

Prediction of Acute Left Main Coronary Artery Obstruction by 12-Lead Electrocardiography

ST Segment Elevation in Lead aVR With Less ST Segment Elevation in Lead V₁

Hirosuke Yamaji, MD,*‡ Kohichiro Iwasaki, MD,‡ Shozo Kusachi, MD,*† Takashi Murakami, MD,* Ryouichi Hirami, MD,* Hiromi Hamamoto, MD,* Kazuyoshi Hina, MD,‡ Toshimasa Kita, MD,‡ Noburu Sakakibara, MD,‡ Takao Tsuji, MD*

Okayama, Japan

OBJECTIVES	We sought to determine the electrocardiographic (ECG) features associated with acute left main coronary artery (LMCA) obstruction.
BACKGROUND	Prediction of LMCA obstruction is important with regard to selecting the appropriate treatment strategy, because acute LMCA obstruction usually causes severe hemodynamic deterioration, resulting in a less favorable prognosis.
METHODS	We studied the admission 12-lead ECGs in 16 consecutive patients with acute LMCA obstruction (LMCA group), 46 patients with acute left anterior descending coronary artery (LAD) obstruction (LAD group) and 24 patients with acute right coronary artery (RCA) obstruction (RCA group).
RESULTS	Lead aVR ST segment elevation (>0.05 mV) occurred with a significantly higher incidence in the LMCA group (88% [14/16]) than in the LAD (43% [20/46]) or RCA (8% [2/24]) groups. Lead aVR ST segment elevation was significantly higher in the LMCA group (0.16 ± 0.13 mV) than in the LAD group (0.04 ± 0.10 mV). Lead V ₁ ST segment elevation was lower in the LMCA group (0.00 ± 0.21 mV) than in the LAD group (0.14 ± 0.11 mV). The finding of lead aVR ST segment elevation greater than or equal to lead V ₁ ST segment elevation distinguished the LMCA group from the LAD group, with 81% sensitivity, 80% specificity and 81% accuracy. A ST segment shift in lead aVR and the inferior leads distinguished the LMCA group from the RCA group. In acute LMCA obstruction, death occurred more frequently in patients with higher ST segment elevation in lead aVR than in those with less severe elevation.
CONCLUSIONS	Lead aVR ST segment elevation with less ST segment elevation in lead V ₁ is an important predictor of acute LMCA obstruction. In acute LMCA obstruction, lead aVR ST segment elevation also contributes to predicting a patient's clinical outcome. (J Am Coll Cardiol 2001; 38:1348–54) © 2001 by the American College of Cardiology

Acute obstruction of the left main coronary artery (LMCA) is not frequently encountered (1). A large part of the myocardium of the left ventricle is perfused by the LMCA, and its acute obstruction thus causes severe hemodynamic deterioration, frequently resulting in rapid fatality (2,3). Prediction of acute LMCA obstruction is important with regard to selecting the appropriate treatment strategy and estimating the prognosis.

See page 1355

Recently, Engelen et al. (4) reported that lead aVR ST segment elevation was observed in acute obstruction of the left anterior descending coronary artery (LAD) proximal to the major septal branch, but not in acute LAD obstruction distal to the major septal branch. They concluded that lead

aVR ST segment elevation associated with proximal LAD obstruction was caused by transmural ischemia of the basal part of the septum. This led us to assume that acute LMCA obstruction also causes lead aVR ST segment elevation through disturbance of major septal branch blood flow—that is, interruption of LAD blood flow. In fact, lead aVR ST segment elevation during angina pectoris attacks has been reported in patients with significant LMCA stenosis (5). The right coronary artery (RCA) also perfuses the septum through its septal perforator branches, to some extent, and acute RCA obstruction may possibly cause lead aVR ST segment elevation in some cases.

Both acute LMCA and LAD obstruction generally produce anterior wall ischemia, resulting in ST segment elevation in the precordial leads. One can easily assume that LMCA, but not LAD, obstruction is ordinarily associated with posterior wall ischemia. Acute LMCA obstruction may show ST segment shifts in the precordial leads different from those found in acute LAD obstruction, due to concomitant posterior wall ischemia, which produces reciprocal changes in the precordial leads.

Based on these reported findings, we hypothesized that

From the *Department of Internal Medicine I, Faculty of Medicine, and †Department of Medical Technology, Faculty of Health Sciences, Okayama University Medical School, Okayama; and ‡Cardiovascular Center, Sakakibara Hospital, Okayama, Japan.

Manuscript received October 5, 2000; revised manuscript received June 6, 2001, accepted July 19, 2001.

Abbreviations and Acronyms

CK	=	creatinine kinase
ECG	=	electrocardiogram
LAD	=	left anterior descending coronary artery
LCx	=	left circumflex coronary artery
LMCA	=	left main coronary artery
MI	=	myocardial infarction
RCA	=	right coronary artery

the ST segment in lead aVR would be elevated in acute LMCA obstruction, and that the relationship between the ST segment elevation in lead aVR and that in the precordial leads would differ between acute LMCA and LAD obstruction. Accordingly, in the present descriptive study, we retrospectively compared the findings on the 12-lead electrocardiograms (ECGs) of patients with acute LMCA obstruction with those of patients with acute LAD and RCA obstruction, by using simple and multivariate analyses, focusing on the findings of ST segment shifts in leads aVR and V₁. We also examined the relationship between the ST segment shift in lead aVR and patients' clinical outcomes in those with acute LMCA obstruction.

METHODS

Patients. From January 1988 to January 1998, 16 patients (14 men and 2 women; mean [\pm SD] age 65 ± 9 years [range 37 to 78]) were admitted to the hospital with acute LMCA obstruction (LMCA group). The 12-lead ECG findings in these patients were compared with those in 46 consecutive patients with the culprit lesion at location segment no. 6 (LAD group) and in 24 patients with the culprit lesion at location segment no. 1, according to the American Heart Association classification. All patients were admitted between December 1994 and January 1998. Patients with significant stenosis ($>75\%$ lumen diameter stenosis), other than that in the culprit lesion, were excluded from the LAD and RCA groups. Patients with acute left circumflex coronary artery (LCx) obstruction was excluded from the present study. The reasons for the exclusion were: 1) characteristic or diagnostic ST segment elevation in the precordial leads or lead aVR was not reported (6); and 2) LCx obstruction was anatomically unable to cause ischemia at the basal part of the septum, which has been thought to elevate the ST segment in lead aVR, as described earlier. Patients with a recurrent myocardial infarction (MI) were excluded from the analysis. The LAD group consisted of 41 men and five women whose ages ranged from 39 to 80 years (61 ± 9 years). The RCA group consisted of 18 men and six women whose ages ranged from 40 to 80 years (61 ± 10 years). There were no significant differences in gender or age among the three groups. All patients in the three groups were admitted within 12 h after the onset of the acute MI. The culprit lesion was determined by emergency coronary angiography and confirmed later by two cardiologists who were not aware of any ECG findings. The culprit lesion was

defined when the lesion was totally occluded or showed severe stenosis. When the lesion showed severe stenosis, the lesion with flow delay (Thrombolysis in Myocardial Infarction [TIMI] flow grade 1), with angiographic findings that suggested local dissection or thrombus, was defined as the culprit lesion (7). Diagnosis of acute MI was established by ST segment elevation, defined subsequently, in more than two leads, associated with typical, severe anterior chest pain and confirmed by elevation of both serum creatine kinase (CK) and its MB isoenzyme (CK-MB) greater than two times the normal upper limit during the patient's clinical course. The upper limits of the range (2 SD above the mean value) for the healthy age- and gender-matched control subjects for serum CK and CK-MB were 180 IU/l and 19 IU/l, respectively. The clinical and angiographic characteristics of the patients in the LMCA group are summarized in Table 1. In three patients in the LMCA group, serum levels of CK and CK-MB were not elevated more than two times the normal upper limit, as these patients died early.

Electrocardiography. The 12-lead ECGs recorded on admission before emergency coronary angiography were analyzed. A ST segment shift was determined as the mean value of five successive beats measured at 60 ms after the J point of the QRS complex. ST segment elevation was defined as present when ST segment elevation was >0.05 mV in the limb leads and ST segment elevation was >0.1 mV in the precordial leads. The data for ST segment shifts were subjected to statistical analysis.

Initially, inter-observer and intra-observer differences were checked by using 10 randomly selected ECG samples from the three groups. Measurements were then performed by two observers who were not aware of any angiographic findings.

Analysis. In emergency coronary angiography, collateral circulation was classed into four grades according to the grading system of Rentrop et al. (8). First, the incidence of ST segment elevation was examined in all leads, including aVR, and compared among the three groups. Further, ST segment elevation in lead aVR and the precordial leads was compared between the LMCA and LAD groups, and the differences in ST segment elevation between leads aVR and V₁ were examined. For comparison between the LMCA and RCA groups, a comparison was made for the inferior leads in addition to lead aVR.

Statistics. Data are expressed as the mean value \pm SD. For univariate analysis, the Fisher exact probability test was used to compare the prevalence of ST segment elevation between two groups. The unpaired Student *t* test was used to compare the extent of ST segment shift between two groups. We also performed stepwise linear multivariate discriminant analysis using a personal computer (Compaq, Prolinea 5120, Houston, Texas) with the appropriate software. In this analysis, the dependent variables were those of the two groups being compared. Because leads II, III and aVF (i.e., the inferior leads) showed essentially the same prevalence of abnormalities on univariate analysis, lead aVF

Table 1. Characteristics of Patients With Acute Left Main Coronary Artery Occlusion

Patient No.	Age (years)	Gender	Duration From Onset to ECG Recording (min)	Maximal CK (IU/ml)	BP on Admission (mm Hg)	HR on Admission (beats/min)	Other Lesions	Collateral Channels*	Dominant Artery	Therapy	Patient Outcome
1	61	M	100	173	50	90	None	G-0	Rt.	ICT/PTCA	Dead
2	70	M	155	4,505	70	112	LAD no. 6, 100%	G-3	Rt.	PTCA	Alive
3	55	M	210	10,636	76	116	None	G-0	Rt.	ICT	Alive
4	63	M	180	161	40	120	Unknown	Unknown	Unknown	PTCA	Dead
5	62	M	90	543	104	130	RCA no. 3, 100%	G-0	Rt.	ICT/emergency CABG	Alive
6	67	M	210	5,369	80	130	RCA, 50%	G-3	Rt.	ICT	Dead
7	62	M	250	3,106	60	54	None	G-3	Rt.	ICT/PTCA/emergency CABG	Alive
8	78	M	70	74	82	84	None	G-1	Rt.	ICT/PTCA	Dead
9	65	M	40	2,327	150	140	None	G-1	Rt.	IVT	Dead
10	69	M	120	3,801	110	104	None	G-3	Rt.	IVT/emergency CABG	Alive
11	70	M	160	12,292	94	68	None	G-2	Rt.	ICT	Dead
12	70	F	170	4,931	96	74	LAD no. 7, 75%	G-1	Rt.	ICT/PTCA	Dead
13	74	M	425	4,991	126	126	None	G-2	Rt.	ICT/PTCA	Alive
14	65	M	690	1,025	110	56	None	G-3	Rt.	ICT/PTCA	Alive
15	74	F	150	7,803	60	140	None	G-1	Rt.	ICT/PTCA	Dead
16	37	M	60	7,296	90	92	None	G-2	Rt.	ICT/PTCA	Alive

*The grade of collateral channels was classed according to the grading system of Rentrop et al. (8).

BP = blood pressure; CABG = coronary artery bypass grafting; CK = creatine kinase; ECG = electrocardiogram; F = female; G = grade; HR = heart rate; ICT = intracoronary thrombolysis; IVT = intravenous thrombolysis; LAD = left anterior descending coronary artery; PTCA = percutaneous coronary angioplasty; RCA = right coronary artery; Rt. = right.

was selected for the stepwise linear multivariate discriminant analysis to avoid multi-collinearity of the variables. For the same reasons, leads V_1 and V_6 were selected among the precordial leads, and lead aVL was selected from the lateral leads. As a result, the independent variables used in this study were the ST segment shifts in leads aVR, aVL, aVF, V_1 and V_6 . A p value <0.05 was considered significant.

RESULTS

When evaluating the sample ECGs, the inter-observer and intra-observer differences in the limb leads averaged 0.01 ± 0.02 mV and 0.01 ± 0.03 mV, respectively. Similarly, the inter-observer and intra-observer differences in the precordial leads averaged 0.01 ± 0.04 mV and 0.00 ± 0.02 mV, respectively. Therefore, intra-observer and inter-observer variations were acceptably small and did not affect the validity of the results.

Incidences of ST segment shift among the LMCA, LAD and RCA groups. Figure 1 shows representative 12-lead ECGs at hospital admission for one patient from each group, and the incidence of ST segment shift (>0.05 mV in the limb leads and >0.1 mV in the precordial leads) on the 12-lead ECG is summarized in Figure 2. Lead aVR showed ST segment elevation in 88% (14/16) of patients in the LMCA group, whereas ST segment elevation was found in 43% (20/46) of patients in the LAD group and only 8% (2/24) of patients in the RCA group. Lead aVF, an inferior lead, clearly showed a higher incidence of ST segment elevation (96% [23/24]) in the RCA group than in the LMCA group (0% [0/16]) or in the LAD group (9% [4/46]). Similar incidences were seen in the other inferior leads (i.e., leads II and III).

Comparison between the LMCA and LAD groups. UNIVARIATE ANALYSIS. Table 2 summarizes the results of a comparison of ST segment elevation in leads aVR and V_1 between the LMCA and LAD groups. Significantly higher ST segment elevation in lead aVR was observed in the LMCA group than in the LAD group. Conversely, ST segment elevation in lead V_1 was significantly lower in the LMCA group than in the LAD group. Lead V_2 showed similar results (LMCA group vs. LAD group: 0.15 ± 0.27 vs. 0.34 ± 0.21 mV; $p < 0.01$). Lead V_3 tended to show similar results (0.27 ± 0.40 vs. 0.44 ± 0.35 mV; $p > 0.05$ and < 0.10).

As a consequence, 13 patients (81%) in the LMCA group and only 9 patients (20%) in the LAD group showed greater or equal ST segment elevation in lead aVR compared with that in lead V_1 . The finding that ST segment elevation in lead aVR was greater than or equal to that in lead V_1 distinguished the LMCA group from the LAD group, with 81% sensitivity, 80% specificity and 81% accuracy.

MULTIVARIATE ANALYSIS. Stepwise linear multivariate discriminant analysis identified leads aVR and V_1 as leads in which ST segment elevation significantly contributed, positively and negatively, respectively, to distinguishing the

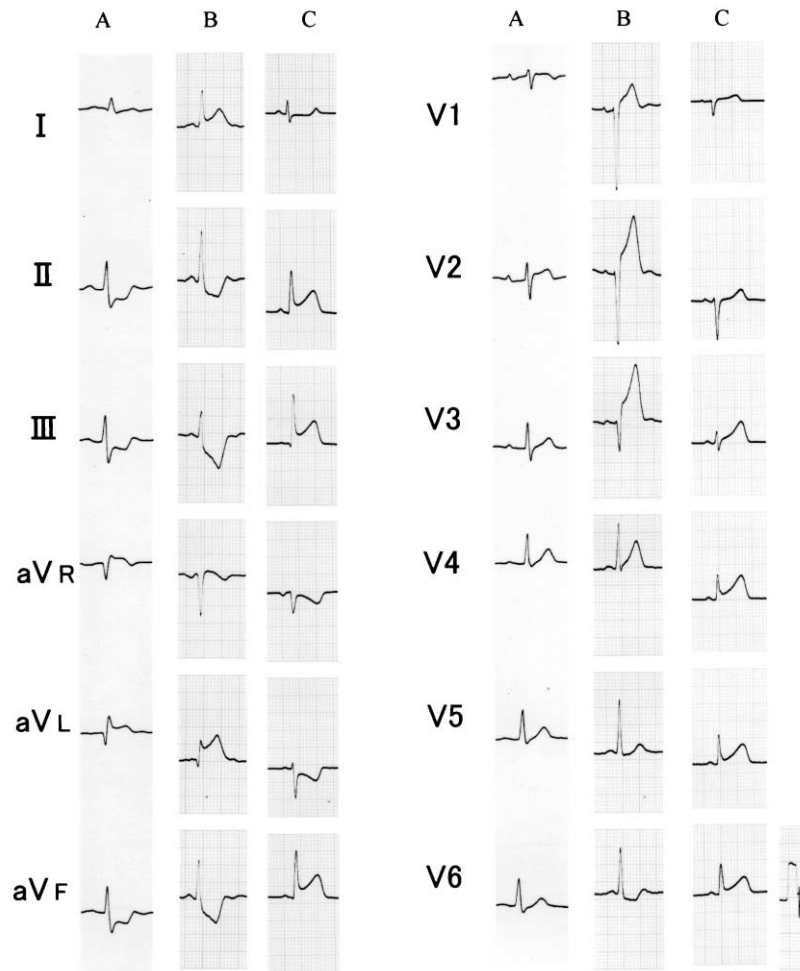


Figure 1. Representative 12-lead electrocardiogram tracings at admission in a patient in (A) the left main coronary artery (LMCA) group, (B) the left anterior descending coronary artery (LAD) group and (C) the right coronary artery (RCA) group. In the patient in the LMCA group, ST segment elevation is apparent in lead aVR. In the patient in the LAD group, marked ST segment elevation in the precordial leads is seen, whereas a ST segment shift in lead aVR is negligible. In the patient in the RCA group, ST segment elevation in the inferior leads is marked.

LMCA group from the LAD group (Table 2). The results were completely in agreement with the results obtained by univariate analysis.

Comparison between the LMCA and RCA groups.

UNIVARIATE ANALYSIS. ST segment elevation in lead aVR (>0.05 mV) occurred with a significantly higher incidence in the LMCA group (88% [14/16]) than in the RCA group (8% [2/24]), and the elevation distinguished the LMCA group from the RCA group, with 88% sensitivity, 92% specificity and 90% accuracy. Conversely, the LMCA group clearly showed a lower incidence of ST segment elevation in leads II (6% [1/16]), III (13% [2/16]) and aVF (0% [0/16]) than the RCA group (92% [22/24], 96% [23/24] and 96% [23/24], respectively). The ST segment elevations in leads II, III and aVF were useful for distinguishing the LMCA group from the RCA group, with high sensitivity (92%, 96% and 96%, respectively), specificity (94%, 88% and 100%, respectively) and accuracy (93%, 93% and 98%, respectively). Lead aVF showed the highest sensitivity,

specificity and accuracy for distinguishing the LMCA group from the RCA group.

MULTIVARIATE ANALYSIS. Stepwise linear multivariate discriminant analysis selected lead aVF rather than lead aVR as the lead whose ST segment shift contributed significantly to distinguishing the LMCA group from the RCA group, which was consistent with the results of univariate analysis in that lead aVF showed the highest sensitivity, specificity and accuracy.

ST segment shift in lead aVR and patients' clinical outcomes in the LMCA group.

Table 3 summarizes the findings about the relationship between the ST segment shift in lead aVR and patients' clinical outcomes in the LMCA group. Death occurred more frequently in patients with higher ST segment elevation in lead aVR than in those with less severe elevation. When ST segment elevation of 0.15 mV was used as the cut-off value, death was predicted with 75% sensitivity, 75% specificity and 75% accuracy.

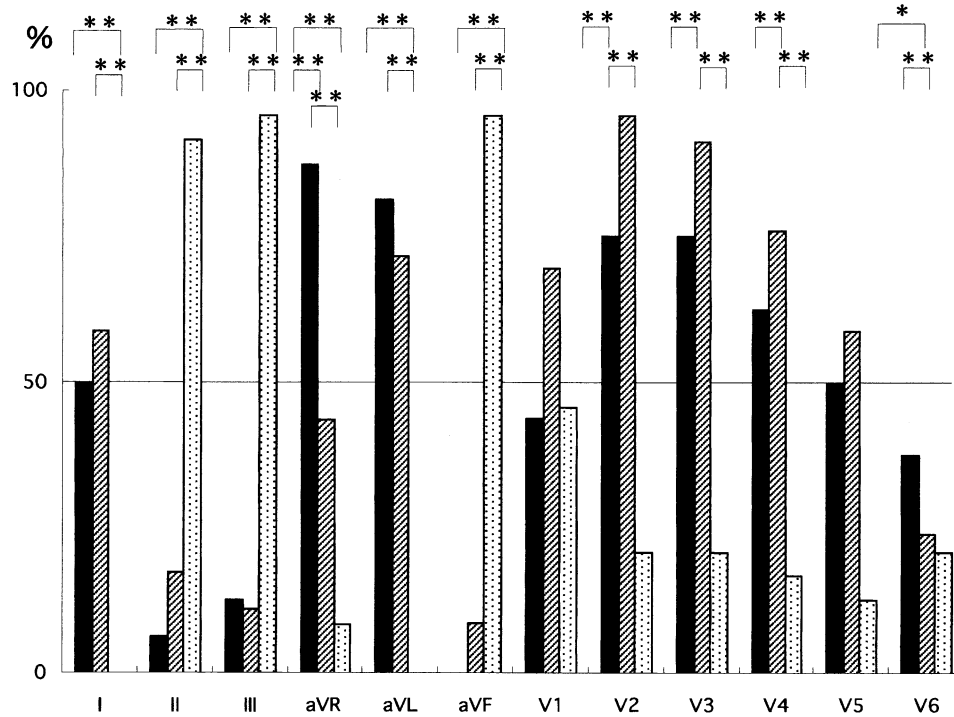


Figure 2. The incidences of ST segment elevation in each lead in the left main coronary artery (LMCA) group (n = 16; solid bars), left anterior descending coronary artery (LAD) group (n = 46; hatched bars) and right coronary artery (RCA) group (n = 24; dotted bars). *p < 0.05. **p < 0.01.

Differences in ST segment elevation between lead aVR and lead V₁ did not contribute significantly to predicting mortality.

Stepwise multivariate linear discriminant analysis also showed that ST segment elevation in lead aVR significantly contributed to predicting patients' clinical outcomes in the LMCA group (Table 3).

DISCUSSION

The present study revealed that high ST segment elevation in lead aVR, compared with lead V₁, was a useful indicator for predicting acute LMCA obstruction, which requires immediate aggressive treatment. In acute LMCA obstruction,

the ST segment elevation in lead aVR also contributed to predicting the patients' clinical outcomes. Thus, the present results are clinically useful for selecting the treatment strategy for patients with acute MI.

Total obstruction or severe stenosis with flow delay in the LMCA lesion was demonstrated by emergency coronary angiography in all patients in the LMCA group. Similarly, neither the LAD group nor the RCA group included patients with significant stenotic lesion(s) other than the culprit lesion. ST segment elevation after acute MI changes with time. This study included only patients who were admitted within 12 h (3.2 ± 2.7 h) from the onset of acute MI, during the period when acute ST segment elevation is

Table 2. Results of Univariate and Multivariate Analyses for Distinguishing Between the LMCA and LAD Groups

Univariate Analysis					
ST Segment Shift	LMCA Group	LAD Group	p Value		
Lead aVR (mV)	0.16 ± 0.13	0.04 ± 0.10	<0.0001		
Lead V ₁ (mV)	0.00 ± 0.21	0.14 ± 0.11	<0.0001		
Leads aVR-V ₁ (mV)	-0.16 ± 0.25	-0.09 ± 0.13	<0.0001		
Multivariate Analysis (Stepwise Linear Multiple Discriminant Analysis)					
Selected Factor	Discriminant Coefficient	Mahalanobis' Sum of Squares	Partial F Value	p Value	F Value of Discriminant Equation
Lead aVR	0.747	2.63	3.31	0.074	8.53 (p < 0.01)
Lead V ₁	-0.817	2.34	5.7	0.02	

Results of discrimination (percent correct): LMCA group: 63%; LAD group: 96%. Sensitivity = 63%, specificity = 96% and accuracy = 87%. Univariate data are presented as the mean value ± SD.

LAD = left anterior descending coronary artery; LMCA = left main coronary artery.

Table 3. Results of Univariate and Multivariate Analyses for Prediction of Clinical Outcome

Univariate Analysis					
ST Segment Shift	Dead	Alive	p Value		
Lead aVR (mV)	0.23 ± 0.14	0.09 ± 0.09	<0.05		
Lead V ₁ (mV)	0.01 ± 0.19	-0.02 ± 0.02	NS		
Leads aVR-V ₁ (mV)	0.21 ± 0.15	0.11 ± 0.33	NS		
Multivariate Analysis (Stepwise Linear Multiple Discriminant Analysis)					
Selected Factor	Discriminant Coefficient	Mahalanobis's Sum of Squares	Partial F Value	p Value	F Value of Discriminant Equation
Lead aVR	-0.96	1.25	5.02	0.042	11.06 (p < 0.01)
Other leads were not selected.					

Results of discrimination (percent correct): dead, 88%; alive, 75%. Sensitivity = 75%; specificity = 88%; accuracy = 81%. Univariate data are presented as the mean value ± SD. NS = not significant.

observed. In all patients studied, the acute MI was the first MI. Univariate and multivariate analyses showed essentially identical results. The selection of these patients and the methods of analysis were such that the ECG findings reflected the location of the myocardium perfused by each coronary artery, so they could be compared among the three groups.

Lead aVR ST segment elevation. The present study found lead aVR ST segment elevation in 43% (20/46) of the patients in the LAD group; this incidence is in good agreement with the incidence reported by Engelen et al. (4) in patients with acute LAD obstruction in which the culprit lesion was located proximal to the first major septal branch. These investigators concluded that lead aVR ST segment elevation in acute, proximal LAD occlusion is the result of transmural ischemia of the basal part of the septum, where the injury's electric current is directed toward the right shoulder. It is certainly reasonable to theorize that acute LMCA obstruction also causes ischemia of the basal part of the septum through disturbance of the major septal branch blood flow—that is, interruption of the proximal LAD blood flow. This would account for lead aVR ST segment elevation associated with acute LMCA obstruction. In two patients in the RCA group with lead aVR ST segment elevation, a well-developed and dominant RCA was observed. This suggested that ischemia of the basal part of the septum might be caused by blood flow disturbance in interventricular branches, arising from the well-developed RCA, thus resulting in lead aVR ST segment elevation in these two patients. We found a higher incidence of lead aVR ST segment elevation in the LMCA group than in the LAD group, as well as a significant relationship between the amplitude of lead aVR ST segment elevation and the patients' clinical outcomes in the LMCA group. However, the present study apparently lacks any direct evidence about the underlying mechanisms that account for these findings. Further discussions on the high incidence of lead aVR ST segment elevation and the association between the amplitude of lead aVR ST segment elevation and patients' clinical outcomes in acute LMCA obstruction are unwarranted.

ST segment elevation in lead aVR versus lead V₁. Acute LMCA obstruction, but not LAD obstruction, ordinarily causes ischemia of the posterior wall through disturbance of LCx blood flow. It is reasonable to assume that the electrical force in posterior wall ischemia counterbalances the ischemia-induced electrical force in the anterior wall. In fact, several reports have shown reciprocal changes in the precordial leads (V₁ and V₂) induced by posterior wall ischemia that was caused by LCx obstruction (9,10). The most likely interpretation of less ST segment elevation in lead V₁ in the LMCA group compared with the LAD group is that it is the result of the electrical force induced by posterior wall ischemia, associated with LMCA obstruction counterbalancing the ischemia-induced electrical force in the anterior wall.

Previous reports of acute LMCA obstruction. A total of 42 patients with acute LMCA obstruction have been described in 21 reports published in English, according to our search results (1-3,11-29). The ECG findings were not described in four reports, representing 10 patients (1,13,16,26). ST segment changes consistent with anterior wall MI were reported in 25 of the remaining 32 patients. ST segment depression in the precordial leads was reported in seven patients and right bundle branch block in two patients. None of these reports described the findings of lead aVR, except for our previous report (19), in which a ST segment shift in lead aVR was observed in five of eight patients. The ECG interpreters often ignore findings in lead aVR. Pahlm et al. (30) investigated whether 35 ECG interpreters ignored lead aVR when using complex electrocardiography in which lead aVR was or was not replaced by lead -aVR, and they found that the vast majority of interpreters did not detect the reversed lead aVR. This would explain why previous studies did not evaluate or describe the findings of lead aVR ST segment changes. Nevertheless, our findings of lead aVR ST segment elevation in association with acute LMCA obstruction appear to be consistent with a study by Engelen et al. (4) and a study in which ECGs were recorded during anginal attack in patients with LMCA disease and multivessel disease (5).

Study limitations. One of the limitations of our study is that it included a relatively small number of patients, because acute LMCA obstruction is not common. Careful patient selection and analysis by multiple methods (i.e., univariate and multivariate analyses), however, may have at least partly compensated for this limitation.

Conclusions. The present study showed that in patients with acute MI, careful attention to lead aVR ST segment elevation to predict acute LMCA obstruction is clinically important with respect to selection of the treatment strategy. The ST segment elevation in lead aVR also contributed to predicting patients' clinical outcomes in acute LMCA obstruction.

Reprint requests and correspondence: Dr. Shozo Kusachi, Department of Internal Medicine I, Okayama University Medical School, 2-5-1, Shikata-cho, Okayama 700-8558, Japan. E-mail skusachi@ccews2.cc.okayama-u.ac.jp.

REFERENCES

1. de Feyter PJ, Serruys PW. Thrombolysis of acute total occlusion of the left main coronary artery in evolving myocardial infarction. *Am J Cardiol* 1984;53:1727–8.
2. Goldberg S, Grossman W, Markis JE, et al. Total occlusion of the left main coronary artery: a clinical, hemodynamic and angiographic profile. *Am J Med* 1978;64:3–8.
3. Ward DE, Valantine H, Hui W. Occluded left main stem coronary artery: report of five patients and review of published reports. *Br Heart J* 1983;49:276–9.
4. Engelen DJ, Gorgels AP, Cheriex EC, et al. Value of the electrocardiogram in localizing the occlusion site in the left anterior descending coronary artery in acute anterior myocardial infarction. *J Am Coll Cardiol* 1999;34:389–95.
5. Gorgels AP, Vos MA, Mulleneers R, et al. Value of the electrocardiogram in diagnosing the number of severely narrowed coronary arteries in rest angina pectoris. *Am J Cardiol* 1993;72:999–1003.
6. Dunn RF, Newman HN, Bernstein L, et al. The clinical features of isolated left circumflex coronary artery disease. *Circulation* 1984;69:477–84.
7. Mueller HS, Dyer A, Greenberg MA, et al., the TIMI Study Group. The Thrombolysis in Myocardial Infarction (TIMI) trial: phase I findings. *N Engl J Med* 1985;312:932–6.
8. Rentrop KP, Cohen M, Blanke H, et al. Changes in collateral channel filling immediately after controlled coronary artery occlusion by an angioplasty balloon in human subjects. *J Am Coll Cardiol* 1985;5:587–92.
9. Matetzky S, Freimark D, Feinberg MS, et al. Acute myocardial infarction with isolated ST-segment elevation in posterior chest leads V₇₋₉: 'hidden' ST-segment elevations revealing acute posterior infarction. *J Am Coll Cardiol* 1999;34:748–53.
10. Khaw K, Moreyra AE, Tannenbaum AK, et al. Improved detection of posterior myocardial wall ischemia with the 15-lead electrocardiogram. *Am Heart J* 1999;138:934–40.
11. Spiecker M, Erbel R, Rupprecht HJ, et al. Emergency angioplasty of totally occluded left main coronary artery in acute myocardial infarction and unstable angina pectoris: institutional experience and literature review. *Eur Heart J* 1994;15:602–7.
12. Cohen MC, Ferguson DW. Survival after myocardial infarction caused by acute left main coronary artery occlusion: case report and review of the literature. *Cathet Cardiovasc Diagn* 1989;16:230–8.
13. Topaz O, Disciascio G, Cowley MJ, et al. Complete left main coronary artery occlusion: angiographic evaluation of collateral vessel patterns and assessment of hemodynamic correlates. *Am Heart J* 1991;121:450–6.
14. Flugelman MY, Shalit M, Shefer A, et al. Survival after sudden obstruction of the left main coronary artery. *Am J Cardiol* 1983;51:900–1.
15. Salvi A, Klugmann S, Della Grazia E, et al. Myocardial reperfusion after acute occlusion of the left main coronary artery. *Am J Cardiol* 1983;51:1791.
16. Groves PH, Ikram S, Hayward MW, et al. Emergency angioplasty of the left main coronary artery. *Eur Heart J* 1989;10:1123–5.
17. Oatfield RG, Nordmark SP. Acute total occlusion of the left main coronary artery associated with long-term survival: a case report. *Angiology* 1989;40:309–12.
18. Biron Y, Laurent M, Bourdonnec C, et al. Thrombolysis of acute total occlusion of the left main coronary artery and long-term survival. *Clin Cardiol* 1987;10:283–5.
19. Iwasaki K, Kusachi S, Hina K, et al. Acute left main coronary artery obstruction with myocardial infarction: reperfusion strategies and the clinical and angiographic outcome. *Jpn Circ J* 1993;57:891–7.
20. von Essen R, Lambert H, Schmidt W, et al. Successful recanalization of a left main coronary artery occlusion. *Am J Cardiol* 1984;53:356–7.
21. Alosilla CE, Bell WW, Ferree J, et al. Thrombolytic therapy during acute myocardial infarction due to sudden occlusion of the left main coronary artery. *J Am Coll Cardiol* 1985;5:1253–6.
22. Sigwart U, Goy JJ, Finci L, et al. Mechanical emergency recanalization of the left main coronary artery. *Clin Cardiol* 1986;9:217–20.
23. Finzi LA, Secches AL, Evora PR, et al. Myocardial reperfusion by thrombolysis after acute total left main artery occlusion: a case report. *Angiology* 1987;38:417–21.
24. O'Shaughnessy MA, Ransbottom JC, Stiles BS, et al. Acute left main coronary artery occlusion: survival following emergent coronary bypass. *Arch Intern Med* 1987;147:2207–9.
25. Siemons L, Ranquin R, Van den Heuvel P, et al. Intravenous streptokinase-mediated thrombolysis of acute occlusion of the left main coronary artery. *Am J Cardiol* 1987;60:1403–4.
26. Smith DF, Higginson LA, Walley VM. Reperfusion hemorrhage following PTCA and thrombolysis for left main coronary artery occlusion. *Can J Cardiol* 1988;4:33–6.
27. Napchan BP, Caramelli B, Tranchesi B Jr, et al. Percutaneous transluminal angioplasty of the left main coronary artery in acute myocardial infarction. *Arq Bras Cardiol* 1990;55:113–5.
28. Prachar H, Dittel M, Enenkel W. Acute occlusion of left main coronary artery without ventricular damage. *Clin Cardiol* 1991;14:176–9.
29. Takayanagi K, Satoh T, Inoue T, et al. Survival from acute occlusion of the left main coronary artery with preexisting collateral vessels: a case report. *Angiology* 1991;42:935–9.
30. Pahlm US, Pahlm O, Wagner GS. The standard 11-lead ECG: neglect of lead aVR in the classical limb lead display. *J Electrocardiol* 1996;29 Suppl:270–4.