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Original Contribution

New ST-segment algorithms to determine culprit artery location in acute inferior myocardial infarction☆

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ABSTRACT

Objectives: In acute inferior ST-segment elevation myocardial infarction (STEMI), multiple criteria have been proposed to predict the culprit artery based on the 12-lead electrocardiogram (ECG). We assessed the utilities of 11 traditional and 2 new criteria to devise a new ECG algorithm to localize the culprit artery in acute inferior STEMI.
Methods: We analyzed electrocardiographic and angiographic findings of 194 consecutive patients with acute inferior STEMI to devise a new ECG algorithm, further validated in another cohort of 80 patients with acute inferior STEMI.
Results: In derivation cohort, the 2 new criteria including (1) ST-segment depression in lead I equal to half of that in lead aVL and (2) equal ST-segment elevation in leads II, III, and aVF did not prove useful. The most powerful electrocardiographic criteria were (1) the ratio of ST elevation in lead III to that in lead II, (2) the ratio of ST depression in lead I to that in lead aVL, and (3) ST changes in lead I; these formed a 3-step algorithm. Application of this algorithm suggested the location of the culprit artery in 192 of 194 patients (nearly 99%) in the derivation cohort. In validation cohort, the algorithm possessed a sensitivity and specificity of 100% and 89%, respectively, for predicting the right coronary artery and 89% and 100%, respectively, for predicting the left circumflex artery.
Conclusions: A new 3-step algorithm based on 12-lead ECG is proposed to localize the culprit artery at the bedside of acute inferior STEMI patients before primary percutaneous coronary intervention, allowing immediate decisions about therapy.

1. Introduction

Acute inferior ST-segment elevation myocardial infarction (STEMI) is a fairly heterogeneous entity, which is usually caused by the occlusion of the right coronary artery (RCA); less often, the left circumflex artery (LCx) or, rarely, the left anterior descending artery (LAD) may be the cause [1]. The severity of inferior STEMI depends on the infarct-related artery and its size [2]. It can be limited to the posterobasal, diaphragmatic, or posterolateral segments; or it can involve 2 or 3 segments. Nearly 50% of patients with acute inferior STEMI have specific hemodynamic and bradycardic complications, usually due to the total occlusion of the proximal RCA [3,4]. It is important to identify high-risk subgroups of inferior myocardial infarction with increased mortality and morbidity [5–10].

The electrocardiogram (ECG) is an easily obtained, noninvasive method to diagnose acute myocardial infarction on admission. Timely identification of culprit coronary artery using ECG could provide clinically important information for early risk stratification to augment decision making and tailor reperfusion therapy. Previous studies [11–26] have proposed ECG criteria based on ST-segment "ups and downs" variation to identify the culprit coronary artery after acute inferior STEMI. The purpose of this study is to devise an effective algorithm to localize culprit coronary artery in acute inferior STEMI.

2. Materials and methods

2.1. Study population

In the present study, all patients with acute inferior STEMI admitted to our coronary care unit and treated with primary percutaneous coronary intervention (PCI) between January 1, 2010, and December 31, 2011, were assessed as derivation cohort; and patients admitted

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between January 1, 2012, and October 31, 2012, as validation cohort. Patients were included if they had been admitted within 12 hours from symptom onset, had sinus rhythm on admission ECG, and had the diagnosis of *first acute inferior myocardial infarction* defined as (1) chest pain or discomfort for more than 30 minutes, (2) ST elevation greater or equal to 1 mm in 2 or more inferior leads (II, III, or aVF), (3) transient increase in cardiac enzymes to more than 2-fold the normal laboratory value, and (4) the development of new Q waves. Patients with electrocardiographic evidence of right or left bundle-branch block, left ventricular hypertrophy, accelerated ventricular rhythm, paced rhythm or severe artifacts, history of previous myocardial infarction, or previous coronary artery bypass grafting or PCI were excluded from the study. The patient with the culprit artery of LAD verified by coronary angiography was also excluded (n = 1). Informed consent was given by all patients before their inclusion. The study protocol was approved by the clinical research Ethics Committee of the First Affiliated Hospital of Xi'an Jiaotong University. Finally, 194 patients comprised the derivation cohort, and 80 patients were in the validation cohort.

2.2. Electrocardiogram

The standard 12-lead ECG, recorded on admission of all patients using a paper speed of 25 mm/s and a standardization of 1 mV/10 mm, was analyzed by 2 investigators who were blinded to coronary angiography findings. Electrocardiograms were recorded using Cardiofax ECG-9130K or ECG-9020P machine (Nihon Kohden Corporation, Tokyo, Japan). Any disagreement between the investigators was resolved by consensus. The TP segment was used as the isoelectric line; the PR segment was used when the P wave and the T wave merged. The J point was determined for each lead independently. Both ST elevation and ST depression were measured at 80 ms after the J point in all leads. The utilities of 11 traditional electrocardiographic criteria previously reported in the prediction of RCA or LCx occlusion in acute inferior STEMI were evaluated in the derivation cohort listed as follows:

1. Lead I: ST-segment depression ≥0.5 mm; ST isoelectric; ST-segment elevation ≥0.5 mm [13,15].
2. Ratio of ST-segment elevation in lead III to that in lead II: >1, 1, <1 [13,26,27].
3. ∑ ST-segment depression in leads V1-V3 vs ∑ ST-segment elevation in leads II, III, and aVF: >1, ≤1 [14].
4. Lead V1: ST-segment depression; ST isoelectric; ST-segment elevation [23].
5. Lead aVR: ST-segment depression ≥1 mm; ST-segment elevation, ST isoelectric, ST-segment depression <1 mm [20].
6. Lead aVL: ST-segment depression; ST isoelectric; ST-segment elevation [15].
7. Ratio of ST-segment depression in lead V3 to ST-segment elevation in lead III: ≤1.2, >1.2 [19].
8. Ratio of ST-segment depression in lead I to that in lead aVL: ≤1, >1 [11,16].
9. ST-segment depression in lead aVR ≥ aVL [22].
10. ST-segment elevation in leads V5 or V6 ≥ 1 mm [1].
11. Maximum ST-segment depression in leads V1-V3 (ST-segment depression ≥1 mm in ≥1 precordial lead with the sum of ST-segment depression greater in leads V1 to V3 than in leads V4 to V6) [28].

In clinical practice, an equal ST-segment elevation in leads II, III, and aVF was often present in patients with acute inferior STEMI caused by occlusion of LCx, whereas ST-segment depression in lead I equal to half of that in lead aVL was observed in patients with occlusion of RCA. Therefore, the utility of the above 2 new electrocardiographic criteria unreported was also tested in the derivation population.

The performance of the new algorithm formed by the most powerful criteria was tested in the validation population.

2.3. Coronary angiography

The *culprit coronary lesion* was defined as the most severe lesion and/or the lesion with local dissection or thrombus. The films were interpreted by 2 experienced investigators without knowledge of the ECG findings. In case of a discrepancy, a third investigator reviewed the coronary angiography. Patients were classified into 2 groups according to the site of the culprit coronary lesion documented by coronary angiography as follows: group RCA and group LCx.

2.4. Statistics

SPSS version 19.0 (SPSS, Chicago, IL) was used for the statistical analysis. Data were expressed as mean ± SD for continuous variables and as number and percentage for categorical variables. For comparison of continuous variables, the analysis of variance was used. For comparison of categorical variables, the χ^2 test or the Fisher exact test was used. $P < .05$ was considered to be statistically significant.

3. Results

3.1. Baseline characteristics of the derivation cohort

The derivation population consisted of 194 patients (171 men and 23 women) with an age of 59 ± 11 years. On coronary angiography, the culprit artery was shown to be the RCA in 166 patients and the LCx in 28 patients. RCA was a dominant artery in 120 (62%) patients, dominant LCx in 12 (6%), and co-dominant circulation in 62 (32%) patients. There was a complete agreement in coronary angiographic readings between the 2 investigators. There were no significant differences between the RCA group and the LCx group concerning the baseline demographic and clinical characteristics (Table 1).

3.2. Electrocardiographic characteristics

The relationship between ECG criteria and culprit artery is presented in Tables 2 and 3. There was a complete agreement in ECG readings between the 2 investigators. The sensitivity, specificity, and positive and negative predictive values (PPV and NPV) of the traditional ECG criteria and 2 new ones to differentiate RCA occlusion from LCx occlusion in

Table 1
Baseline characteristics of the derivation cohort

	LCx group (n = 28)	RCA group (n = 166)	*P*
Female	5 (17.9%)	18 (10.8%)	.254
Age (y)	57.3 ± 11.7	58.9 ± 11.4	.908
Time from symptom onset to ECG (h)	6.5 ± 0.5	6.1 ± 0.2	.289
Time from onset of symptoms to ECG ≤ 6 h	20 (71.4%)	133 (80.1%)	.547
Time from onset of symptoms to CAG	7.0 ± 0.3	6.8 ± 0.4	.398
HR on admission (beat per min)	72.5 ± 14.1	70.7 ± 15.8	.977
SBP on admission (mm Hg)	124 ± 20.8	118 ± 24.2	.648
DBP on admission (mm Hg)	77.9 ± 14.1	74.3 ± 14.7	.790
Diabetes mellitus	5 (17.9%)	46 (27.7%)	.135
Hypertension	11 (39.3%)	83 (50.0%)	.111
Ischemic heart disease	4 (14.3%)	14 (8.4%)	.114
Family history	6 (21.4%)	36 (21.7%)	.959
Smoking	18 (64.3%)	115 (69.3%)	.123
Hyperlipidemia	2 (7.1%)	12 (7.2%)	.761
LVEF (%)	54.4 ± 8.2	57.7 ± 10.5	.826
Peak CK level (U/L)	2492.7 ± 1379.1	2805.8 ± 1904.1	.132
Peak CK-MB level (U/L)	237.5 ± 148.4	253.3 ± 168.1	.319
Killip class>1	5 (17.9%)	45 (27.1%)	.798
Multivessel disease	15 (53.6%)	78 (47.0%)	.996
In-hospital death	0	2 (1.2%)	.999

All continuous variables are presented as mean ± SD. All categorical variables are presented as number and percentage. CAG, coronary angiography; HR, heart rate; SBP, systolic blood pressure; DBP, diastolic blood pressure; LVEF, left ventricular ejection fraction; CK, creatine kinase; CK-MB, creatine kinase-MB.