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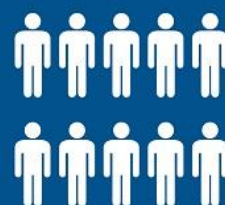
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Relationship Between Electrocardiographic Patterns and Angiographic Features in Isolated Left Circumflex Coronary Artery Disease

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Summary: The relation of electrocardiographic (ECG) patterns to clinical and angiographic features was assessed in 89 patients with isolated left circumflex coronary artery (LCx) disease (46 with and 43 without myocardial infarction). ECG abnormalities were present in 75 patients; there were isolated Q waves in 20, an abnormal R wave in lead V_1 with or without inferior and/or lateral Q waves in 21, and isolated ST-T wave changes in 34 cases. Inferior abnormalities on the electrocardiogram were similar in patients with proximal or distal stenoses of the LCx, but an abnormal R wave in lead V_1 correlated with proximal LCx stenosis ($p < 0.01$). Lateral abnormalities were more common in stenoses of the obtuse marginal branch and proximal LCx than in distal stenosis (all $p < 0.01$). Compared with patients without myocardial infarction with or without ST-T-wave changes and those with infarction without an abnormal R wave in lead V_1 , patients with LCx-related infarction and an abnormal R wave in lead V_1 associated with inferior and/or lateral Q waves had larger left ventricular end-diastolic and end-systolic volumes, lower ejection fraction, higher incidence of total occlusion of proximal LCx without collateral vessels, and more cardiac events during follow-up. This study suggests that an abnormal R wave in lead V_1 associated with lateral abnormalities on the standard electrocardiogram may be clinically useful in predicting proximal LCx stenosis and identifying a subset of postinfarction patients with left ventricular dysfunction due to a large infarct size.

Key words: left circumflex artery disease, angiography, electrocardiography

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Introduction

Isolated left circumflex coronary artery (LCx) disease is uncommon and occurs only in 2–3% of patients at angiography; the clinical and angiographic features for these patients, therefore, remain poorly characterized.^{1–3} Occlusion of the LCx may be associated with an electrocardiographic (ECG) pattern of inferior myocardial infarction (MI), similar to the feature of right coronary artery occlusion,⁴ and patients with LCx-related infarction have different myocardial damage.^{5,6} As the amount of myocardium in jeopardy is proportional to the sum of the amount of myocardium distal to each lesion in the coronary artery tree,⁷ and the effectiveness of interventional therapy appears to be more significant in larger than in smaller myocardial injuries independent of the site of infarction,⁸ it is pertinent to assess the relation of ECG patterns to the site of coronary stenosis and the status of left ventricular function in patients with isolated LCx disease with or without myocardial infarction.

Materials and Methods

Patients

The study population was selected retrospectively from a computerized information system and from a review of cardiac catheterization log between January 1980 and December 1988. Eighty-nine patients with significant isolated LCx stenosis ($\geq 50\%$ luminal narrowing) were included. Patients with isolated LCx disease who met the following criteria were excluded: (1) age > 70 years, (2) history of previous myocardial infarction, (3) evidence of valvular and pulmonary heart disease, cardiomyopathy, or noncardiac conditions involving a substantial increased risk of death, (4) thrombolytic therapy, percutaneous transluminal coronary angioplasty (PTCA) or coronary artery bypass grafting (CABG) during acute phase of infarction, and (5) bundle-branch block, Wolff-Parkinson-White syndrome, and right ventricular hypertrophy.

Clinical Features

Medical records of each patient were reviewed for age, sex, risk factors for coronary artery disease (hypertension,

cigarette smoking, serum cholesterol ≥ 250 mg/dl, diabetes mellitus, and family history of cardiovascular disease). The follow-up period was defined as commencing with the day of cardiac catheterization and ending with the most recent available date of follow-up or death. Information about death, myocardial infarction or reinfarction, angina, medical and interventional treatments, and New York Heart Association (NYHA) functional class $\geq$ II were obtained by having patients or their relatives or physicians complete a standard questionnaire.

ECG Evaluation and Exercise Testing

Electrocardiograms recorded within 24 hours of cardiac catheterization were interpreted according to the criteria of American Heart Association.⁷ Pathologic Q waves and ST-T-wave changes were classified according to their locations as (1) inferior when present in leads II, III, and aVF, (2) lateral when present in leads I and aVL with or without V_5 or V_6 , and (3) inferolateral when present in both inferior and lateral leads. True posterior infarction was defined by the presence of an abnormal R wave (duration ≥ 0.04 s and/or R to S ratio > 1) in lead V_1 .

Treadmill exercise stress testing was performed in 67 of 89 patients within a mean time of 2 weeks of angiography using the standard Bruce protocol. Exercise was discontinued if angina, ischemic ECG changes, or maximal leg fatigue occurred. A horizontal or downsloping ST-segment depression of ≥ 1 mm was considered positive for myocardial ischemia.

Cardiac Catheterization

This was performed within 5 months of myocardial infarction by a standard technique.⁹ Heart rate and left ventricular systolic and end-diastolic pressures were measured before ventriculography which was performed in 30° right anterior oblique projection. A 1 cm grid was filmed to correct magnification in each patient. Selective coronary arteriography was performed in multiple standard and angulated views for optimal visualization of the stenotic segments.

Left ventricular end-diastolic and end-systolic volumes were determined by the area-length method and corrected for overestimation using the Kennedy equation.¹⁰ Left ventricular ejection fraction was then calculated. Left ventricular regional wall motion abnormalities were assessed only in those patients who had left ventriculography in both right and left anterior oblique projections, as much of the abnormal wall motion cannot be visualized in a single right anterior oblique view for patients with isolated LCx disease.¹¹

The most severe narrowing of the LCx was recorded, and the location was classified as (1) proximal when present between its origin and the takeoff of the major obtuse marginal branch, (2) obtuse marginal, and (3) distal when present after the takeoff of the obtuse marginal branch. The LCx was defined as subtotally occluded if prompt or complete antegrade filling of the distal segment was

demonstrated during selective coronary injection. Vessels showing no antegrade flow beyond the point of occlusion were recorded as totally occluded, as were those with perceptibly slow antegrade flow and only minimal or incomplete filling of the vessel past the obstruction. Good collateral filling was defined as identifiable collateral vessels leading to near normal opacification and rapid washout of the distal segment of the LCx.

The pattern of right and left coronary dominance was assessed. The left coronary dominance was recognized when posterior descending branch arose from the LCx or in the presence of total occlusion of the LCx without collaterals, and it was defined based on the absence of posterior descending branch from the right coronary artery.

Statistical Analysis

Continuous data are expressed as mean $\pm$ SD and comparison among groups was made by analysis of variance. Correlated proportions for unrelated and related samples were analyzed with Yates corrected chi-square test and McNemar test, respectively. A probability (p) of < 0.05 was considered significant.¹²

Results

Clinical and ECG Features

The mean age of 89 patients with isolated LCx disease was 53 ± 10 years (range 32–70). There were 75 men and 14 women. A total of 66 patients had angina pectoris, and 46 had suffered a myocardial infarction. All but two patients had one or more risk factors. Hypertension was present in 48%, 70% smoked cigarettes, 40% had hypercholesterolemia, 13% had diabetes mellitus, and 19% had a family history of cardiovascular disease. The number of risk factors was 2.4 per patient.

Isolated inferior and/or lateral Q waves occurred in 20 patients, 80% involving inferior leads. Isolated ST-T-wave changes were present in 34 patients and an abnormal R wave in lead V_1 developed in 21 patients, 20 of whom had associated Q waves in inferior (5 patients) or lateral (2 patients) leads or both (13 patients) (Table I).

Forty-six patients had a positive exercise stress test (16 with and 30 without MI), 43 of whom had ST-segment depression in inferior and/or lateral leads and 3 of whom had ST-segment depression in anterior or anteroapical leads.

Relation Between ECG Patterns and Coronary Lesions

The relationship between ECG patterns and the site of coronary lesions was assessed in 75 patients who had an abnormal electrocardiogram (Table II). The occurrence of ECG abnormalities in inferior leads was similar in patients with proximal or distal LCx stenoses. The ECG abnormalities in lateral leads was present in 73% and 68% of patients with stenosis in the obtuse marginal branch or proximal segment of the LCx ($p < 0.01$ vs. distal lesions). An

TABLE I Electrocardiographic abnormalities in isolated left circumflex artery disease

	Total	ECG leads		
		Inferior	Lateral	Inferolateral
Q waves	20	16	2	2
Isolated ST-T-wave changes	34	11	12	11
Abnormal R wave in lead V ₁ + ST-T wave changes	1			
Abnormal R wave in lead V ₁ + Q waves	20	5	2	13
Normal	14			

abnormal R wave in lead V₁ was most common in patients with proximal LCx stenosis.

The incidence of total LCx occlusion without collateral vessels was significantly higher in patients with infarction and an abnormal R wave in lead V₁. Of 89 patients, 34 had a left coronary dominance pattern (17 with and 17 without MI). In 17 patients with infarction and left coronary dominance, 9 of the 11 patients who had an abnormal R wave in lead V₁ developed total occlusion of proximal LCx without collaterals; in contrast, all remaining 6 patients without an abnormal R wave in lead V₁ had residual flow to the infarct region (5 subtotal occlusion and 1 total occlusion with collaterals) ($p < 0.01$).

Hemodynamics and Left Ventricular Function

Hemodynamic findings and left ventricular volumes and ejection fraction were compared among patients with normal electrocardiograms, those with isolated ST-T-wave changes without infarction, and those with infarction with or without an abnormal R wave in lead V₁ (Table III). Heart rate, systolic blood pressure, left ventricular volumes, and ejection fraction were normal in patients with isolated ST-T-wave changes without infarction. Left ventricular volumes were larger and ejection fraction was lower in patients with infarction and an abnormal R wave in lead V₁ than in those with infarction without an abnormal R wave in lead V₁. A

normal ejection fraction (≥ 0.50) was present in all patients with normal electrocardiograms, in 30 patients (88%) with isolated ST-T-wave changes without infarction, in 16 patients (80%) with infarction without an abnormal R wave in lead V₁, and in only 7 patients (33%) with infarction and an abnormal R wave in lead V₁ ($p < 0.01$). Left ventriculography was performed in both right and left anterior oblique projections for 16 patients, 6 with isolated inferior (2 patients) or lateral (4 patients) Q waves and 10 with an abnormal R wave in lead V₁ associated with inferior (2 patients) or lateral (2 patients) Q waves or both (6 patients). Thirteen patients (81%) had abnormal lateral or posterolateral segmental wall motion.

Follow-Up

The mean follow-up period was 49 months. Three patients with infarction and an abnormal R wave in lead V₁ died suddenly within 2 years. Three patients suffered myocardial infarction (1 patient with an abnormal R wave in lead V₁ had reinfarction and 2 with isolated ST-T-wave changes had a first infarction). Sixteen patients were in NYHA functional class $\geq$ II, including 3 with isolated ST-T-wave changes and 13 with myocardial infarction (10 with and 3 without an abnormal R wave in lead V₁). Therefore, the cardiac event rate was significantly higher in patients with infarction and an abnormal R wave in lead V₁ than in those without an abnormal R wave in lead V₁ with or without infarction ($p < 0.01$).

All patients had medical treatments. Nitrates were used in 42%, while 46% were taking beta blockers, and 42% were on calcium antagonists. Eleven patients underwent percutaneous transluminal coronary angioplasty, and one had coronary artery bypass grafting. The remaining 74 patients were either angina free (58 patients) or had stable exertional angina (16 patients) under medical therapy.

Discussion

Relation Between ECG Patterns and LCx Stenosis

Our results have shown ECG abnormalities in inferior leads were present in a majority of patients with proximal or distal LCx stenoses, suggesting that changes in inferior

TABLE II Relation between electrocardiographic patterns and site of circumflex artery stenosis

	No. of patients	ECG patterns		
		Inferior	Lateral	Abnormal R wave in lead V ₁
Proximal	22	17 (77%)	15 (68%) ^a	10 (45%) ^a
Obtuse marginal	22	12 (55%) ^a	16 (73%) ^a	7 (32%)
Distal	31	28 (90%)	9 (29%)	4 (13%)

^a $p < 0.01$ vs. distal stenosis.

TABLE III Hemodynamic and angiographic data

	Normal ECG (n = 14)	ST-T-wave changes without infarction (n = 29)	Infarction without abnormal R wave in lead V ₁ (n = 25)	Infarction with abnormal R wave in lead V ₁ (n = 21)
Heart rate (beats/min)	71 ± 15	75 ± 15	72 ± 11	73 ± 15
Systolic blood pressure (mmHg)	133 ± 19	135 ± 22	136 ± 23	131 ± 26
LV End-diastolic pressure (mmHg)	12 ± 4	13 ± 6	13 ± 4	13 ± 4
End-diastolic volume (ml)	128 ± 18	128 ± 40	132 ± 38	149 ± 26 ^{a-c}
End-systolic volume (ml)	43 ± 9	46 ± 25	57 ± 18 ^{b, c}	78 ± 18 ^{a-c}
Ejection fraction	0.66 ± 0.08	0.64 ± 0.11	0.59 ± 0.06 ^{b, c}	0.47 ± 0.08 ^{a-c}
LCx Total occlusion				
without collaterals	0	1	1	14 ^{a-c}
Subtotal occlusion	14	28	14	7 ^{a, b}
Total occlusion				
with collateral	—	—	6	0 ^d

^ap < 0.01 vs. normal ECG.^bp < 0.01 vs. ST-T-wave changes.^cp < 0.01.^dp < 0.05 vs. infarction without abnormal R wave in lead V₁.

leads may not differentiate proximal and distal lesions of the LCx. Dunn *et al.* reported a higher prevalence of inferior pattern in patients with peripheral obstruction than in those with central lesions of the LCx.¹ However, in their study, patients with proximal LCx stenosis and those with lesions of the obtuse marginal branch were grouped together as a subset of central obstruction. From the data they provided, we also did not find significant differences in ECG abnormalities in inferior leads between patients with proximal and those with distal stenoses of the LCx.

The ECG abnormalities in lateral leads were specifically associated with a stenosis in the obtuse marginal branch of the LCx. This is predictable because the obtuse marginal branch usually supplies the posterolateral myocardium.¹³ Previous exercise thallium scintigraphic studies have shown that patients with stenosis of the obtuse marginal branch of the LCx often had defects in the lateral segment of the left ventricular wall.^{13, 14} Braat *et al.* have shown that the recording of lead V_{4R} in the acute phase of an inferior myocardial infarction may be useful in predicting coronary artery occlusion. Patients with ST-segment elevation ≥ 1 mm in lead V_{4R} often had a proximal occlusion of the right coronary artery and those who had no ST-segment elevation in lead V_{4R} with an upsloping ST-segment developed a distal occluded right coronary artery. A downsloping ST segment in lead V_{4R} or ST-segment elevation ≥ 1 mm in leads aVL, V₅, and V₆ predicted a circumflex artery occlusion.¹⁵

The incidence of an abnormal R wave in lead V₁ in our patients with isolated LCx disease was similar to that previously reported,^{1, 6} and it occurred more frequently in patients with proximal stenosis than in those with distal lesion of the LCx. An abnormal R wave in lead V₁ was often associated with inferior and/or lateral Q waves, reflecting the fact that the proximal LCx supplies both posterolateral and

posteroinferior myocardium, and a single occlusion in such a vessel could readily cause simultaneous infarction of the lateral and inferior walls. Our study directly supports the concept that a "true posterior," "inferolateral," or "posterolateral" infarction is most likely the result of proximal LCx occlusion,^{6, 16} and further substantiates previous findings that disease in the proximal segment of the LCx was associated with thallium defects in the lateral, inferior, and/or posterior segments.¹ Brembilla-Parrot *et al.* found that in some patients a tall R wave in lead V₁ during posterior myocardial infarction may also be caused by conduction disturbance in the His-Purkinje system rather than just mirroring loss of activation forces at the posterolateral region.¹⁷

Correlation Between ECG and Left Ventricular Function

Our results show that in patients with isolated LCx disease status of left ventricular function differs too greatly for them to be considered as a homogeneous patient population. Isolated LCx stenosis without myocardial infarction often does not cause left ventricular dysfunction, as the mean left ventricular ejection fraction was greater than 0.60. Patients with LCx-related infarction who had abnormal R waves in lead V₁ associated with inferior and/or lateral Q waves exhibited larger left ventricular volumes and lower ejection fraction than those with LCx-related infarction who had isolated inferior or lateral Q waves or ST-T-wave changes. These findings are supported by the study of Movahed and Becker,⁵ who reported that 51% of their patients with scintigraphically localized lateral infarction who had an abnormal R wave in lead V₁ appeared to have a large transmural myocardial infarction. Likewise, Bough *et al.*, using radionuclide ventriculography, observed that patients with an abnormal R wave in lead V₁ and inferior Q waves had severe wall motion abnormalities involving

basal, inferior, and lateral regions of the left ventricular wall. They suggested that a prominent right precordial R wave may be an ECG hallmark of inferior and lateral wall infarctions that have extended into the basal lateral left ventricular segments.^{6, 18}

In this study, the majority of patients with LCx-related infarction and an abnormal R wave in lead V₁ had total occlusion of proximal LCx without collateral vessels, with almost half of these patients having left coronary dominance. These findings further substantiate our previous reports on the importance of residual flow to the infarct region in the preservation of left ventricular function after myocardial infarction.¹⁹ Our results also indirectly confirm the fact that the size of LCx circulation and the location of coronary artery occlusion are crucial to the infarct size and explain in part the ECG changes in some patients with infarct extension.^{1, 5, 6}

Conclusions

Our data obtained on a selected group of patients show that proximal LCx stenosis may be suspected by evidence of lateral abnormalities and an abnormal R wave in lead V₁ on the standard electrocardiograms, whereas abnormal changes in inferior leads are not of discriminating value between proximal and distal LCx stenoses. Patients with LCx-related infarction and an abnormal R wave in lead V₁ associated with inferior and/or lateral Q waves may have proximal LCx occlusion with poor collateral vessels. In these patients, left ventricular function is reduced and the incidence of cardiac events is high during late follow-up, probably related to a large infarct size. Previous studies of coronary anatomy in patients with posterior myocardial infarction suggest that presence of an abnormal R wave in lead V₁ in inferior myocardial infarction may be predictive of multivessel disease.⁶ Therefore, the simple observation of an abnormal R wave in lead V₁ associated with inferior and/or lateral Q waves may provide useful information regarding identification of a subset of patients at increased risk for subsequent cardiac events.

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