anterior myocardial infarction.

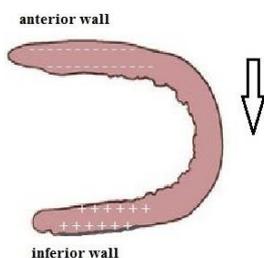


Figure 11: Potential of inferior myocardial infarction.

extracellular potential of inferior myocardial wall stays positive during MI, reason of ST-segment elevation in inferior leads could be the vector that head for negative to positive extracellular potential (Figure 11) [8].

Conclusion

The present case demonstrates that, inferior ST-segment elevation is not always associated with RCA or LCX occlusion. Rapid electrical changes, especially after stent thrombosis or occlusion of LAD distal collaterals may cause this condition. We would like to say that ECG still offers us new information despite a lot of know-how.

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