

ST-segment depression in aVR as a predictor of culprit artery and infarct size in acute inferior wall ST-segment elevation myocardial infarction

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Abstract

Background: ST-segment depression in lead aVR in acute inferior wall ST-segment elevation myocardial infarction (STEMI) has recently been suggested as a predictor of left circumflex (LCx) artery involvement. The purpose of this study is to evaluate the clinical significance of aVR depression during inferior wall STEMI.

Methods: This study included 106 consecutive patients who presented with inferior wall STEMI and underwent urgent coronary angiogram. Clinical and angiographic findings were compared between patients with and without aVR depression ≥ 0.1 mV.

Results: The sensitivity and specificity of aVR depression as a predictor of LCx infarction were 53% and 86%, respectively. In patients with right coronary artery infarction, aVR depression was associated with increased cardiac enzymes and the involvement of a large posterolateral branch, which may explain the larger infarction.

Conclusions: ST-segment depression in lead aVR in inferior wall STEMI predicts LCx infarction or larger RCA infarction involving a large posterolateral branch.

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Keywords:

ECG; ST-elevation myocardial infarction; Inferior wall; Lead aVR

Introduction

In patients with acute ST-segment elevation myocardial infarction (STEMI), identifying the culprit artery on presenting electrocardiogram (ECG) can lead to earlier risk stratification and better guidance of therapy for reperfusion. The culprit artery of anterior STEMI is nearly always the left anterior descending artery (LAD), but inferior STEMI can be caused by an occlusion of either the right coronary artery (RCA) or left circumflex (LCx) artery. Various ECG criteria have been suggested to predict the culprit artery based on analysis of ST-segment elevation and ST-segment depression in different leads.^{1–9} More recently, ST-segment depression in lead aVR has been suggested as a predictor of LCx artery involvement.^{10–12} aVR depression was also

shown to be associated with significantly impaired myocardial perfusion.¹³

Predicting the culprit artery in inferior STEMI can be challenging because the dominance of the RCA and LCx can vary significantly among patients. Zhong-Qun et al¹⁴ recently showed that the conventional ECG findings used to predict culprit artery in inferior STEMI are less useful in patients with dominant LCx infarction. Few studies have correlated aVR depression with the relative dominance of the RCA and LCx. The purpose of this study is to evaluate the clinical significance of aVR depression during inferior wall STEMI and correlate the anatomical findings.

Methods

Study population

We identified 128 consecutive patients who were referred for coronary angiography and primary percutaneous coronary intervention for inferior wall STEMI at our institution

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between March 2004 and March 2009. Acute inferior myocardial infarction was diagnosed based on the presenting symptoms of chest pain for more than 30 minutes, ST-segment elevation of more than 1 mm in at least 2 of 3 inferior leads, and the typical increase in cardiac enzymes. Patients who had missing clinical or angiographic data, a history of inferior wall myocardial infarction, and right or left bundle branch block on ECG were excluded.

During the hospitalization, cardiac enzymes were measured every 6 hours from admission until their peak. Left ventricular ejection fraction was estimated with either left ventriculogram during coronary angiography in the right anterior oblique view or transthoracic echocardiogram within 24 hours of admission.

ECG analysis

Standard 12-lead electrocardiograms were recorded at a speed of 25 mm/s and voltage of 10 mm/mV. The presenting ECGs were analyzed by two independent readers blinded to the angiographic results. ST-segment deviation was measured at 60 ms after the J-point. The following findings were identified;

1. ST-segment elevation lead III > lead II
2. ST-segment depression in lead I ≥ 0.05 mV
3. The ratio of ST-segment depression in V3 to ST-segment elevation in III ≥ 1.2
4. ST-segment depression in lead aVR ≥ 0.1 mV

Coronary angiography

All patients underwent emergent or urgent coronary angiography. Angiographic findings were evaluated by an experienced angiographer blinded to the results of the ECG findings. The culprit artery was determined from angiographic characteristics of occlusion (occlusion due to thrombus formation or ulceration with decreased contrast density). Coronary artery stenosis of more than 70% was defined as obstructive and multivessel coronary artery disease was defined as having two or more coronary arteries with obstructive lesions. The coronary flow was determined using TIMI flow grading system. Based on the origins of the posterior descending artery (PDA) and the posterolateral branch (PL), the RCA was classified as dominant RCA (both PDA and PL are provided by RCA), non-dominant RCA (both PDA and PL are provided by LCx), or codominant RCA (PDA is provided by RCA and PL is provided by LCx). A “large” PL branch was defined as a PL branch larger than 2 mm in diameter and twice as long as the AV groove portion after the bifurcation of the RCA and the PDA.

Statistical analysis

Continuous variables are expressed as means \pm SD and discrete variables are presented as percentages. Sensitivity and specificity of ECG findings to predict the culprit artery were calculated. Patients were divided into two groups based on the presence or absence of aVR depression, and their clinical and angiographic characteristics were compared. Student *t* test was used to compare the continuous

variables and χ^2 analysis or Fisher’s exact test was used to compare the categorical variables. *P* < .05 was considered statistically significant.

Results

One hundred and six patients (mean age 60 ± 15 years, 74% male) were included in the study. The culprit artery was found to be the RCA in 86 patients, the LCx in 19 patients, and the LAD in 1 patient. Overall, 23 patients (22%) were found to have aVR depression. Baseline clinical characteristics were similar between the 2 groups (Table 1). The aVR depression group did have proportionally more patients with LCx infarction.

aVR depression as a predictor of culprit artery

aVR depression was seen in 53% of patients with LCx infarction compared to 14% of patients with RCA infarction, which results in a sensitivity of 53% and specificity of 86% to predict the LCx as the culprit artery. Other conventional electrocardiographic findings to help differentiate the culprit artery were analyzed and are listed in Table 2.

aVR depression as a predictor of size of infarction

Patients with aVR depression had significantly larger infarctions, as estimated by peak creatine phosphokinase (CPK) levels, than patients without aVR depression (CPK 3151 ± 2682 U/L vs 1933 ± 1954 U/L, *P* = .0169) (Table 1). Among patients with RCA infarction, those with aVR depression had significantly larger infarcts than those without aVR depression based on peak cardiac enzyme level (CPK 3692 ± 3403 U/L vs 1714 ± 1729 U/L, *P* = .0024). In addition, more of these patients were found to have a large PL branch (67% vs 16%, *P* = .0006) compared to those without aVR depression (Table 3). Among patients with LCx infarction, there was no significant difference

Table 1
Clinical and angiographic characteristics of the patients with and without aVR depression

	aVR depression (+) n = 23	aVR depression (-) n = 83	<i>P</i>
Age	58 \pm 12	61 \pm 15	.3793
Male	20 (87%)	57 (69%)	.1399
DM	5 (22%)	25 (30%)	.5975
HTN	13 (57%)	50 (60%)	.9351
Dyslipidemia	10 (43%)	41 (49%)	.7895
Smoking	12 (52%)	33 (40%)	.4079
Culprit artery			
LAD	1 (4%)	0	
LCx	10 (43%)	9 (11%)	.0006
RCA	12 (52%)	74 (89%)	
Killip Class I	19 (83%)	68 (82%)	.8167
CPK (U/L)	3151 \pm 2682	1933 \pm 1954	.0169
CK-MB (ng/mL)	209 \pm 201	124 \pm 96	.0050
LVEF (%)	53 \pm 10	52 \pm 11	.6722
Multivessel disease	9 (39%)	48 (58%)	.1753
TIMI0	19 (83%)	61 (73%)	.5319

DM; diabetes mellitus, HTN; hypertension, CPK; creatinine phosphokinase, LVEF, left ventricular ejection fraction.

Table 2
The sensitivity and specificity of various ECG criteria to predict the culprit artery

	Sensitivity	Specificity	PPV	NPV
III/II > 1 (RCA)	94	58	91	69
ST depression I (RCA)	86	63	91	50
ST depression V3 /				
ST elevation III (LCx) ≥ 1.2	21	98	67	85
aVR depression (LCx)	53	86	45	91

PPV, positive predictive value; NPV, negative predictive value.

between those with and without aVR depression. Of note, 47% of patients who had LCx infarction had either a left dominant or co-dominant coronary artery distribution.

Discussion

Various ECG findings^{1–7} and two algorithms (Fiol et al,⁸ Tierala et al⁹) have been evaluated to differentiate the culprit artery in acute inferior wall STEMI.^{1–7} Still, there is a small portion of patients whose culprit artery can not be accurately identified by these criteria, likely because of the variation in their coronary anatomy and dominance. The main finding of our study is that aVR depression represents myocardial infarction involving either the LCx artery or RCA with a large posterolateral branch, which both supply blood flow to the inferolateral wall and does not necessarily differentiate the culprit artery. This finding is consistent with the recent study showing that the conventional ECG findings are less useful to predict the culprit artery in patients with a dominant LCx, since both dominant RCA and dominant LCx arteries supply the same areas regardless of which vessel they originated from.¹⁴

The display of lead aVR (–150°) in an inverted format as lead –aVR (+30°) lies between lead I (0°) and lead II (60°) (Fig. 1). Thus, aVR depression means –aVR elevation, which represents the infarct of the apical and inferolateral walls, usually supplied by the posterolateral branch of either the RCA or LCx itself.¹⁰ This explains our finding that aVR

Table 3
Comparison of patients with and without aVR depression in subgroup of patients with RCA infarction and LCx infarction

	aVR depression (+)	aVR depression (–)	P
RCA	n = 12	n = 74	.8201
CPK (U/L)	3692 ± 3403	1714 ± 1729	.0024
CKMB (ng/mL)	259 ± 249	112 ± 79	.0001
multivessel	8 (67%)	45 (61%)	.9466
Large PL	8 (67%)	12 (16%)	.0006
Left/co-dominance	0 (0%)	9 (12%)	.3484
LVEF (%)	52 ± 10	52 ± 10	1.0000
LCx	n = 10	n = 9	
CPK (U/L)	2718 ± 1522	3733 ± 2786	.3313
CKMB (ng/mL)	165 ± 119	224 ± 159	.3603
multivessel	1 (10%)	3 (33%)	.3034
Left/co-dominance	5 (50%)	4 (44%)	1.0000
LVEF (%)	53 ± 11	46 ± 11	.1840

DM; diabetes mellitus, HTN; hypertension, LVEF: left ventricular ejection fraction.

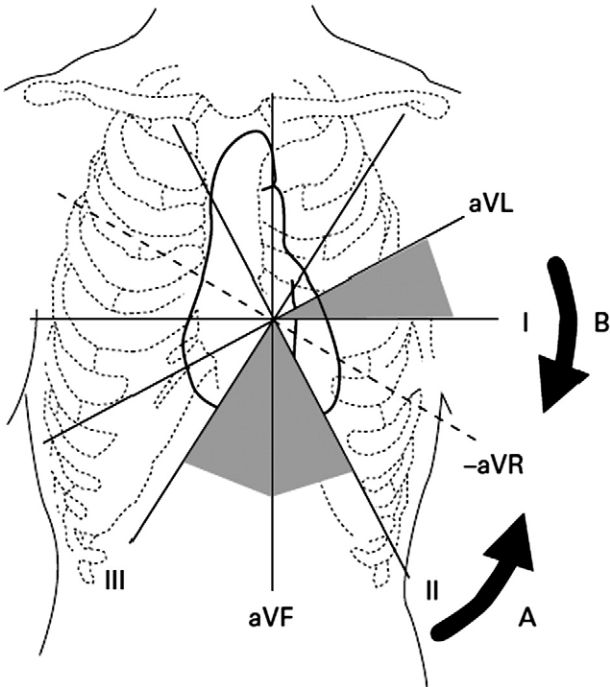


Fig. 1. Diagrammatic illustration of ST elevation in lead –aVR. Arrow A indicates more lateral involvement in patients with inferior ST elevation (shadow). Arrow B indicates more inferior involvement in patients with lateral limb lead ST elevation (shadow). Reproduced from *Heart* 2000;83:657 with permission from BMJ Publishing Group Ltd.

depression represents larger infarcts in the RCA group because the RCA must have a relatively large-sized posterolateral branch to supply the inferolateral wall in addition to the inferior wall. However, this finding of a large posterolateral branch being necessary to supply the inferolateral wall did not hold true for patients in the LCx group because the LCx itself provides blood supply to the inferolateral wall. Accordingly, aVR depression suggests the involvement of LCx or a large RCA with a large posterolateral branch. This finding is consistent with the recent observation that 60% of both dominant RCA and dominant LCx infarctions had aVR depression.¹⁴

Several studies have investigated the clinical implications of ST-segment depression in lead aVR in inferior wall STEMI in addition to differentiating which is the culprit artery. Menown and Adgey initially reported that aVR depression is associated with larger infarct size in inferior wall STEMI.¹⁰ Kosuge et al correlated aVR depression with impaired perfusion after primary percutaneous coronary intervention in inferior wall STEMI.¹³ In our study, we demonstrated that the infarct size was larger only in patients with aVR depression in RCA infarction but not in LCx infarction.

We found that 47% of LCx infarctions had a left dominant or co-dominant coronary artery system. Patients with LCx infarcts that present as STEMI generally have large LCx arteries, so as to involve the inferior wall. LCx infarcts can often present as non–ST-segment elevation myocardial infarction or can be electrocardiographically silent on the traditional 12-lead ECG, and only 48% of LCx infarctions presented as STEMI in one series.¹⁵ In the previous

literature, the ratio of RCA to LCx involvement in inferior STEMI varied from 2.4:1 to 7:1.^{1-5,12,13}

One of our study limitations includes its retrospective design. In addition, the size of infarction was estimated using peak CPKs measured every 6 hours. This is not necessarily the most accurate method to evaluate size of infarction, although it is a standard clinical practice to estimate extent of myocardial damage and infarct. In addition, in patients who did not have left ventriculograms performed during coronary angiogram, we used the transthoracic echocardiogram result to evaluate the left ventricular function. We acknowledge these two methods for measuring left ventricular function are not completely comparable. Lastly, our study lacked the evaluation of right-sided precordial leads (V4R), which may have further helped to identify the culprit artery in inferior STEMI.

Conclusion

In patients with inferior wall STEMI, ST-segment depression in aVR is more common in LCx infarcts than RCA infarcts. The finding of aVR depression with the RCA as the culprit artery is associated with larger infarct size involving a large posterolateral branch. aVR depression suggests the involvement of the inferolateral and apical walls, which are either supplied by the LCx artery or large-sized RCA with a large posterolateral branch.

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