Case Report

Acute Inferior Wall Myocardial Infarction with Total Occlusion of Distal Left Anterior Descending Coronary Artery in a Young Adult

Keyur Vora*
Department of Cardiology, CARE Hospitals, Institute of Medical Sciences, India

Abstract

We report a case of an acute inferior wall myocardial infarction as per electrocardiogram (ECG) criteria. Customarily, this represents thrombotic occlusion of right coronary artery (RCA) or left circumflex coronary artery (LCX). Contradicting, the coronary angiogram revealed total occlusion of distal left anterior descending coronary artery (LAD) and lesion free LCX & RCA. Our case report highlights the rare occurrences of dissociation of electrophysiological abnormalities and standard coronary artery perfusion zones.

ABBREVIATIONS

ECG: Electrocardiogram; CAG: Coronary Angiography; LAD: Left Anterior Descending Coronary Artery; LCX: Left Circumflex Coronary Artery; RCA: Right Coronary Artery

INTRODUCTION

Electrocardiographic interpretation of acute ST segment elevation myocardial infarction (MI) is a basic diagnostic practice in cardiology emergencies. Also it is imperative to correlate ST segment changes in the precordial and limb leads to define anatomy of myocardial wall being compromised. The anatomical correlation also gives considerable suggestion about culprit vessels. Typically, inferior or inferolateral wall MI is expected to arise from culprit vessel of RCA (Right coronary artery) or LCX (Left circumflex coronary artery). We report a case of acute inferior wall MI with ST elevation in II, III and aVF and contrary angiographic evidence of total occlusion of left anterior descending coronary artery.

CASE PRESENTATION

A 31-year-old male patient presented with chest pain radiating to left shoulder and upper arm with stable hemodynamics. First episode of self-limiting angina was noticed before 6 hours. However, physical activity was continued with intermittent angina which aggravated since last 1 hour on the pain scale from 3/10 to 7/10. Patient was overweight (BMI 26.6), had 6 pack years of smoking history and no co-morbidities. On physical examination, blood pressure was 126/90 mmHg, heart rate was 76/min regular and there were no abnormal findings on examination. ECG reveals ST segment elevation of 5 mm in II, III and aVF as well as V5, V6 leads (Figure 1). Reciprocal ST depression changes were noted in V1-V3 and aVL (Figure 1). Echocardiography study reveals severely hypokinetic basal to mid inferior wall and inferolateral wall with preserved wall thickness, mild mitral regurgitation and nearly normal contractility of other segments.

Institutional STEMI protocol was followed and patient was transferred for emergency coronary angiography (CAG). CAG reveals distal LAD total occlusion (Figure 2A), nondominant and lesion free LCX (Figure 2B) and lesion free RCA continuing as PDA (Figure 2C). Thrombus aspiration followed by plain balloon angioplasty was done to distal LAD as diameter of distal part was too less to perform stenting. Post procedure TIMI III flow was not achieved. However, ECG reveals complete resolution of ST elevation changes to isoelectric line in II, III & aVF (Figure 3). Moreover, immediate post procedure ECG reveals ST elevation in V3-V6 leads. Complete resolution of ST segment was appreciated at 24 hours post procedure (Figure 3).

DISCUSSION

ECG interpretation is a basic and crucial diagnostic study to
Figure 1 12 lead ECG reveals acute phase of inferior wall myocardial infarction with laterisation. ST segment elevation in II, III, aVF, V5 & V6 with upright T wave merging with the ST segment.

Figure 2 A. Coronary angiography reveals distal total occlusion of left anterior descending coronary artery (depicted by red arrow). B. Left circumflex coronary artery is free of lesion and nondominant. C. Right coronary artery is free of lesion with continuation of posterior descending coronary artery.

Figure 3 (Top panel) Post plain balloon angioplasty to distal LAD ECG revealing complete resolution of ST segment to baseline level (red arrow); appearance of ST elevation among V3-V6 (red arrow). (Lower panel) Post balloon angioplasty ECG at 24 hours reveals complete resolution of ST segments in V3-V6 (red arrow).
rule out cardiac illness in emergencies. However, differential diagnosis of coronary artery diseases is not easier as thought previously. Especially, NSTEMI and vector counter-effects demands meticulous and comprehensive examination in addition to ECG. Thottuvelil Narayanan Sunil Roy et al. described a case of isolated acute inferior myocardial infarction due to occlusion of a wrapped LAD at the apex which continues as the large posterior descending coronary artery (PDA) beyond the occlusion [1]. Continuation of the left anterior descending coronary artery to form the posterior descending artery is rare coronary anomaly [2]. Collateral circulation affecting the ECG evolution has been described by Honda et al [3]. In this case, post procedure resolution of ST changes in inferior leads is the result of reestablishment of perfusion mostly by collaterals. However, new ST elevation in V3-V4 is possible if there is embolization of micro-thrombi to collaterals supplying lateral walls. Finally, complete resolution of all ST changes after 24 hours is the end result of complete dissolution of microthrombi by anticoagulant effect of glycoprotein IIb/IIIa inhibitors.

Interestingly, our case depicts that definitive changes in ECG is incompetent for determination of ischemic territory of culprit vessels. In general, the LAD artery and its branches supply most of the interventricular septum; the anterior, lateral, and apical wall of the left ventricle. Relevant ischamic changes in ST segments are noted in V1-V4. Reciprocal ST changes are also not uncommon in inferior leads during ST elevation MI due to LAD occlusion. Paradoxically, isolated ST elevation in inferior wall leads along with absence of ST segment changes in precordial leads during single LAD occlusion is rare. Moreover, echocardiographic evidence of inferior wall-motion abnormalities demands more understanding of electrophysiological properties for widely available ECG facilities. In the observation of a case at our institute, a patient with both anterior and inferior wall MI with angiographic evidence of proximal and distal occlusion of wrapped LAD, anterior wall MI was resolved to isoelectric line by primary direct stenting to proximal lesion but guide wire could not be crossed from distal lesion and inferior wall MI persisted which resolved after 24 hours. In our case, angiographic evidence of PDA arising from dominant RCA rules out any possibility of anomalous PDA origin from LAD. However, small sized PDA doesn’t rule out the possibility of wrapped LAD supplying inferolateral wall as well. This challenges the need to push the technical limits of angiographically visible coronary system including collaterals.

CONCLUSION

Our case depicts the rare occurrence of left dominant circulation affecting the inferior wall of myocardium. Even though this case reveals angiographic evidence of LAD total occlusion and revascularization by coronary intervention, it has posted an open question that angiographic evidence of end organ supply is not a complete calculation of anatomical and physiological contribution of individual coronary arteries. In case of double or triple vessel disease, it would be critical to determine culprit vessel if the patient presents with acute myocardial infarction with regional ST changes on ECG. The benefit of revascularization is completely determined upon door to balloon time and specific cases need ample consideration to develop tools to support interventionists and surgeons for meticulous decisions and favorable outcomes. Our case reveals an exceptional ECG with significant ST segment elevation changes in the absence of any lesion in proximal or middle segments of major coronary artery. Also, it is an exceptional ECG with significant ST changes of acute inferior wall MI with total occlusion of LAD.

REFERENCES