VIA MEDICA

INTERESTING ELECTROCARDIOGRAM

Cardiology Journal
2011, Vol. 18, No. 2, pp. 204–206

ISSN 1897–5593

Dilemma in predicting the infarct-related artery in acute inferior myocardial infarction: A case report and review of the literature

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Abstract

The electrocardiogram (ECG) has being used for decades as a reliable and inexpensive tool to diagnose acute myocardial infarction (AMI). ECG diagnosis of an occluded coronary artery is of the utmost importance. We present the case of a 46 year-old man admitted to our hospital for inferior AMI. The ECG findings suggested right coronary artery occlusion. Coronary angiography showed left circumflex artery occlusion. We also briefly review the literature. (Cardiol J 2011; 18, 2: 204–206)

Key words: electrocardiogram, myocardial infarction

Introduction

Inferior acute myocardial infarction (AMI) represents 40–50% of all AMIs. The culprit lesion is almost exclusively either in the right coronary artery (RCA) or the left circumflex coronary artery (LCx). Without doubt, the electrocardiogram (ECG) plays a crucial role in identifying the infarct-related artery because it is cheap and universally available. ECG information about the culprit vessel is important because the prognosis and therapeutic strategies may vary between RCA- and LCx-related inferior AMI [1].

Case report

A 46 year-old man presented at the emergency department (ED) of our hospital complaining of typical chest pain, which had begun one hour prior to admission to the ED. The patient was on antihypertensive medication and had no previous history of coronary artery disease. Twelve-lead ECG revealed sinus rhythm 75 bpm, and ST-segment elevations in the inferior leads II, III and aVF, and ST-segment depressions in leads I, aVL and V1 through V4. Modified right ventricular leads V3R through V5R showed ST elevations (Fig. 1). The patient had no symptoms during the ECG recording. Blood pressure was 110/70 mm Hg, and on physical examination the patient was in mild respiratory distress. The plasma level of both troponin I and creatine kinase-MB were elevated on admission, 7.6 ng/mL (reference value < 0.01 ng/mL), and 37.1 ng/mL (reference value: 0.54–4.19 ng/mL), respectively. Other laboratory tests were within normal limits. Bedside echocardiography revealed wall motion abnormalities in the inferior and posterior walls. Global ejection fraction was 48%. No signs of valve disease were observed. Coronary angiography was immediately (within two hours of symptom onset) performed. Because of ECG findings suggesting RCA occlusion, the left coronary artery was injected first. Surprisingly, total occlusion of the LCx distal to the first obtuse marginal branch (OM) was shown. The RCA was dominant without significant (≥ 50%) lesions (Figs. 2A, B). Balloon angioplasty followed by

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Received: 31.10.2010 Accepted: 07.01.2011

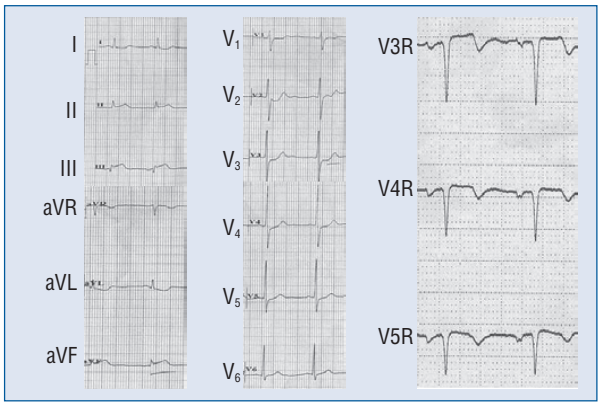

Figure 1. Electrocardiogram at admission shows ST-segment elevations of 1.0, 2.0, and 1.2 mm, respectively, in the inferior leads II, III and aVF. ST-segment depressions are observed in leads I, aVL and V1 through V4. Modified right ventricular leads, i.e. V3R through V5R, show 1.0 mm ST elevations.

stenting was performed with a stent of 3.0 mm in diameter and 12 mm in length. Normal TIMI grade 3 flow was achieved. The patient was discharged four days after admission to our clinic.

Discussion

Various ECG criteria have been suggested to predict the culprit artery in acute inferior AMI based on analysis of ST-segment elevation and depression in different leads [2]. The RCA occlusion results in an ST-segment vector directed inferiorly and rightward, while in LCx occlusion, the vector is directed more posteriorly and leftward. In RCA occlusion, there will therefore be more ST elevation in lead III than in lead II, with resultant ST depression in lead I. In a case of LCx occlusion, the vector will point towards lead II, leading to ST elevation or an isoelectric ST segment in lead I and more ST elevation in lead II than in lead III [3]. Greater ST segment depression in lead aVL than in lead I has also been found to be highly predictive of RCA occlusion [4]. More recently, Kanei et al. [5] pointed out the importance of ST-segment depression in lead aVR as a sign of LCx occlusion. In their study, ST-segment depression in lead aVR represented involvement of either the LCx or the RCA with a large posterolateral (PL) branch, which supplies blood flow to the inferolateral wall. The specificity and sensitivity for lead aVR to predict LCx involvement was 94% and 70%, respectively [6]. Assali et al. [7] found that a decrease in R-wave amplitude and an increase in S-wave amplitude with an S/R ratio of greater than 1:3 in lead aVL predicted RCA occlusion, whereas an S/R ratio of 1:3 or less predicted LCx occlusion. In our case, the ECG showed: (1) ST↑ III > ST↑ II, (2) ST↓ aVL > ST↓ I, and (3) an S/R < 1/3 in aVL, suggesting RCA occlusion, although the culprit artery was in fact the LCx.

In RCA occlusion, the presence of right ventricular involvement is important because it identifies a subgroup of patients at high risk [8]. Therefore it is necessary to record the right precordial leads in inferior AMI. ST elevation of at least 1.0 mm in V4R identified proximal RCA occlusion with a sensitivity of 95% and specificity of 69% [9]. In contrast, dominant LCx occlusion was suggested by reciprocal ST-segment depression in lead V4R [9].

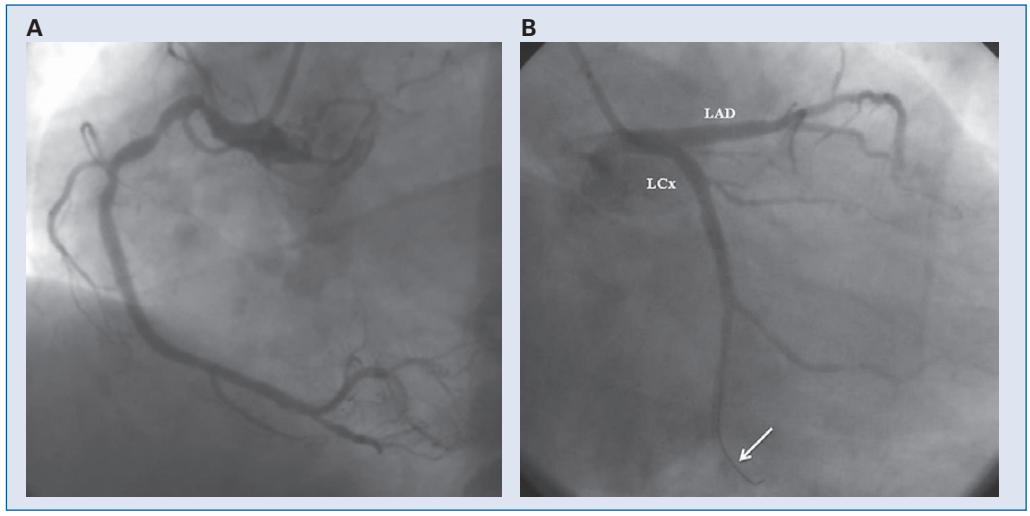

Figure 2. **A.** Coronary angiography shows dominant right coronary artery with diffusely diseased posterolateral branches; **B.** Left anterior oblique caudal angiographic view shows left circumflex artery (LCx) with large distal branches and wire (arrow) has passed through the occlusion before stenting; LAD — left anterior descending artery.

In our case, in spite of 1.0 mm ST elevation in V4R, RCA occlusion was not observed. We suggest two possible explanations for this finding. Firstly, it is known that the LCx provides some vascular supply to the right ventricle in some individuals. Secondly, a posteriorly directed ST vector, which is represented as ST depressions in leads V2–V3 in the horizontal plane, could result in reciprocal ST elevations in the right-sided leads RV3–RV5 representing the right ventricular free walls.

Kosuge et al. [10] reported that magnitude of ST depression in V3 relative to the ST elevation in III (ST↓ V3/ST↑ III) was useful in predicting the culprit artery in inferior AMI. They found that ST↓ V3/ST↑ III of less than 0.5 indicated proximal RCA occlusion; a ratio of 0.5 to 1.2 indicated a distal RCA occlusion; and a ratio of more than 1.2 was related to an LCx artery occlusion. According to these criteria, we suspected a proximal RCA occlusion, but the angiography and ECG findings did not agree.

Fiol et al. [3] described three steps in indentifying the occluded artery (RCA or LCx). In the first step of the algorithm, ST deviation in lead I was checked. In the case of depression, the occlusion was located in the RCA; in the absence or in the case of an elevation, it was located in the LCx. When the ST-segment is isoelectric, a second step is needed. ST elevation in leads II and III were assessed. When the ST elevation in II was greater than or equal to that of III, the occlusion was located in the LCx. When the ST elevation in III was greater than or equal to that in II, one should proceed to the third step. When the sum of ST depression in leads V1 through V3 divided by the sum of ST elevation in leads II, III and aVF was greater than 1, the culprit artery was the LCx; when it was equal to or less than 1, the culprit artery was the RCA.

Using all the above-mentioned ECG criteria, in our case a RCA occlusion was predicted before coronary angiography. We think that the patient's coronary angiography findings may provide an explanation for the unexpected finding. The posterolateral branches of the RCA are diffusely diseased (Fig. 2A), while the LCx has rather large distal branches, probably subtending large parts of the infero-posterolateral wall of the left ventricle (Fig. 2B). Hence, although the RCA is dominant, the LCx could be the more important provider of blood supply to these myocardial segments. Despite anatomic right dominance, diffuse disease of the posterolateral wall could result in balanced dominant circulation from a physiological perspective. Also individual variation of the heart's position within the thorax could explain differences in ST deviations in the 12-lead ECG system. For example, differences in the position of the inferior wall have been observed [11].

Conclusions

In conclusion, our case highlights that while ECG criteria and algorithms are very useful and important in detecting the site of coronary artery occlusion in most cases, there are limitations, as in our case, and therefore, there need to be further studies in this area.

Acknowledgements

The authors do not report any conflict of interest regarding this work.

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