

Value of lead aVR in predicting acute occlusion of proximal left anterior descending coronary artery and in-hospital outcome in ST-elevation myocardial infarction: an electrocardiographic predictor of poor prognosis

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Abstract

Background: We aimed to investigate the value of ST elevation in lead aVR (ST \uparrow aVR) in predicting the left anterior descending coronary artery (LAD) occlusion site proximal to first septal perforator (S₁) and its effect on in-hospital outcome in ST-elevation myocardial infarction (STEMI).

Methods: The study included 950 patients with STEMI. Patients were divided into 2 groups as aVR (+) and aVR(–) according to the presence of an ST \uparrow aVR of 0.5 mm or greater.

Results: ST elevation in lead aVR was seen in 155 (16%) patients, and LAD occlusion proximal to S₁ was detected in 52% of patients in the aVR(+) group and in 9% of patients in the aVR(–) group. aVR positivity was associated with higher heart rate, lower systolic blood pressure and ejection fraction, and worse Killip class at the hospital admission. In-hospital mortality was 19% in the aVR(+) group and 5% in the aVR(–) group. aVR positivity was an independent predictor of in-hospital death.

Conclusion: This study revealed that ST \uparrow aVR was not only a good indicator of LAD occlusion proximal to S₁ but also a source of valuable information about in-hospital outcome in patients with STEMI.

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Keywords:

Electrocardiography; In-hospital mortality; Lead aVR; Left anterior descending coronary artery; ST elevation myocardial infarction

Introduction

ST-elevation myocardial infarction (STEMI) is still associated with high in-hospital mortality. Anterior STEMI is known to have worse prognosis than nonanterior STEMI does. On the other hand, the prognosis of anterior STEMI varies according to the site of occlusion. Anterior STEMI due to acute obstruction of left main coronary artery (LMCA) or proximal left anterior descending coronary artery (LAD), both of which have worse

prognosis, causes severe hemodynamic instability as a consequence of greater infarction area.^{1–3} In addition, the presence of significant lesions in LMCA or proximal LAD, even when they are not infarct-related artery (IRA), is a factor affecting the prognosis and treatment of STEMI. Early and noninvasive prediction of these lesions may help to choose more aggressive treatment options, thereby helping the improvement of prognosis.

Electrocardiography (ECG) is a valuable, cheap, and noninvasive method used in the diagnosis and risk stratification of STEMI. The use of lead aVR for risk stratification in acute coronary syndromes has been a subject of great interest recently. Significant lesions in LMCA or proximal LAD lead to transmural ischemia in the basal part of the septum, which results in ST elevation in lead aVR (ST \uparrow aVR).⁴ Based on this information, it was reported that

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the ST \uparrow aVR may be suggestive of LMCA or proximal LAD occlusion in patients with STEMI, but this has not been tested satisfactorily in studies with great sample sizes. There is some evidence on the fact that analysis of ST-segment deviation in lead aVR might provide valuable data about prognosis in patients with non-STEMI and unstable angina pectoris.^{5,6} Although the effect of ST \uparrow aVR on mortality has been assessed in acute obstruction of LMCA,⁷ it has not been evaluated in patients with acute obstruction of other major coronary arteries.

The aim of this study was to investigate the value of ST \uparrow aVR in predicting acute proximal LAD occlusion, the extent of coronary artery disease (CAD), and in-hospital mortality in patients with STEMI.

Materials and methods

Study population

In this prospective study, we consecutively included 950 patients (742 men and 208 women; mean \pm SD age, 59 \pm 12 years [range, 24–96 years]) admitted to the Cardiology Clinic between the years 2001 and 2007 with the diagnosis of STEMI who underwent coronary angiography. ST-elevation myocardial infarction was defined as typical chest pain lasting 30 minutes or more with typical ST elevation (≥ 1 mm in at least 2 contiguous derivations) in surface 12-lead ECG. Blood samples were obtained from the patients within the first 24 hours to analyze lipid and creatine kinase levels. Creatine kinase measurements were repeated every 6 hours until they reached maximal levels. Patients with complete left bundle branch block, onset of symptom more than 24 hours, previous history of coronary artery bypass surgery, severe valvular heart disease other than ischemic mitral regurgitation, congenital cardiac anomaly, or left ventricular hypertrophy were excluded from the study.

Coronary angiography

Coronary angiography had been performed on 73% of the patients within the first 6 hours, 25% within 1 to 7 days, and 2% within 8 to 12 days after admission (mean \pm SD, 0.9 \pm 0.06 days). Angiographic images were assessed by 2 independent cardiologists. Infarct-related artery was determined angiographically, and significant stenoses in other major coronary arteries were also recorded. A stenosis was considered significant when the reduction in diameter in any projection was equal to or greater than 50% of the adjacent normal diameter of LMCA or equal to or greater than 70% of the adjacent normal diameter of other major epicardial coronary arteries. In addition, collateral circulation was assessed, and dominant artery was determined. Proximal LAD was defined as the segment proximal to the first “visible” septal artery (S₁) without considering its size.⁸ Lesions in LAD were classified into 2 groups as proximal or distal to S₁. The S₁ takeoff from LAD was recorded as proximal or distal to the first diagonal branch (D₁). Modified Gensini scoring system was used to assess the extent of CAD.⁹ Any significant lesion in LMCA or proximal LAD

was defined as “significant vessel disease” without taking IRA into consideration.

Electrocardiography

The ECG with the highest ST elevation obtained in acute phase before reperfusion therapy was evaluated. The time interval between the onset of chest pain and the ECG showing maximal ST elevation was 253 \pm 218 minutes (15–1440 minutes). Two independent cardiologists evaluated the ECG samples. Disagreement of ECG interpretation was resolved by consensus. All ST-segment deviations of 0.5 mm or greater at 60 milliseconds after the J point were recorded using the TP-segment as isoelectric line. Patients with an ST \uparrow aVR of 0.5 mm or greater (Fig. 1) were considered as aVR(+) group, and the remaining patients were considered as aVR(–) group. Apart from ST-segment deviation, right bundle branch block (RBBB) and complete atrioventricular block were recorded as defined before.¹⁰

In addition to angiographic and electrocardiographic findings, the risk factors for CAD, revascularization procedures, Killip class at admission, and left ventricular ejection fraction (LVEF) calculated with modified Simpson’s method by echocardiography within 3 days after admission and death during hospitalization period were recorded.

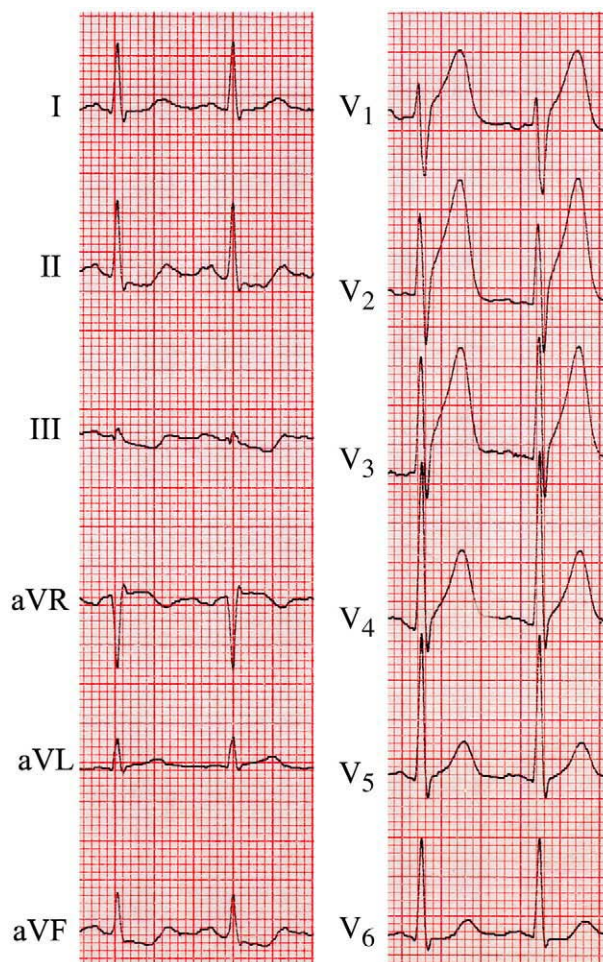


Fig. 1. An electrocardiogram of the patient with proximal LAD occlusion with typical ST \uparrow aVR.

Statistics

Statistical analyses were performed with SPSS for Windows 13.0 software (SPSS Inc, Chicago, IL). The results were expressed as mean \pm SD. Clinical and laboratory variations of the aVR(+) group were compared with those of the aVR(−) group. Sensitivity, specificity, positive and negative predictive values, and diagnostic accuracy were calculated by standard formulas. The correlation between ST-segment deviation in lead aVR and Gensini score was investigated.

Nonparametric values of the 2 groups were compared using Mann-Whitney *U* test and χ^2 test, whereas the parametric values were compared by Student *t* test. The correlation between the parameters was evaluated by Pearson correlation coefficient. Logistic regression analysis was used to determine the factors affecting in-hospital mortality. In 70 randomized patients, ST-segment measurements in aVR were repeated by another observer blinded to clinical and angiographic findings, and then interobserver correlation coefficient and absolute agreement were calculated. A *P* value $< .05$ was considered significant.

Results

Demographic and clinical characteristics

The baseline demographic characteristics of the patients are shown in Table 1. In the aVR(+) group, there were 155 (16%) patients. The proportion of female patients and history of hypertension were higher in the aVR(+) group than in the aVR(−) group (*P* = .001 and *P* = .03, respectively). aVR positivity was associated with higher heart rate, lower systolic and diastolic blood pressure, lower LVEF, and worse Killip class at hospital admission (*P* $< .001$, *P* = .001, *P* = .001, *P* $< .001$, and *P* = .001, respectively). Peak serum creatine kinase levels were higher in the aVR(+) group (*P* = .04). Moreover, the number of patients who had undergone coronary artery bypass surgery as emergency revascularization therapy was significantly higher in the aVR(+) group than in the aVR(−) group (*P* $< .001$) (Table 2).

Table 1
Demographic characteristics of the study patients

	aVR(+) group (n = 155)	aVR(−) group (n = 795)	<i>P</i>
Age (y)	60 \pm 12	59 \pm 11	.14
Sex (male)	106 (68)	636 (80)	.001
Hypertension	66 (45)	276 (35)	.03
Diabetes mellitus	36 (25)	167 (21)	.41
Smoking history	76 (52)	429 (55)	.55
Heredity	32 (22)	181 (23)	.75
Previous myocardial infarction	12 (8)	38 (5)	.13
Total cholesterol (mg/dL)	192 \pm 46	186 \pm 48	.18
Triglycerides (mg/dL)	126 \pm 67	125 \pm 81	.84
HDL-cholesterol (mg/dL)	38 \pm 10	37 \pm 10	.47
LDL-cholesterol (mg/dL)	130 \pm 40	124 \pm 40	.09

Values are presented as mean \pm SD or n (%), where appropriate. HDL indicates high-density lipoprotein; LDL, low-density lipoprotein.

Table 2

Clinical characteristics of the study patients

	aVR(+) group (n = 155)	aVR(−) group (n = 795)	<i>P</i>
Heart rate on admission (beats/min)	86 \pm 22	77 \pm 19	<.001
Systolic blood pressure (mm Hg)	110 \pm 29	119 \pm 27	.001
Diastolic blood pressure (mm Hg)	69 \pm 21	75 \pm 16	.001
LVEF (%)	41 \pm 10	45 \pm 9	<.001
Time from angina to admission (min)	268 \pm 219	250 \pm 217	.36
Creatine kinase	2827 \pm 2180	2358 \pm 1825	.04
Killip class			.001
I	96 (62)	620 (78)	
II	45 (29)	151 (19)	
III	9 (6)	16 (2)	
IV	5 (3)	8 (1)	
In-hospital death	29 (19)	41 (5)	<.001
Revascularization procedure			<.001
Fibrinolytic therapy	41 (26)	270 (34)	.07
Rescue PCI	3 (2)	37 (5)	.12
Primary PCI	71 (46)	403 (51)	.27
Coronary artery bypass grafting	23 (15)	20 (2)	<.001
Other ^a	17 (11)	65 (8)	.26

Values are presented as mean \pm SD or n (%), where appropriate. PCI indicates percutaneous coronary intervention.

^a Spontaneous recanalization or patients' denial of the therapy.

Electrocardiographic characteristics

In the aVR(+) group, 76% of the patients had anterior STEMI, and the remaining patients (24%) had inferior STEMI. aVR was positive in 24% of the patients with anterior STEMI and in 8% of those with inferior STEMI (*P* $< .001$). Right bundle branch block was more prevalent in the aVR(+) group than in the aVR(−) group (*P* = .03) (Table 3).

Table 3
Electrocardiographic and coronary angiographic characteristics of the patients

	aVR(+) group (n = 155)	aVR(−) group (n = 795)	<i>P</i>
Anterior STEMI	118 (76)	366 (46)	<.001
RBBB	16 (10)	45 (6)	.03
Complete AV block	3 (2)	33 (4)	.19
No. of diseased vessels			
1	69 (44)	478 (60)	<.001
2	48 (31)	236 (30)	.75
3	38 (25)	81 (10)	<.001
Significant vessel disease ^a	86 (56)	86 (11)	<.001
Gensini score	183 \pm 68	119 \pm 49	<.001
IRA			
LMCA	4 (2.6)	1 (0.1)	<.001
LAD	114 (73.6)	365 (45.9)	<.001
Proximal to S ₁	80 (52)	72 (9)	<.001
Proximal to S ₁ and D ₁	73 (47)	66 (8)	<.001
Proximal to S ₁ , distal to D ₁	7 (5)	6 (1)	<.001
Distal to S ₁	34 (22)	293 (37)	<.001
Distal to S ₁ , proximal to D ₁	8 (5)	48 (6)	.852
Distal to S ₁ and D ₁	26 (17)	245 (31)	.001
Left circumflex artery	3 (1.9)	94 (11.9)	<.001
Right coronary artery	34 (21.9)	335 (42.1)	<.001

Values are presented as mean \pm SD or n (%), where appropriate. AV indicates atrioventricular; D₁ first diagonal artery; S₁, first septal artery.

^a The patients with LMCA or proximal LAD lesion regardless of IRA.

The following were analyzed to predict acute occlusion of LMCA or proximal LAD in patients with anterior STEMI: ST \uparrow aVR, ST elevation in lead V1 and DI/aVL, ST depression in inferior leads and V₅/V₆, and RBBB (Table 4). An ST \uparrow aVR of 0.5 mm or greater was the strongest predictor of LMCA or LAD occlusion proximal to S₁ in the analysis of logistic regression (Table 5).

Interobserver correlation coefficient and absolute agreement in ST-segment measurements in aVR were 97% and 98%, respectively.

Coronary angiographic characteristics

One-vessel disease was observed in 57% of the patients; 2-vessel disease, in 30%; and 3-vessel disease, in 13%. Whereas 1-vessel disease was more prevalent in the aVR(–) group, 3-vessel disease had a higher prevalence in the aVR(+) group. Gensini score, an indicator of the extent of CAD, was higher in the aVR(+) group (Table 3). Interestingly, it was lower in patients with ST depression in aVR of 1 mm or greater than in those without ST-segment deviation (109 ± 47 vs 122 ± 49 , respectively; $P = .008$). ST-segment deviation in aVR had a weak but statistically significant positive correlation with Gensini score ($r = 0.34$, $P < .001$) (Fig. 2). Significant vessel disease was more prevalent in the aVR(+) group. An ST \uparrow aVR of 0.5 mm or greater was suggestive of significant vessel disease, with a low sensitivity but a high specificity and negative predictive value (Table 6).

The distribution of IRA

The segmentary distribution of IRA in the aVR(+) and aVR(–) groups is presented in Table 3. Infarct-related artery was determined as LMCA in 5 patients and as LAD proximal to S₁ in 152 patients. Four of the 5 patients in whom IRA was LMCA were in the aVR(+) group. The prevalence of LAD occlusion proximal to S₁ was higher in the aVR(+) group. aVR positivity was significantly higher in the site of occlusion proximal to S₁ and distal to D₁. However, the number of patients in whom IRA was left circumflex (LCx) or right coronary artery (RCA) was higher in the aVR(–) group (Table 3). Interestingly, there were 3 patients in the aVR(+) group in whom IRA was LCx, the dominant artery in those 3 patients.

Table 4
Electrocardiographic predictors of acute LMCA or LAD occlusion proximal to S₁ in patients with anterior STEMI

	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)	Diagnostic accuracy (%)
ST \uparrow aVR ≥ 0.5 mm	54	90	71	80	78
ST \downarrow II ≥ 1 mm	47	85	59	77	73
ST \downarrow III ≥ 1 mm	65	68	49	80	67
ST \downarrow aVF ≥ 1 mm	58	79	53	79	70
ST \uparrow V ₁ ≥ 2.5 mm	22	85	41	69	64
ST \uparrow D ₁ /aVL ≥ 1 mm	51	61	39	72	58
ST \downarrow V ₅ /V ₆	29	88	54	72	69
RBBB	18	93	56	70	69
RBBB and ST \uparrow aVR ≥ 0.5 mm	9	99	88	69	70

NPV indicates negative predictive value; PPV, positive predictive value; ST \uparrow x, ST-segment elevation in lead x; ST \downarrow x, ST-segment depression in lead x.

Table 5

The results of logistic regression analysis for the electrocardiographic predictors of acute LMCA or LAD occlusion proximal to S₁

Predictors	Odds ratio	95% Confidence interval		P
		Lower	Upper	
ST \uparrow aVR ≥ 0.5 mm	10.192	5.730	18.130	<.001
ST \downarrow II ≥ 1 mm	1.522	0.791	2.931	.209
ST \downarrow III ≥ 1 mm	4.421	2.078	9.404	<.001
ST \downarrow aVF ≥ 1 mm	1.627	0.761	3.480	.210
ST \uparrow V ₁ ≥ 2.5 mm	1.255	0.681	2.312	.467
ST \uparrow D ₁ /aVL ≥ 1 mm	1.166	0.623	2.184	.631
ST \downarrow V ₅ /V ₆	1.824	1.041	3.194	.036
RBBB	4.415	2.066	9.433	<.001
RBBB and ST \uparrow aVR ≥ 0.5 mm	0.903	0.142	5.733	.913

ST \uparrow x, ST-segment elevation in lead x; ST \downarrow x, ST-segment depression in lead x.

In-hospital outcome

Seventy (7%) patients died during hospitalization period. Mortality rate was 19% in the aVR(+) group and 5% in the aVR(–) group ($P < .001$). Statistical analyses revealed that age, sex, history of hypertension and diabetes mellitus, heart rate, systolic and diastolic blood pressure, LVEF, Killip class, RBBB, and ST \uparrow aVR were significantly associated with in-hospital mortality (Table 7).

However, a logistic regression analysis including the parameters of Killip class, ST \uparrow aVR, history of diabetes mellitus, age, systolic blood pressure, heart rate, and LVEF showed that Killip class, an ST \uparrow aVR of 0.5 mm or greater, and systolic blood pressure were independent predictors of death during hospitalization period (Table 8).

Discussion

The findings of this study showed that an ST \uparrow aVR of 0.5 mm or greater was a valuable parameter that might be used to predict that IRA is proximal LAD in patients with anterior STEMI and that significant vessel disease exists in anterior and nonanterior STEMI. Moreover, this parameter was a strong predictor of in-hospital death.

Lead aVR has been largely ignored by electrocardiographers until recent years. This was partly because of the belief that lead aVR reflected reciprocal changes in the lateral part of the heart derivations (D₁, aVL, V₅, and V₆).¹¹ In contrast to this consideration, a few studies performed recently in patients with non-ST-elevation acute coronary syndrome revealed that lead aVR might give valuable information about the extent of CAD. Gorgels and colleagues⁵ reported that ST \uparrow aVR indicated LMCA lesion or 3-vessel disease in patients with unstable angina pectoris. Similarly, Barrabes and colleagues⁶ reported that an ST \uparrow aVR of 1 mm or greater indicated LMCA lesion or 3-vessel disease in patients with non-STEMI.

The data about lead aVR in STEMI are relatively limited. An acute obstruction of LMCA or proximal LAD, which is defined as “the segment proximal to first septal artery,” results in a greater area of necrosis in comparison to more distal lesions. Noninvasive and early detection of these

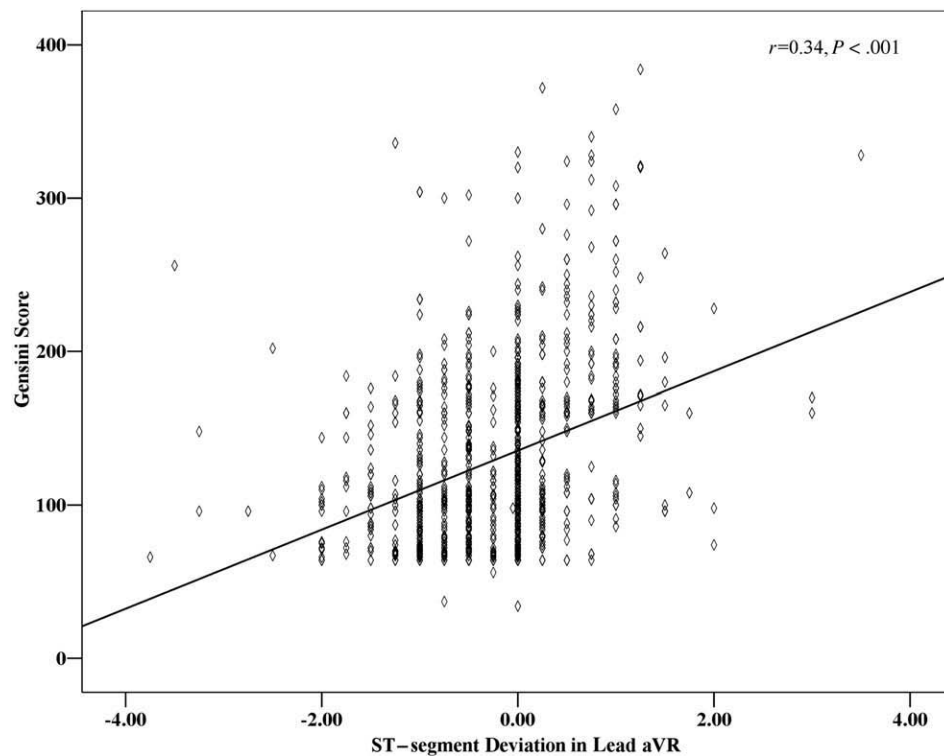


Fig. 2. The correlation between ST-segment deviation in lead aVR and Gensini score in patients with acute STEMI.

lesions and administration of invasive reperfusion procedures were shown to improve the prognosis.^{12–15} A few electrocardiographic criteria were defined for the prediction of LMCA or proximal LAD occlusion in cases of anterior STEMI. Newly developed RBBB, ST depression of 1 mm or greater in inferior leads, ST elevation of 2.5 mm or greater in lead V₁, and ST depression in lead V₅ were some of these criteria defined in previous studies.⁴ In addition, it was shown that the ST \uparrow aVR might also be a valuable parameter. In a study including 100 patients with anterior STEMI, it was shown that ST \uparrow aVR indicated proximal LAD occlusion with 43% sensitivity and 95% specificity.⁴ In another study that compared the changes in lead aVR in cases with acute occlusion of LMCA, proximal LAD, or RCA, Yamaji and colleagues⁷ reported that an ST \uparrow aVR of 1 mm or greater predicted acute LMCA occlusion with 88% sensitivity. We found aVR positivity in 54% of acute LMCA or proximal LAD occlusion, whereas Engelen⁴ and Yamaji⁷ reported this ratio as 43%. This difference may be due to the variations in

definitions of S₁ and/or aVR positivity criteria. We defined S₁ as the first “visible” septal perforator branch without considering its size,⁸ whereas Engelen and Yamaji regarded it as the first “major” septal perforator artery. Moreover, we considered the level of significance for ST \uparrow aVR as 0.5 mm at 60 milliseconds after the J point, but Engelen⁴ defined this level as 1 mm at J point.

In our study, ST \uparrow aVR was the strongest of all ECG diagnostic parameters used to predict acute proximal LAD occlusion in anterior STEMI. Some hypotheses have been

Table 6

Sensitivity, specificity, positive and negative predictive values, and diagnostic accuracy of an ST \uparrow aVR of 0.5 mm or greater in patients with STEMI in predicting significant vessel disease^a

	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)	Diagnostic accuracy (%)
Overall STEMI	50	91	55	89	84
Anterior STEMI	53	88	64	82	77
Nonanterior STEMI	36	94	27	95	90

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

^a Significant vessel disease: the patients with LMCA or proximal LAD lesion regardless of IRA.

Table 7

The association between demographic, clinical, and electrocardiographic variables and in-hospital mortality

	Nonsurvivors (n = 70)	Survivors (n = 880)	P
Age (y)	65 \pm 11	59 \pm 11	<.001
Sex (male)	42 (60)	700 (80)	<.001
Hypertension	37 (59)	305 (35)	<.001
Diabetes mellitus	27 (43)	176 (20)	<.001
Previous myocardial infarction	6 (9)	44 (5)	.2
Anterior STEMI	43 (61)	445 (51)	.08
Heart rate (beats/min)	88 \pm 26	78 \pm 19	<.001
Systolic blood pressure (mm Hg)	92 \pm 31	120 \pm 26	<.001
Diastolic blood pressure (mm Hg)	55 \pm 23	76 \pm 15	<.001
LVEF (%)	36 \pm 11	45 \pm 9	<.001
Killip class			<.001
I	25 (36)	695 (79)	
II	27 (38)	158 (18)	
III	14 (20)	18 (2)	
IV	4 (6)	9 (1)	
ST \uparrow aVR \geq 0.5 mm	29 (41)	126 (14)	<.001
RBBB	15 (21)	46 (5)	<.001

Values are presented as mean \pm SD or n (%), where appropriate.

Table 8

The results of logistic regression analysis for the prediction of clinical outcome

Predictors	Odds ratio	95% Confidence interval		P
		Lower	Upper	
Killip class	2.535	1.328	4.837	.005
ST \uparrow aVR \geq 0.5 mm	3.981	1.518	10.441	.005
Diabetes mellitus	2.452	0.956	6.289	.062
Age	1.042	0.996	1.090	.072
Systolic blood pressure	0.981	0.963	0.999	.037
Ejection fraction	1.007	0.954	1.063	.800
Heart rate	1.007	0.986	1.029	.516

devised to explain the mechanism of ST \uparrow aVR in acute occlusion of LMCA or proximal LAD. One of these hypotheses is based on the fact that LMCA and LAD supply a large left ventricular area. Therefore, a sudden obstruction of the LMCA or proximal LAD induces an increase in left ventricular end-diastolic pressure, resulting in extensive ischemia of the subendocardium. The electrical vector is shifted from the epicardium toward the endocardium, causing ST depression with inverted T waves in the precordial leads. Lead aVR is electrically opposite to the lateral precordial leads. Thus, this ST depression in leads V₄ and V₅ induces reciprocal ST \uparrow aVR.¹⁶ In our study, history of hypertension, multivessel CAD, and acute occlusion of LMCA or proximal LAD were more prominent in the aVR (+) group, supporting this hypothesis. In another hypothesis, it is speculated that the basal region is the richest part of the heart in blood supply; therefore, ischemia of this region can be seen only in severe CAD. The basal part of the septum takes its blood supply through the septal perforator arteries of LAD, whereas infundibular septum is supplied by the RCA proximal conus branch. Some investigators presumed that ST \uparrow aVR was caused by the direction of electrical flow toward the right shoulder as a consequence of injury resulting from transmural ischemia in the basal part of septum because of LMCA and proximal LAD lesions.^{4,7} In our study, the finding that LAD occlusion in the site proximal to S₁ and distal to D₁ was more often observed in the aVR(+) group than in the aVR(-) group supports this theory. All these data suggest that ST \uparrow aVR may result from a complex mechanism, rather than from a simple one.

Evaluation of the extent of CAD

The presence of any lesion in LMCA or proximal LAD, whether or not it is IRA, may play a role in the choice of the type of revascularization procedure. Therefore, we described these lesions as “significant vessel disease,” no matter whether they are responsible for STEMI or not. Although there are data regarding the value of ST \uparrow aVR in patients with non-STEMI⁴⁻⁷ whose LMCA or proximal LAD is culprit lesion, there are few data evaluating the value of lead aVR in patients with STEMI. In this study, we investigated the value of lead aVR in predicting significant vessel disease in STEMI. We found that ST \uparrow aVR was suggestive of significant vessel disease with a sensitivity of 50% and specificity of 91%. In addition, ST-segment deviation in lead

aVR showed a weak but significant positive correlation with Gensini score, and this finding suggested that lead aVR was a predictor of the extent of CAD.

The effect of ST \uparrow aVR on prognosis

There are some studies investigating whether lead aVR was an indicator of prognosis during hospitalization in patients with non-STEMI. In early studies, ST \uparrow aVR was found to be related to a higher rate of in-hospital complications and mortality⁶; however, a recently published study revealed that ST \uparrow aVR was not an independent predictor of in-hospital and 6-month mortality in patients with non-STEMI.¹⁷ However, available information about the effect of ST \uparrow aVR on mortality is more limited in patients with STEMI. In 2 studies in which patients with inferior STEMI were evaluated, it was found that ST depression in lead aVR had no effect on prognosis during hospitalization.^{18,19} Nevertheless, Yamaji and colleagues⁷ reported that death occurred more frequently in patients with higher ST \uparrow aVR than in those with less severe elevation in acute LMCA obstruction. In our study, besides the other factors, aVR positivity was also found to be related to in-hospital death. Moreover, aVR positivity was an independent predictor of in-hospital mortality besides other factors, including Killip class^{20,21} and systolic blood pressure, that are well known to affect in-hospital mortality.²² This finding revealed that an ST \uparrow aVR of 0.5 mm or greater was not only a good predictor of LMCA or proximal LAD occlusion but also a source of valuable information on in-hospital prognosis.

Limitations

Although all the evaluators were blinded to clinical and angiographic data, ST-segment deviation in lead aVR followed by the observers was a potential source of errors. The analysis of ST \uparrow aVR is sometimes difficult because the change in its amplitude is small. Moreover, inability to determine the end of the QRS complex more precisely in lead aVR may affect the measurement of ST elevation. However, we showed that interobserver correlation coefficient and absolute agreement of ST analysis were high in our study. Another limitation of this study is that only patients who underwent coronary angiography were included. This is a study with a relatively small number of patients. Therefore, it will be useful if these findings are confirmed by other studies including more patients.

Conclusion

We concluded that an ST \uparrow aVR of 0.5 mm or greater not only determined proximal LAD occlusion in patients with anterior STEMI but also predicted LMCA or proximal LAD lesions in patients with any STEMI. We also concluded that an ST \uparrow aVR of 0.5 mm or greater might be an independent predictor of prognosis during hospitalization period.

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