Case Reports

Dilemma of localization of culprit vessel by electrocardiography in acute myocardial infarction

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ABSTRACT

Acute coronary syndrome (ACS) and electrocardiography showing ST elevation in Lead aVR > V1 are considered specific for left main coronary artery lesion and also suggest extensive anterior wall myocardial infarction. In this backdrop, we are presenting an incidental observation of an association of ST elevation in lead aVR > V1 in isolated proximal left circumflex lesion in the setting of ACS, who later underwent successful primary percutaneous coronary intervention.

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1. Introduction

Management of Acute coronary syndrome (ACS) is based on the changes imparted in ST segment by ischemia. Total occlusion of an epicardial coronary artery leads to ST elevation, while non-occlusive lesion leads to ST depression. The lead aVR is an exception. The lead aVR is directed towards the left ventricular cavity. Global or basal (septal) subendocardial ischemia can lead to reciprocal ST segment elevation in lead aVR. So chest pain with lead aVR ST elevation is traditionally being classified in high-risk non-ST elevation ACS. We are reporting an interesting case of lead aVR ST elevation which turned out to be an isolated circumflex thrombotic total occlusion, and successful primary PCI normalized the ST changes in electrocardiogram (ECG).

2. Case report

A 40-year-old male with class II (NYHA/CSS) recent onset effort angina (< 3 h) was admitted to the ICU. On clinical examination, he had stable vital signs, jugular venous pressure was not raised, and normal heart sounds, with no murmurs and added sounds. ECG was showing ST elevation in aVR (2.5 mm) > V1 (2 mm) with an upright T wave in lead aVL, significant ST depression in leads II (2.5 mm), III (1 mm), aVF (1.5 mm), and lead I (1 mm) (Fig. 1A) associated with positive troponin I test. Two-dimensional echocardiography (2DEcho) did not show significant regional wall motion abnormalities. He was taken for emergency coronary angiogram (CAG), which was showing short and normal left main coronary artery (LMCA) bifurcating into left anterior descending artery (LAD) and left circumflex...
LAD was type II normal. LCx was co-dominant with total occlusion in proximal LCx about 20 mm away from LCx ostium (Fig. 2A). RCA was co-dominant and normal with a normal PDA branch. Subsequently, PCI to proximal LCx was done with a Xience Prime 2.75 mm/C2 18 mm at 8 atm followed by post-dilation 2.75 mm/C2 8 mm NC balloon at 14 atm with good TIMI III flow (Fig. 1B) and post-procedure ST segment in lead aVR became normal along with tall R wave and upright T wave in leads V1 and V2, suggesting evolved posterior wall MI.

3. Discussion

In ST elevation acute myocardial infarction (AMI), the ST segment changes in 12-lead ECG form the basis of diagnosis, management, and prognosis. Transmural infarction usually causes ST elevation. Lead aVR is neglected due to the lead being not adjacent to other electrocardiographic leads and being directed to LV cavity. Lead aVR has a frontal plane vector of −150°, which faces the inside of heart from the right shoulder and is oriented to look at the outflow area of right ventricle and basal part of interventricular septum. Lead aVR ST segment elevation has been reported in cases of acute LMCA occlusion, LAD with large first septal occlusion, and also rarely in isolated LCx occlusion. Global subendocardial ischemia of left ventricle leads to reciprocal ST elevation in aVR in LMCA occlusion. Large first septal infarction creates the current of injury towards the superior part of heart leading to aVR ST elevation in proximal LAD occlusion. In circumflex occlusion when there is involvement of isolated posterobasal area can also lead to aVR ST elevation because of the superior direction of current of injury. In general, the current of injury resulting from occlusion of the LCx has a mean vector that forms an obtuse angle with the axis of aVR, i.e. towards leads I and aVL. Therefore, one would expect ST-segment depression in aVR with acute LCx occlusion. Yamaji et al. proposed that lead aVR ST elevation > lead V1 ST elevation suggests left main occlusion. The major criticism in the editorial by Wellens et al. was that Yamaji et al. excluded circumflex occlusions from their study. Our case is a classical example of lead aVR ST elevation in isolated circumflex occlusion. In our case, the axis was superior, as both aVR and aVL were upright (Fig. 3). Therefore, the ST depressions in II, III, and aVF were secondary changes. The superior ST axis is likely due to the involvement of the high basal lateral area in our patient, which could be the cause lead aVL was not showing ST depression, while lead I had >1 mm ST depression. We observed this as an interesting electrocardiographic clue that can differentiate LMCA from LCx occlusion. ST segment discordance in leads I and aVL is a clue to differentiate LCx occlusion from LMCA and LAD occlusion. Since aVL is a unipolar lead, it is more specific to direction of current of injury, than LI which is bipolar.
Usually in LMCA occlusion, aVR > V1 ST segment elevation is associated with ST depression in both lead aVL and LI along with other leads. We suggest that this ST segment discordance between the adjacent leads aVL and LI can be an electrocardiographic clue to differentiate isolated LCx from LMCA occlusion. This rare variation may be due to short left main (LMCA), Type II LAD, and early origin of circumflex artery. Early origin circumflex along with absent ramus intermedius might be the reason for preferential involvement of high lateral region in our case.

An ECG with high lateral leads might have given a better clue. Since the area of infarction faces more to lead aVL than L1 being a high lateral infarction may be the reason for aVL-L1 discordance. Therefore, ST elevation in lead aVR > V1 need not be always due to LMCA lesion. With the findings of present clinical case, we suggest that ST elevation in lead aVR > V1, with no ST depression in aVL may be an electrocardiographic clue to suspect LCx lesion. However, further studies with large number of cases, which involve circumflex occlusion along with similar ECG changes, are needed to confirm this finding.

Conflicts of interest

The authors have none to declare.

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