

Published as:

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Year: 2007

Title: The value of ECG lead aVR in the differential diagnosis of acute inferior wall myocardial infarction

Journal Internal Medicine

Volume: 46

Issue: 12

Pages: pp795-799

ISSN: 1349-7235

URL: http://www.jstage.jst.go.jp/article/internalmedicine/46/12/46_795/_article

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The value of ECG lead aVR in the differential diagnosis of acute inferior wall myocardial infarction

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Methods: The ECG features were studied in 90 patients with acute inferior myocardial infarction where IRA was confirmed by coronary angiography.

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Conclusions: ST depression in aVR is common in patients with LCX-related acute inferior myocardial infarction. The ST changes in this lead are associated with an excellent specificity and a good sensitivity in differentiating LCX from RCA as the IRA.

Key words: acute myocardial infarction; infarct-related artery; coronary angiography; ECG.

Introduction

Inferior myocardial infarction, which accounts for 40-50% of all acute myocardial infarctions, can be caused by the occlusion of either the right coronary artery (RCA) or the left circumflex coronary artery (LCX) (the infarct-related artery, IRA).^[1] Timely identification of IRA, using 12-lead ECG at presentation, is important because the prognosis and therapeutic strategies may differ between LCX- and RCA-related inferior myocardial infarction (MI).^[2-5]

Previous studies have proposed ECG criteria in identifying the culprit artery following acute inferior MI. ST-segment elevation in lead I and /or ST-segment depression in both lead V₁ and V₂ indicate LCX occlusion.^[6-11] A greater ST-segment elevation in lead II than in lead III, plus a greater ST-segment depression in lead I than in lead aVL also suggests LCX rather than RCA occlusion.^[6-11]

The value of lead aVR in the identification of IRA in patients with inferior myocardial infarction is unclear. In a recent study in a small group of patients, the presence and amount of ST segment depression in lead aVR has been found to predict LCX involvement during acute inferior myocardial infarction.^[12] The aim of the present study is to further investigate the sensitivity and specificity of aVR in differentiating LCX from RCA as a culprit artery for inferior myocardial infarction.

Methods

Patients

This study was approved by the Institutional Review Board of the First Affiliated Hospital of Zhengzhou University. Between July 2002 and December 2004, 160 consecutive patients were admitted to the coronary care unit of the First Affiliated Hospital of Zhengzhou University with acute inferior myocardial infarction.

Patients who met the following criteria were included in the study: 1) typical anginal chest pain lasting ≥ 30 min before hospital admission; 2) the time from the onset of chest pain to the ECG recording was less than 24 h; 3) ST elevation ≥ 0.1 mV in at least 2 of the 3 inferior leads (II, III, and aVF); 3) ST deviation in aVR; 4) elevation of the creatine kinase and its MB fraction to greater than twice of the upper limit; 5) underwent coronary angiography during hospitalization, where IRA were confirmed.

Patients with the following conditions were excluded from the study: 1) with a previous history of acute myocardial infarction, coronary artery bypass surgery or percutaneous coronary intervention prior to the current hospitalization; 2) ECG evidence of bundle branch block or left ventricular hypertrophy; and 3) significant stenosis in both RCA and LCX so that a single IRA could not be defined.

Of the 160 patients reviewed, 90 (62 men and 28 women, aged 27 to 78 years, mean 54.5 ± 10.3) met the inclusion criteria. Their ECG features and coronary angiograms were subsequently analyzed.

Seventy patients were excluded from the study because of a previous episode of myocardial infarction (n=26), bypass surgery for left and or right coronary artery lesions (n=2), previous percutaneous coronary intervention (n=1); left bundle branch block (n=19), stenosis of both RCA and LCX (n=32).

Coronary angiography

Coronary angiography was performed during hospitalization in all patients. The coronary angiography films were reviewed by two investigators who were blinded to the ECG findings. The IRA was identified by either 1) a total occlusion or a significant stenosis ($\geq 70\%$) of the RCA or LCX, or their major branches; or 2) arteriographic evidence of an intraluminal thrombus.

Electrocardiographic analysis

The 12 leads ECGs recorded on admission of all patients were analyzed by two investigators who were blinded to coronary angiography findings. In each of the standard 12 standard leads, ST segment deviation from the isoelectric line was measured 0.06 sec after the J point.^[12] Measurement was made to the nearest 0.25 mm.^[12] The TP segment was used as the isoelectric line unless tachycardia had caused fusion of the T and P waves, in which case the PR segment was used.^[11] There was an excellent agreement in ECG and coronary angiographic readings between the two investigators in a pilot study of 10 patients. In the case of a

disagreement between the two investigators on a particular ECG or coronary angiogram, a senior cardiologist from the cardiology department will be invited to make an arbitration agreement.

Statistical analysis

Data were expressed as means \pm SD. Chi-square test and Fishers exact test were used to compare differences in discrete variables, such as the ST deviation in different leads, between the LCX and RCA groups. Multivariate logistic regression analysis was performed to assess the predictive value of ST depression in lead aVR in LCX-caused inferior infarction against other ECG criteria. $P < 0.05$ was considered to be statistically significant.

Results

General findings

After reviewing the coronary angiogram, RCA and LCX was identified as the IRA in 70 and 20 patients, respectively. Proximal RCA occlusion was identified in 38 patients whereas proximal LCX occlusion was found in 11 patients. There was no significant difference in the proportion of proximal section occlusion between the two groups (54.3% vs 55.0%, $p > 0.05$).

In the LCX group, half of the patients had ST elevation in lead I, whereas only 1 patient in the RCA group had such ECG feature ($P < 0.001$, Table 1). A greater proportion of patients in the LCX group also had ST depression in leads V_1 and V_2

($P < 0.01$, Table 1).

ST depression in lead aVR of 0.1mv or more was found in 14 (70%) patients with LCX related infarction, and 4 (5.7%, $P < 0.001$) patients with RCA-related infarction. Within LCX group, those with ST depression in lead aVR has a higher peak blood CK—MB (160.2 ± 83.4 vs 120.5 ± 42.3 U/L, $P < 0.01$).

More patients in the RCA group than in the LCX group experienced ST elevation in lead II and III, and ST depression in lead I ($P < 0.01$, Table 1). Isoelectric ST segment on V_1 and depression on V_2 was also a feature when RCA was involved ($P < 0.01$, Table 1).

RCA as the culprit artery

The value of the traditional ECG criteria and lead aVR in diagnosing LCX or RCA as a culprit artery was listed in Table 2. With RCA-related infarct, a greater ST elevation in lead III than in lead II (Fig 1) had a sensitivity of 85% but a low specificity of 55%. A Greater ST depression in lead aVL than in lead I was associated with 90% specificity but 42.9% sensitivity. A high specificity and low sensitivity were identified when ST segment was isoelectric or elevated in lead V_1 , but depressed in lead V_2 (Table 2).

LCX as the culprit artery

As shown in Table 1, ST elevation in lead I was associated with a high specificity and

moderate sensitivity in diagnosing LCX involvement. ST elevation in lead I was observed in only one patient who had a proximal total occlusion of a very dominant RCA.

ST depression in both lead V₁ and V₂ was associated with 80% sensitivity and 87% specificity, respectively. ST depression in aVR was associated with 94% specificity and 70% sensitivity in diagnosing LCX as the IRA (Table 2).

Multivariate logistic regression analysis showed that ST depression on lead aVR was an independent predictor for LCX-related inferior infarction (OR=1.028, 95% CI 1.002-1.469, P=0.013).

Discussion

The RCA supplies blood mainly to the inferior myocardium, whereas the LCX supplies blood to the posterior, posterolateral, or posteroinferior myocardium.^[8] The present study found that in patients with inferior myocardial infarction, the RCA is much more likely than the LCX to be the culprit artery, with a ratio of 3.5:1. These results are consistent with previous studies by other groups where the RCA to LCX ratio was found to vary from 2.2:1 to 7.0:1.^[6-11]

In most patients the myocardial distribution of the RCA is slightly rightward in the frontal plane, and consequently the current of injury resulting from its occlusion will be reflected more in lead III than lead II. Conversely, the distribution of the LCX is slightly leftward in the frontal plane, and the current of injury from its closure

will be seen more in lead II than in lead III. An injury vector leftward enough to cause ST-segment elevation in lead I is common with LCX occlusion, but rare with RCA occlusion.

The current of injury with RCA occlusion is more or less perpendicular to the axis of lead aVR, whereas the current of injury resulting from occlusion of the LCX has a mean vector that forms an obtuse angle with the axis of aVR. Therefore, significant ST-segment depression in aVR is more likely to occur with LCX occlusion, as shown in the present study.

ST segment in lead V₁ may be elevated when RCA is occluded because it is influenced by the current of injury from right ventricular infarction. ST-segment depression both in V₁ and V₂ is the reciprocal changes resulting from ST-segment elevation in posterior wall of the left ventricle and is typical of LCX occlusion.^[9] The aVL lead faces the high-lateral segment of the left ventricle, and is the only lead truly reciprocal to the inferior wall. Inferior-wall myocardial infarction caused by RCA occlusion has greater ST-segment depression in lead aVL than in lead I.^[7]

The present study demonstrated that ST-segment depression of 0.1 mV or more in lead aVR is a good indicator for LCX occlusion, with a sensitivity of 70% and a specificity of 94%.

aVR is frequently ignored during the analysis of inferior myocardial infarction, but some investigators have suggested that this lead can provide information useful for the characterization of inferior myocardial infarction.^[13, 14] Menown and Adgey

^[14] reported that the display of lead aVR in an inverted format as lead -aVR improves the ECG classification of inferior- or lateral-wall myocardial infarction. However, Menown and Adgey's study lacked angiographic data and did not confirm the presence or absence of recanalization of the infarct-related artery. Kosuge and coworkers ^[15] studied inferior myocardial infarction with inferior ST-segment elevation in which TIMI grade 3 flow of the RCA or LCX was achieved within 6 h from symptom onset. They found that the degree of ST-segment depression in lead aVR is a useful predictor of impaired myocardial reperfusion in patients who have experienced inferior myocardial infarction; a significant ST depression in lead aVR is useful for identifying patients who are most likely to benefit from aggressive therapeutic strategies designed to improve myocardial reperfusion. ^[15]

Our study found only a small proportion of the patients with RCA-related myocardial infarction has a ST depression in aVR. However, 70% of patients with LCX related inferior myocardial infarction had a ST depression of 0.1 mV or more in aVR. The specificity and sensitivity of using aVR criteria in diagnosing LCX involvement is 94% and 70%, respectively. These results indicate that use a combination of traditional ECG criteria plus aVR ST depression may enhance the accuracy of 12-lead ECG in differentiating the culprit artery in patients with inferior-wall myocardial infarction.

The value of lead aVR and more importantly, its reciprocal lead -aVR, in diagnosing IRA may be further enhanced by orderly display of ECG leads. The

conventional 12-lead ECG displays the limb leads in a disorderly fashion with lead aVR at -150 degrees. ECG interpretation would be enhanced by displaying the limb leads in an orderly arrangement that starts with lead aVL and ends with lead III. Recent evidence show that many ECG changes would be ideally reflected on the orderly display ECG where lead -aVR is at 30 degrees. ^[16, 17]

One of the limitations of the present study is that the role of V3-V6 and V3R, V4R in the differential diagnosis was not evaluated and compared with aVR. There have been reports showing that in patients with LCX related acute inferior myocardial infarction, there is a higher frequency of ST elevation in V4-V6. ^[18] In LCX related inferior myocardial infarction, the amount of ST depression is often greater than the amount of ST elevation in V3. ^[19] ST elevation in V3R and V4R is a compelling evidence of RCA involvement and presence of right ventricular infarction. ^[19] In addition, there were some differences in the sensitivity or specificity of ST elevation in Lead III > II and ST depression in aVL > Lead I between our study and previous reports. We could not find any definitive causes for these gaps but differences in patient population may be one of the explanations.

In conclusion, the present study confirms the previous ECG findings that that, in inferior myocardial infarction, ST-segment elevation in lead I and/or ST-segment depression in both lead V₁ and V₂ indicate LCX involvement. A greater ST-segment elevation in lead III than in lead II, and a greater ST-segment depression in lead aVL than in lead I are the indicators for RCA occlusion. Since significant ST depression

in aVR is associated with a higher specificity and good sensitivity for LCX lesions, the ST changes in this lead should be carefully examined in all patients who are suspected of having inferior-wall myocardial infarction.

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Table 1. Relationship between ECG Changes and infarcted- related artery.

ST segment change	Infarcted- related artery		P value
	RCA (n=70)	LCX (n=20)	
Elevation in lead I	1	10	<0.001
Elevation in lead III > II	57	11	<0.001
Depression in lead aVL > lead I	30	2	0.006
Isoelectric or elevation in lead V ₁ , depression in lead V ₂	27	3	0.002
Depression in both lead V ₁ and V ₂ (n=25)	9	16	<0.001
Depression in lead aVR $\geq 0.1\text{mV}$	4	14	<0.001

Table 2. Utility of ECG Criteria in identifying the infarcted- related artery.

ST segment change	Sensitivity	Specificity	positive predictive value	negative predictive value
RCA				
Elevation in lead III > II	81.4	55.0	83.8	40.9
Depression in lead aVL > lead I	42.9	90	93.8	31.0
Isoelectric or elevation in lead V ₁ , depression in lead V ₂	39.0	85.0	90.0	28.4
LCX				
Elevation in lead I	50.0	98.6	90.9	87.4
Depression in lead aVR ≥ 0.1mV	70.0	94.3	77.8	91.6
Depression in both lead V ₁ and V ₂	80.0	87.1	64.0	92.3

RCA and LCX: right coronary artery and left circumflex coronary artery.

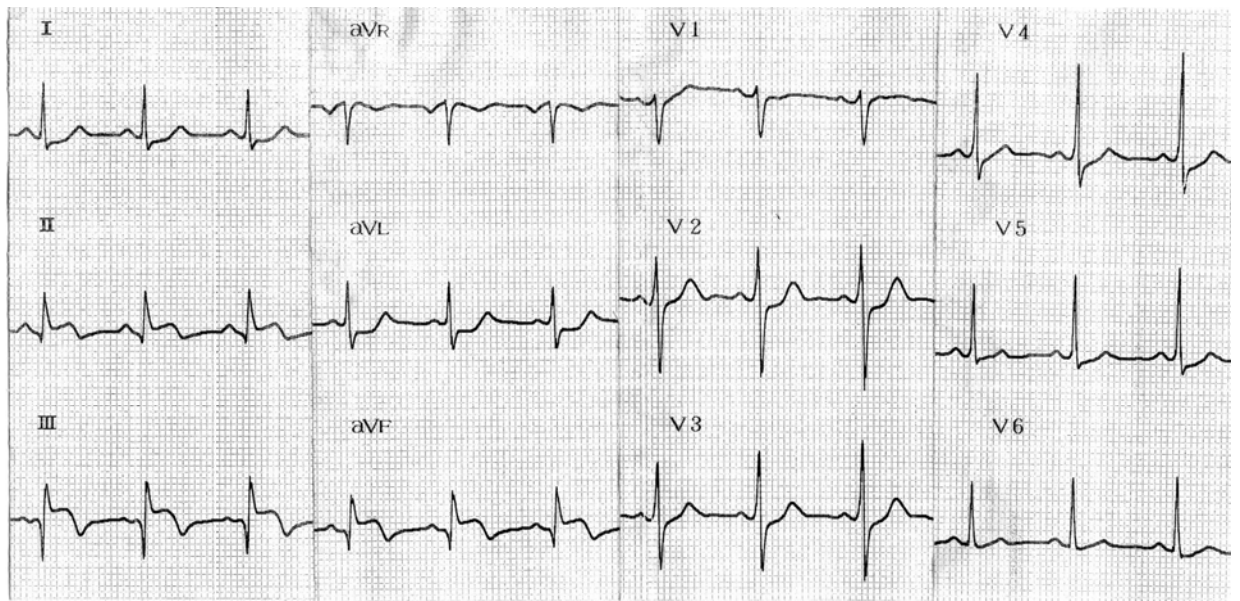


Fig 1. Occlusion of the right coronary artery (above), resulting in ST elevation in Lead III > II (below), ST depression in Lead aVL > Lead I, isoelectric or elevation in lead V₁, and depression in lead V₂,

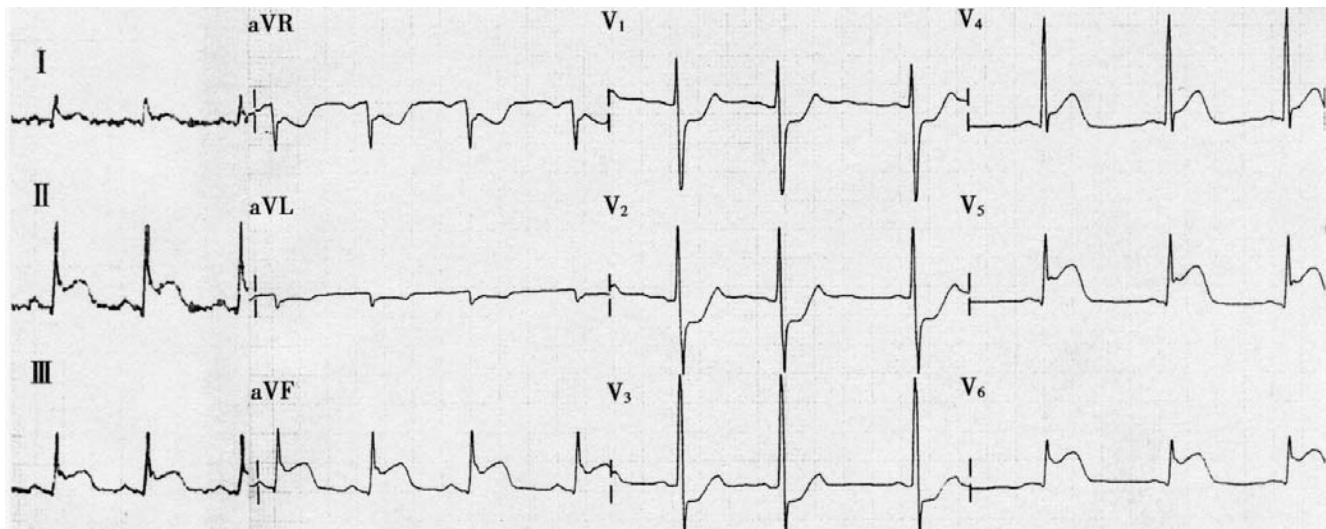


Fig 2. Occlusion of left circumflex coronary artery (above), resulting ST elevation in Lead I, V₄₋₆, and ST depression in Lead aVR, V₁ and V₂