

Electrocardiographic differentiation between occlusion of the first diagonal branch and occlusion of the left anterior descending coronary artery

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Abstract

Purpose: We sought to electrocardiographically distinguish ST-segment elevation (STE)-acute myocardial infarction (AMI) caused by occlusion of the first diagonal branch (D1) from STE-AMI caused by occlusion of the left anterior descending coronary artery (LAD).

Methods: We examined 28 patients with STE-AMI caused by D1 occlusion (G-D) and 342 with STE-AMI caused by LAD occlusion (G-L).

Results: G-D had a higher prevalence of STE ≥ 0.5 mm in each of leads I and aVL and a lower prevalence of STE ≥ 1 mm in each of leads V₁ through V₆ than G-L. The prevalence of STE ≥ 0.5 mm in lead aVL without STE ≥ 1 mm in lead V₁ was higher in G-D (82.1%) than in G-L (9.4%, $P < .01$).

Conclusion: ST-segment elevation ≥ 0.5 mm in lead aVL without STE ≥ 1 mm in lead V₁ may be useful to distinguish STE-AMI caused by occlusion of the D1 from STE-AMI caused by occlusion of the LAD.

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Keywords:

Electrocardiography; ST-segment elevation; Acute myocardial infarction; Diagonal branch; Left anterior descending coronary artery

Introduction

Acute occlusion of the left anterior descending coronary artery (LAD) and the first diagonal branch (D1) may produce ST-segment elevation (STE) in both the lateral and precordial leads. The infarct size would differ between the 2 occlusions, namely, the former would be generally expected to be associated with a larger infarct than the latter. Therefore, it is clinically important to electrocardiographically distinguish between the 2 occlusions. However, there are only a few studies investigating electrocardiographic features that distinguish between the 2 occlusions.^{1–3} Iwasaki et al¹ demonstrated that electrocardiographic abnormalities in leads I and aVL, including STE, abnormal Q waves, and negative T waves, combined with a normal finding in lead V₁ can distinguish acute occlusion of the D1

from that of the LAD. Their study included patients whose electrocardiograms were recorded within 24 hours of the onset of symptoms. The time elapsed from the onset of symptoms would affect ST-segment level in acute myocardial infarction (AMI). It is yet unclear whether there are any electrocardiographic differences in the hyperacute phase between STE-AMI caused by occlusion of the D1 and STE-AMI caused by occlusion of the LAD. Furthermore, differences in the electrocardiographic findings between acute occlusion of the D1 and that of the proximal LAD, mid-LAD, or distal LAD have not yet been fully investigated. Accordingly, this study sought to clarify these points.

Methods

Study population

Between January 1995 and December 2006, 28 patients (G-D) with STE-AMI caused by occlusion of the D1 and 342 patients (G-L) with STE-AMI caused by occlusion of the

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LAD were selected for this retrospective study from computer databases of patients with AMI at 4 hospitals (Oita University Hospital, Almeida Memorial Hospital, Koseiren Tsurumi Hospital, and Oita Nakamura Hospital) and a computer database of patients with AMI at 1 hospital (Almeida Memorial Hospital), respectively. The inclusion criteria were as follows: (1) hospital admission within 6 hours of the onset of chest pain, followed by emergency coronary angiography; (2) typical ischemic chest pain lasting 20 minutes or more; (3) STE ≥ 0.5 mm in leads I and/or aVL on the admission electrocardiogram in G-D and STE in at least 2 contiguous precordial leads with the following cut-off points on the admission electrocardiogram in G-L: ≥ 2 mm in men or ≥ 1.5 mm in women in leads V_2 through V_3 and/or ≥ 1 mm in other precordial leads; (4) an increase in the serum creatine kinase level of ≥ 2 times of the normal value; (5) no previous myocardial infarction (MI); (6) no electrocardiographic findings such as bundle-branch block, intraventricular conduction disturbance, or ventricular rhythm; (7) no other heart or lung disease affecting the electrocardiographic findings; and (8) the identification of an infarct-related lesion by emergency (within 60 minutes of hospital admission) coronary angiography. “The universal definition of MI”⁴ defines STE as new STE at the J point in 2 contiguous leads with the following cutoff points: ≥ 2 mm in men or ≥ 1.5 mm in women in leads V_2 through V_3 and/or ≥ 1 mm in other leads. However, in our preliminary investigation, STE ≥ 1 mm in lead aVL was observed in only 11 (40%) of 28 patients with AMI caused by occlusion of the D1, and 14 (50%) of the 28 patients did not met the universal definition of MI, suggesting that the definition may overlook some patients with AMI caused by occlusion of the D1. Therefore, we included patients with AMI caused by occlusion of the D1 whose admission electrocardiogram revealed STE ≥ 0.5 -mm in leads I and/or aVL. G-L was subdivided into the following 3 groups: patients with occlusion of the LAD proximal to the D1 (G-L1, $n = 166$), those with occlusion of

the LAD distal to the D1 and proximal to the second diagonal branch (D2) (G-L2, $n = 155$), and those with occlusion of the LAD distal to the D2 (G-L3, $n = 21$).

Standard 12-lead electrocardiography

The standard 12-lead electrocardiogram was recorded at a paper speed of 25 mm/s and a standardization of 10 mm = 1 mV. The magnitude of STE relative to the TP-segment was measured to the nearest 0.5 mm at the J point. We examined the prevalence of STE ≥ 0.5 mm in the limb leads and that of STE ≥ 1 mm in the precordial leads. The measurements were obtained by the consensus of 2 observers who were blinded to all clinical and angiographic data.

Emergency coronary angiography

Emergency coronary angiography was performed by a radial, brachial, or femoral approach. The coronary blood flow in the D1 or LAD was graded according to the classification used in the Thrombolysis in Myocardial Infarction trial.⁵ A multivessel disease was defined as a luminal diameter stenosis of more than 50% in at least 2 of the 3 coronary arteries. The grade of collateral filling to the D1 or LAD was determined by the classification of Rentrop et al.⁶ A collateral circulation with a grade of 2 or 3 was defined as “good.” The length of the LAD was evaluated using the post-recanalization left coronary angiogram recorded in the 30° right anterior oblique view, and a long LAD was defined as an LAD that wrapped around the apex and that perfused $\geq 25\%$ of the inferior wall of the left ventricle.^{7,8} The angiographic data were evaluated by the consensus of 2 observers who were blinded to all clinical and electrocardiographic data.

Statistical analysis

The data are expressed as mean \pm SD or number (%). Comparisons of categorical data between G-D and G-L or

Table 1
Patient characteristics

| | G-D n = 28 | G- L n = 342 | G- L1 n = 166 | G- L2 n = 155 | G- L3 n = 21 |
|-------------------------|---------------|-----------------|------------------|------------------|-----------------|
| Age (y) | 65 \pm 12 | 62 \pm 11 | 62 \pm 12 | 63 \pm 11 | 65 \pm 10 |
| Men | 23 (82%) | 257 (75%) | 129 (78%) | 113 (73%) | 15 (71%) |
| Time to admission (min) | 187 \pm 114 | 146 \pm 77 | 138 \pm 74 | 152 \pm 80 | 161 \pm 76 |
| Hypertension | 18 (64%) | 178 (52%) | 94 (56%) | 72 (46%) | 12 (57%) |
| Diabetes mellitus | 7 (25%) | 86 (25%) | 42 (25%) | 35 (21%) | 12 (57%) |
| Smoking history | 15 (54%) | 223 (65%) | 109 (66%) | 101 (65%) | 13 (62%) |
| Angiographic findings | | | | | |
| TIMI flow grade | | | | | |
| 0 | 22 (79%) | 250 (73%) | 119 (72%) | 114 (74%) | 17 (81%) |
| 1 | 4 (14%) | 29 (8%) | 17 (10%) | 11 (7%) | 1 (5%) |
| 2 | 2 (7%) | 41 (12%) | 22 (13%) | 17 (11%) | 2 (10%) |
| 3 | 0 (0%) | 22 (6%) | 8 (5%) | 13 (8%) | 1 (5%) |
| Good collaterals | 2 (7%) | 95 (27.8%)** | 57 (34.3%)* | 36 (23.2%) | 2 (7%) |
| Multivessel disease | 9 (32%) | 100 (29%) | 52 (31%) | 40 (26%) | 8 (38%) |
| Long LAD | 9 (32.1%) | 122 (35.7%) | 57 (34.3%) | 56 (36.1%) | 9 (42.9%) |

TIMI indicates Thrombolysis In Myocardial Infarction. Good collaterals were defined as Rentrop grade of 2 or 3.

* $P < .01$ vs G-D.

** $P < .05$ vs G-D.

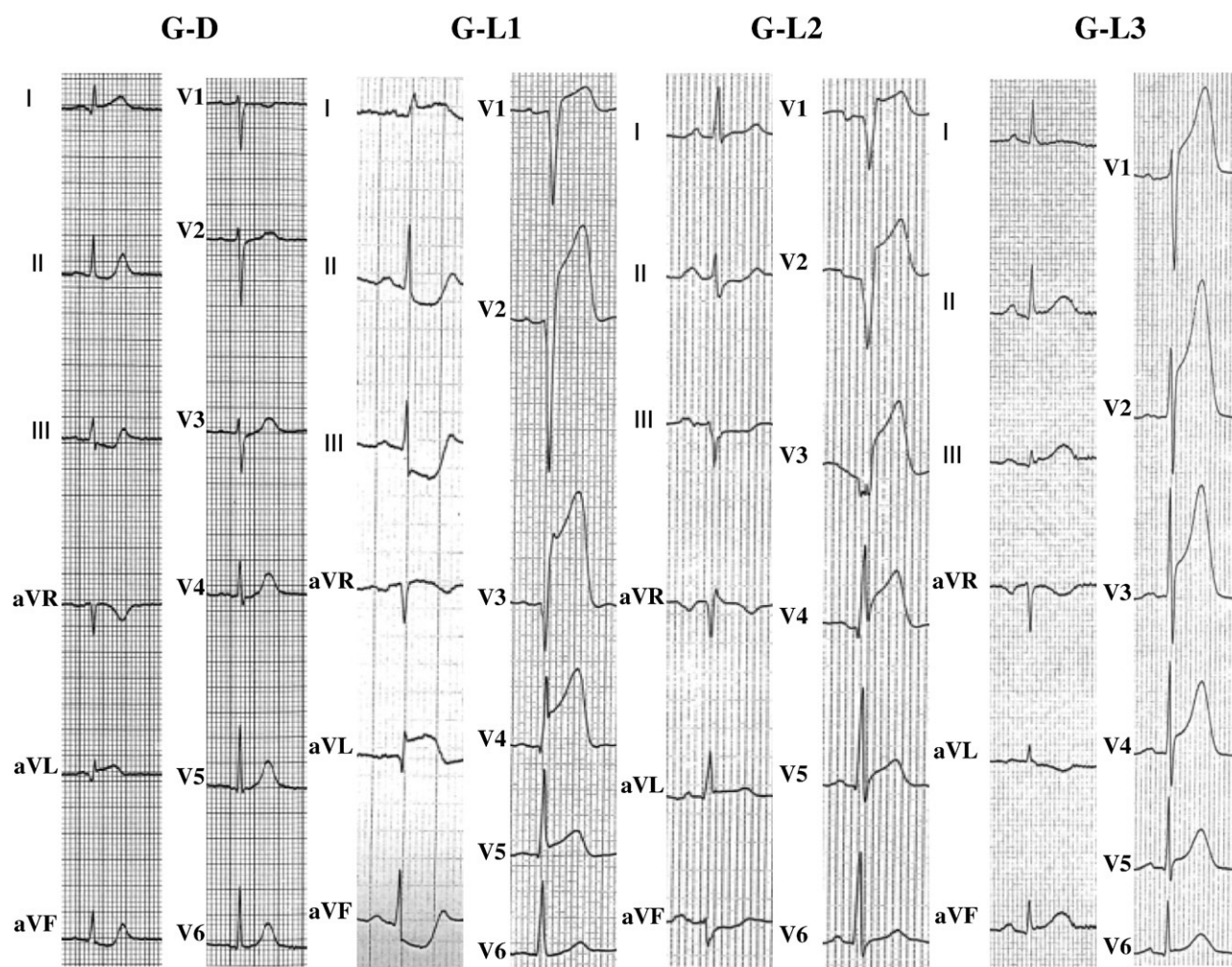


Fig. 1. Representative electrocardiograms in each group. G-D indicates patients with STE-AMI caused by occlusion of the first diagonal branch (D1); G-L, those with STE-AMI caused by occlusion of the LAD; G-L1, those with STE-AMI caused by occlusion of the LAD proximal to the D1; G-L2, those with STE-AMI caused by occlusion of the LAD distal to the D1 and proximal to the second diagonal branch (D2); G-L3, those with STE-AMI caused by occlusion of the LAD distal to the D2.

each subgroup were performed using the Fisher exact or χ^2 test. Comparisons of the continuous variables between G-D and G-L were performed using the unpaired t test.

Comparisons of the continuous variables among G-D and the 3 subgroups were performed using a 1-factor analysis of variance. A P value of less than .05 was considered to be

Table 2
Prevalences of STE ≥ 0.5 mm in the limb leads and STE ≥ 1 mm in the precordial leads

| Lead | G-D n = 28 | G- L n = 342 | G- L1 n = 166 | G- L2 n = 155 | G- L3 n = 21 |
|----------------|---------------|-----------------|------------------|------------------|-----------------|
| I | 22 (78.6%) | 178 (52.0%)* | 98 (59.0%)** | 73 (47.1%)* | 7 (33.3%)* |
| II | 2 (7.1%) | 33 (9.6%) | 7 (4.2%) | 18 (11.6%) | 8 (38.1%)** |
| III | 1 (3.6%) | 21 (6.1%) | 1 (0.6%) | 12 (7.7%) | 8 (38.1%)* |
| aVR | 6 (21.4%) | 110 (32.2%) | 68 (41.0%) | 39 (25.2%) | 3 (14.3%) |
| aVL | 28 (100%) | 239 (69.9%)* | 136 (81.9%)* | 93 (60.0%)* | 10 (47.6%)* |
| aVF | 2 (7.1%) | 24 (7.0%) | 3 (1.8%) | 13 (8.4%) | 8 (38.1%)** |
| V ₁ | 5 (17.8%) | 302 (88.3%)* | 146 (87.9%)* | 137 (88.3%)* | 19 (90.4%)* |
| V ₂ | 16 (57.1%) | 337 (98.5%)* | 163 (98.2%)* | 153 (98.7%)* | 21 (100%)* |
| V ₃ | 12 (42.9%) | 331 (96.8%)* | 158 (95.2%)* | 152 (98.1%)* | 21 (100%)* |
| V ₄ | 7 (25%) | 304 (88.9%)* | 144 (86.7%)* | 140 (90.3%)* | 20 (95.2%)* |
| V ₅ | 5 (17.8%) | 241 (70.5%)* | 111 (66.9%)* | 113 (72.9%)* | 20 (95.2%)* |
| V ₆ | 2 (7.1%) | 119 (34.8%)* | 55 (33.1%)* | 56 (36.1%)* | 8 (38.1%)** |

* $P < .01$ vs G-D.

** $P < .05$ vs G-D.

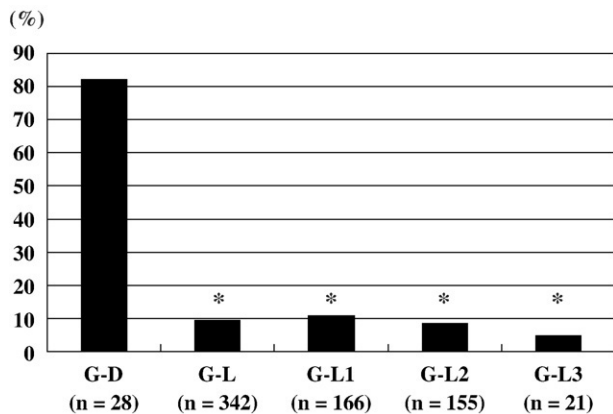


Fig. 2. The prevalence of STE ≥ 0.5 mm in lead aVL without STE ≥ 1 mm in lead V_1 . * $P < .01$ vs G-D.

statistically significant. All analyses were performed using the SPSS 12.0J software program for Windows (SPSS Inc, Tokyo, Japan).

Results

The patient characteristics are shown in Table 1. The prevalence of good collaterals to the infarct region was significantly lower in G-D than in G-L and G-L1.

A representative electrocardiogram in each group is shown in Fig. 1. The prevalence of STE of ≥ 0.5 or ≥ 1.0 mm in each lead is shown in Table 2. The prevalence of STE ≥ 0.5 mm in each of leads I and aVL was significantly higher in G-D than in G-L and 3 subgroups. The prevalence of STE ≥ 1.0 mm in each of leads V_1 through V_6 was significantly lower in G-D than in G-L and 3 subgroups. The prevalence of STE ≥ 0.5 mm in each of leads II, III, and aVF was significantly lower in G-D than in G-L3.

Fig. 2 shows the prevalence of STE ≥ 0.5 mm in lead aVL without STE ≥ 1 mm in lead V_1 . Its prevalence was significantly higher in G-D (82.1%; 95% confidence interval [CI], 64.4%–92.1%) than in G-L (9.4%; 95% CI, 6.3%–12.4%; $P < .01$), G-L1 (10.8%; 95% CI, 7.5%–14.1%; $P < .01$), G-L2 (8.4%; 95% CI, 5.5%–11.4%; $P < .01$), and G-L3 (4.8%; 95% CI, 2.4%–6.9%; $P < .01$). ST-segment elevation ≥ 0.5 mm in lead aVL without STE ≥ 1 mm in lead V_1 had a sensitivity of 82.1%, a specificity of 90.6%, a positive predictive value of 41.8%, and a negative predictive value of 98.4% for identifying G-D patients in this study population.

Discussion

The present study indicates that STE ≥ 0.5 mm in lead aVL without STE ≥ 1 mm in lead V_1 may be useful to distinguish STE-AMI caused by occlusion of the D1 from STE-AMI caused by occlusion of the LAD.

Lead aVL faces the basal portion of the lateral wall of the left ventricle. As the D1 supplies this area with the blood, acute occlusion of the D1 would be expected to produce STE in lead aVL. Iwasaki et al.¹ reported that STE ≥ 0.5 mm in

lead aVL was observed in all of 34 patients with acute (≤ 24 hours) occlusion of the D1. In the present study also, STE ≥ 0.5 mm in lead aVL was observed in all of 28 patients with STE-AMI (≤ 6 hours) caused by occlusion of the D1. Birnbaum et al.⁹ showed that STE of more than 1 mm in lead aVL is observed not only in STE-AMI caused by occlusion of the LAD proximal to the D1 but also in STE-AMI caused by occlusion of the LAD distal to the D1 or D2. In the present study, STE ≥ 0.5 mm in lead aVL was observed in 81.9% of G-L1, 60.0% of G-L2, and 47.6% of G-L3. These suggest that transmural ischemia in the territory of D2 may produce STE in lead aVL. Therefore, STE ≥ 0.5 mm in lead aVL alone is insufficient to distinguish STE-AMI caused by occlusion of the D1 from STE-AMI caused by occlusion of any site of the LAD.

Because STE in lead V_1 generally represents transmural ischemia in the basal part of the anterior interventricular septum and basal anterior wall of the left ventricle, STE in lead V_1 would be expected to be rarely observed in patients with STE-AMI caused by occlusion of the D1. Iwasaki et al.¹ found that the prevalence of STE ≥ 1 mm in lead V_1 was significantly lower in patients with acute occlusion of the D1 than in those with acute occlusion of the LAD. In the present study also, the prevalence of STE ≥ 1 mm in lead V_1 was extremely lower in STE-AMI caused by occlusion of the D1 than in STE-AMI caused by occlusion of the LAD. Of importance, the prevalence of STE ≥ 1 mm in lead V_1 was extremely higher (87.9%–90.4%) in each subgroup than in G-D. Accordingly, we hypothesized that STE ≥ 0.5 mm in lead aVL without STE ≥ 1 mm in lead V_1 may help to differentiate between the 2 occlusions and investigated the prevalence of this electrocardiographic finding in each group or subgroup. As a result, the finding was observed in 82.0% of patients with STE-AMI caused by occlusion of the D1, whereas it was observed only in about 10% of each group with STE-AMI caused by occlusion of the LAD, suggesting that the electrocardiographic finding is useful for distinguishing STE-AMI caused by occlusion of the D1 from STE-AMI caused by occlusion of any site of the LAD. The electrocardiographic finding had a high sensitivity and a high specificity for identifying STE-AMI caused by occlusion of the D1 in our study population, but a positive predictive value was low. This is because the number of patients with STE-AMI caused by occlusion of the D1 was extremely small compared to the number of patients with STE-AMI caused by occlusion of the LAD. Although Birnbaum et al.³ reported that STE ≥ 1 mm in leads aVL and V_2 without STE ≥ 1 mm in lead V_3 favors occlusion of the D1, this electrocardiographic finding was observed in only 3 (10.7%) of 28 patients with STE-AMI caused by occlusion of the D1 in the present study.

In the present study, the prevalence of STE ≥ 0.5 mm in each of the inferior leads was significantly higher in G-L3 than in G-D. However, its prevalence in G-L3 was only 38.1%. ST-segment elevation ≥ 0.5 mm in each of the inferior leads appears to be relatively specific in STE-AMI caused by distal LAD occlusion but insensitive to distinguish STE-AMI caused by occlusion of the D1 from STE-AMI caused by distal LAD occlusion.

In general, STE-AMI caused by occlusion of the D1 would be expected to be associated with a smaller infarct than STE-AMI caused by occlusion of the LAD. Indeed, in the present study, patients with STE-AMI caused by occlusion of the D1 ($n = 25$) had a lower peak creatine kinase level than those with STE-AMI caused by occlusion of the LAD ($n = 339$) (1053 ± 483 vs 3177 ± 2413 IU/L, $P < .001$). Patients with the latter, especially those with proximal LAD occlusion, would need a more aggressive approach to revascularization to prevent extensive myocardial damage resulting in pump failure, the possible development of sub-atrioventricular-nodal conduction disturbance, and the occurrence of life-threatening ventricular arrhythmias. Therefore, the present study demonstrating that STE ≥ 0.5 mm in lead aVL without STE ≥ 1 mm in lead V₁ on the admission electrocardiogram can distinguish between the 2 occlusions has an important clinical implication.

The present study has certain following limitations.

1. The number of patients with STE-AMI caused by occlusion of the D1 was very small. Because STE-AMI caused by occlusion of the D1 is generally very rare, multicenter studies with a large population are needed to confirm our results. In addition, the present study was retrospective. However, we have been prospectively collecting data of patients with AMI.
2. The present study included only patients with first STE-AMI. Therefore, the results of the present study may be unable to apply to patients with non-STE-AMI or previous MI.
3. We did not evaluate the size of the D1 and conal branch in the right coronary artery, which can affect electrocardiographic findings.^{10,11}
4. We examined the electrocardiogram on admission but not immediately before emergency coronary angiography. There may have been any changes in the coronary blood flow in the infarct-related artery until the starting time of emergency coronary angiography, which could affect electrocardiographic findings.
5. Various factors including time to admission, multi-vessel disease, collateral circulation to the infarct region, and the length of the LAD can affect ST-segment levels in AMI. In the present study, there were no significant differences in these factors except the prevalence of good collaterals to the infarct region between G-D1 and G-D or each subgroup.

In conclusion, the present study with a relatively small number of patients indicates that STE ≥ 0.5 mm in lead aVL without STE ≥ 1 mm in lead V₁ may be useful to distinguish STE-AMI caused by occlusion of the D1 from STE-AMI caused by occlusion of the LAD. Because the sensitivity and specificity of the electrocardiographic finding cannot be definitively obtained from such a small sample, evaluation of the sensitivity and specificity must be performed in a large population.

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