ORIGINAL ARTICLES

Prediction of Left Main Coronary Artery Obstruction by 12-Lead Electrocardiography: ST Segment Deviation in Lead V_6 Greater than or Equal to ST Segment Deviation in Lead V_1

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Background: Acute coronary syndrome (ACS) resulting from culprit lesion in left main coronary artery (LMCA) can cause rapid hemodynamic deterioration. It is important to identify these patients early to facilitate timely revascularization. ST segment elevation in aVR greater than or equal to V_1 (aVR-$V_1 \geq 0$) has been suggested as a sensitive predictor of LMCA disease. As a result of balanced forces, we hypothesized that ST deviation in V_6 greater than or equal to ST deviation in V_1 (V_6-$V_1 \geq 0$) might be a good determinant of LMCA disease.

Methods: We compared admission 12-lead ECGs of ACS resulting from culprit LMCA lesion (n = 75, group I) with ACS resulting from culprit left anterior descending lesion (n = 81, group II). Group I was selected over a period of 10 years. We compared V_6-$V_1 \geq 0$ to aVR-$V_1 \geq 0$ in both groups. We also looked at ratios of ST deviations in V_6,V_1 ($V_6/V_1 \geq 1$) and aVR,V_1 (aVR/$V_1 \geq 1$) in patients where ST segment in V_1 was not isoelectric (group I = 54 and group II = 55).

Results: ST deviation in V_6 was significantly greater in group I as compared to group II ($P < 0.001$). The reliabilities of V_6-$V_1 \geq 0$, $V_6/V_1 \geq 1$, aVR-$V_1 \geq 0$, and aVR/$V_1 \geq 1$ in predicting LMCA disease were determined.

Conclusion: This is the largest series of ECG analysis on ACS resulting from culprit LMCA lesion. V_6-$V_1 \geq 0$ and $V_6/V_1 \geq 1$ were more sensitive in predicting LMCA as culprit vessel in comparison to previously reported greater ST segment elevation in aVR than V_1. **A.N.E. 2006;11(2):102–112**

left main coronary, reciprocal electrocardiographic changes, acute coronary syndrome

Left main coronary artery (LMCA) stenosis was first described by James Herrick in a patient with acute myocardial infarction in 1912.[1] LMCA disease is found in 3–5% of patients undergoing cardiac catheterization for ischemic chest pain, congestive heart failure, or cardiogenic shock.[2]

LMCA disease is the most critical coronary lesion and is associated with higher mortality as compared to patients with left anterior descending (LAD), left circumflex (LCX), or right coronary artery (RCA) disease.[3] Acute coronary syndrome (ACS) resulting from culprit lesion in LMCA causes rapid hemodynamic deterioration and carries grave prognosis. The American College of Cardiology guidelines consider group 11b/111a inhibitors and antiplatelet agents like clopidogrel as part of standard protocol of treatment for ACS.[4] The use of clopidogrel is associated with significantly higher bleeding and reexploration following coronary artery bypass graft (CABG) surgery.[5] Since the effect of clopidogrel

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lasts for a few days, its administration leads to a critical time lag between cardiac catheterization and CABG in these patients. Any delay in performing revascularization adversely affects the outcome in such patients. It is important to identify these patients as early as possible to withhold clopidogrel and facilitate timely revascularization that will impact prognosis.

Attempts have been made in the past to use the admission 12-lead ECG as a tool to identify patients with LMCA disease. Initial study failed to detect any significant historical, clinical, or ECG markers.[6] Subsequent studies pointed toward ST segment depression in both inferior and precordial leads,[7] diffuse ST segment depression,[8] ST segment depression with inverted T waves in leads V_4-V_6,[9] and ST segment elevation in aVR as possible markers of patients with LMCA disease.[10] ST segment elevation in lead aVR does not seem to be a specific marker for LMCA stenosis and is also seen in patients with proximal LAD and triple vessel disease.[11] Diffuse ST segment depressions can be seen in ACS resulting from additional lesions in multiple epicardial coronary vessels.

LMCA disease is frequently accompanied by significant involvement of other epicardial vessels.[12,13] It is an infrequent entity, and studies thus far have been limited to small numbers. The largest sample of patients with LMCA disease used in the retrospective studies to date has been 16.[7,8,14–16]

ACS resulting from culprit lesions in LMCA and LAD vessels generally produces anterior wall ischemia resulting in ST segment deviations in anterior precordial leads. LMCA disease also causes ischemia in LCX territory. Hence, LMCA disease may cause reciprocal changes in anterior precordial leads resulting from posterior wall ischemia caused by impaired blood supply to LCX territory. This balancing of forces may result in ST segment deviations in the anterior precordial leads caused by LMCA disease being different from those produced by LAD disease alone. We hypothesized that ST segment deviation in lateral lead V_6 would be greater than or equal to ST segment deviation in anterior lead V_1 (V_6-$V_1 \geq 0$) in patients with ACS resulting from culprit LMCA lesion as a result of less counterbalancing in lateral leads. We looked at using the ratio of absolute ST segment deviations in leads V_6, V_1 ($V_6/V_1 \geq 1$) as possible predictor of LMCA disease in patients where ST segment in V_1 is not isoelectric.

It has been suggested that ST segment elevation in aVR greater than or equal to ST segment elevation in V_1 predicts LMCA disease.[16] Therefore, we also looked at the reliability of ST segment deviation in aVR greater than or equal to V_1 (aVR-$V_1 \geq 0$) in predicting LMCA disease and at using the ratio of absolute ST segment deviations in leads aVR,V_1 (aVR/$V_1 \geq 1$) as possible predictor of LMCA disease in patients where ST segment in V_1 is not isoelectric.

Since ST depression with T inversion in V_4-V_6 has been suggested as a predictor of LMCA disease,[9] this study looked at the prevalence of T inversion in lateral leads in group I.

METHODS

We reviewed the database of cardiac catheterizations done at our Cardiac Institute from January 1994 to December 2003. This involved a review of a total of 44,320 cardiac catheterizations performed at our catheterization center. It led to the identification of 1850 patients with significant LMCA disease accounting for 4.17% of patients who underwent cardiac catheterization over the period of 10 years.

The angiograms of these 1850 patients were reviewed independently by 2 experienced cardiologists and patients with culprit LMCA lesions were identified. The culprit lesion was defined when lesion was ≥50% and associated with thrombus or thrombolysis in myocardial infarction flow grade 1. The culprit lesion was defined purely on the basis of angiography in all the selected patients with the reviewers being unaware of any ECG findings. Patients with angiographically significant lesions in other coronary vessels (defined as percent diameter stenosis ≥70% of luminal diameter of LAD, LCX, or RCA) were subsequently excluded from further data analysis. This was done in order to avoid the confounding effects of the lesions in coronary vessels other than the LMCA on 12-lead ECG. This led to the identification of 98 patients with significant LMCA disease and insignificant other vessels disease, accounting for 5.3% (98/1850) of patients with significant LMCA disease.

Patients with bundle branch block, paced rhythm, and presenting complaint other than chest pain were excluded. The remaining 75 patients formed group I. Group I was further subcategorized into groups IA and IB. Group IA consisted of patients with pure LMCA disease without angiographically visible lesions in other major epicardial

vessels. Group IB consisted of patients with significant LMCA lesion and subcritical (percent diameter stenosis <70% of luminal diameter) lesions in other major epicardial vessels.

The control group was also retrospectively chosen. Group II comprised 81 consecutive patients who presented with ACS resulting from isolated and significant LAD lesion (without angiographically visible lesion in LMCA). This group was chosen in view of the fact that lesions in LAD are more frequent in common practice than other coronary artery diseases and present with ST-T changes in precordial leads including ST changes in precordial leads and ST elevation in aVR.

Myocardial infarction was defined by the presence of ECG changes associated with typical chest pain and elevated cardiac enzymes. These enzymes were measured at 6- to 8-hour intervals during the first 24 hours. The cardiac enzymes were considered elevated if Troponin I or both creatine kinase (CK) and its MB isoenzyme (CK-MB) were greater than two times the upper normal limit. Unstable angina was defined by the presence of typical anterior chest pain with ECG changes in the absence of elevated cardiac enzymes.

Demographic data were collected from patient's medical records. In this retrospective study, we analyzed and compared the 12-lead electrocardiogram at admission of patients with ACS resulting from culprit lesions in LMCA and LAD vessels by using single and multivariate analyses. For comparison of ST segment deviations, the absolute magnitude of ST segment deviations in leads V_6, aVR, and V_1 were used regardless of the direction of ST segment deviation. Because deviations in opposing myocardial segments are localization determinants, absolute deviations rather than arithmetic sums are of necessity used.

ECG Analysis

Investigators blinded to the angiographic findings of study population and to the clinical outcome analyzed the 12-lead ECG recorded on admission at presentation to the emergency room. ST segment elevations were measured at J + 20 ms, while ST depressions were measured at J + 80 ms using preceding TP segment as the baseline. In patients with tachycardia (defined as ventricular response >100), ST segment depressions were measured at J + 60 ms while ST elevation was measured at J + 20 ms. Left bundle branch block was defined as the presence of monophasic QRS complexes, with QS complexes in V_1 and R waves in leads I, aVL, and V_6.[16] Right bundle branch block was defined as the presence of RSR in lead V_1 with widening of S wave in V_1.[17] Anterior, inferior, and lateral ST segment deviations were defined as ST segment shifts in ≥2 leads oriented anteriorly (V_1, V_2, V_3, and V_4), laterally (1, aVL, V_5, and V_6), or inferiorly (II, III, and aVF), respectively. T wave changes, QRS axis, and QT intervals were also determined. The data for ST segment deviations were subjected to statistical analysis. All the ST changes and T wave magnitudes were measured using calipers with a precision of ±0.02 mm.

Initially, interobserver and intraobserver variations were checked using 20 randomly selected ECG samples, and measurements performed by 2 observers blinded to the results of angiography.

Statistics

Unless otherwise indicated, data are expressed as the mean value ± SD. Statistical analyses were conducted with SPSS Version 11.5 and GB-STAT (Dynamic Microsystems, Inc., Silver Springs, MD). For univariate analysis, the unpaired Student's *t* test was used to compare the differences between ST segment deviations (regardless of direction of ST segment deviation) for the two groups. A stepwise logistic regression was done to identify the ST segment deviations that best predict membership in one of the two groups. Among inferior leads, aVF was selected for the logistic regression to avoid multicollinearity of the variables as leads II, III, and aVF showed similar prevalence of abnormalities on univariate analysis. For similar reasons, leads V_1 and V_6 were selected among precordial leads and lead aVL among lateral leads. The independent variables used in this study were ST segment deviations in leads aVR, aVL, aVF, V_1, and V_6. A P value <0.05 was considered significant.

RESULTS

Baseline Characteristics

The comparison of demographic and atherosclerotic risk factor profile showed significantly higher baseline heart rate and a trend toward older males, a higher risk profile (diabetes, hypertension, and smoking) in group I (Table 1). The mean time from symptom onset to angiogram was 8 hours and 7 hours in groups I and II, respectively. The

Table 1. The Comparison of Demographic Profile of the Two Study Groups

	Variable	Group I	Group II	P Value
1	Age (in years)	Males = 67 ± 17; Females = 68 ± 14	Males = 60 ± 14; Females = 72 ± 9	Nsd[a]
2	Gender	Males = 40 (53%); Females = 35 (47%)	Males = 55 (68%); Females = 26 (32%)	Nsd
3	Risk factors			
	Hypertension	60 (83%)	57 (70%)	Nsd
	Hypercholesterolemia	48 (64%)	55 (68%)	Nsd
	Diabetes mellitus	30 (40%)	24 (30%)	Nsd
	Smoking	21 (28%)	19 (23%)	Nsd
	Family history	24 (32%)	28 (35%)	Nsd
4	Clinical presentation	Angina = 51 (68%); Myocardial infarction = 24 (32%)	Angina = 49 (61%); Myocardial infarction = 32 (39%)	Nsd
5	Heart rate (bpm[b])	90 ± 10	80 ± 11	<0.001
6	Systolic blood pressure (mmHg)	136 ± 28	130 ± 24	Nsd

[a]Nsd = not statistically significant; [b]bpm = beats per minute.

angiography characteristics of the study groups are shown in Table 2.

Electrocardiographic Analysis

In group I, 69 patients had normal sinus rhythm and 5 patients had atrial fibrillation (ventricular rate below 100). All the patients in group II were in normal sinus rhythm on presentation to hospital. The representative 12-lead electrocardiograms at admission of the two groups are shown in Figure 1.

When evaluating the sample ECGs, the interobserver and intraobserver differences averaged 0.01 ± 0.02 mV and 0.01 ± 0.02 mV, respectively. Therefore, the intraobserver and interobserver

Table 2. The Angiographic and Echocardiographic Findings of Study Groups

	Variable	Group I	Group II	P Value
1	Stenosis (%)			–
	50–60%	21 (28%)	0	
	60–80%	22 (29%)	16 (20%)	
	≥80%	32 (43%)	65 (80%)	
2	Lesion location			–
	Distal	40 (54%)	7 (9%)	
	Ostial	28 (37%)	0	
	Proximal	4 (5%)	22 (27%)	
	Middle	3 (4%)	52 (64%)	
3	Segmental wall motion			–
	Normal	36 (49%),	44 (54%)	
	Anterolateral	25 (34%),	30 (37%)	
	Apical	25 (34%)	30 (37%)	
	Diffuse	15 (20%)	6 (7%)	
	Diaphragmatic	20 (27%),	19 (23%)	
	Posterior wall	20 (27%)	6 (7%)	
4	Left ventricular ejection fraction	44 ± 15	48 ± 14	Nsd[a]

Echocardiography (available for 39 patients in group I); mitral regurgitation 33 (85%); tricuspid regurgitation 24 (62%); aortic regurgitation 13 (33%); pulmonary regurgitation 9 (23%); aortic stenosis 8 (20%); left atrial enlargement 25 (64%); left ventricular enlargement 10 (26%); right atrial enlargement 7 (18%); RVE right ventricular enlargement 1 (3%).
[a]Nsd = not statistically significant.

variations did not affect the validity of the results. Figure 2 shows the magnitude of actual ST segment deviations on 12-lead electrocardiogram in the two groups. There is a trend toward ST segment depression in leads V_4, V_5, V_6, I, II, aVL, and ST elevation in lead aVR in group I. Patients in group I had significantly higher prevalence of ST elevation in lead aVR and ST segment depression in lateral and inferior leads (Fig. 2). The mean ST segment deviation in lead V_6 is greater than mean ST deviation in lead V_1 in group I. The mean ST elevation in lead aVR is greater than the mean ST elevation in lead V_1 in group I.

The incidences of ST segment deviations ($\geq$0.05 mV) in leads V_6 (65% vs 35%) and aVR (62% vs 33%) are significantly higher in group I.

The magnitude of absolute ST segment deviations (regardless of direction of ST segment deviation) on 12-lead electrocardiogram in groups I and II is shown in Figure 3. There is significantly greater ST segment deviation in leads V_5, V_6 as compared to ST segment deviation in lead V_1 in group I.

There were no significant differences between the mean magnitudes of T waves on 12-lead ECG in the study groups. However, T wave inversions in lateral leads were common in group I. In group I, T inversion was most common in lead aVL (44/75; 59%). T inversions were also seen in lead I (38/75; 51%), V_5 (35/75; 47%), and V_6 (35/75; 47%). Overall, lateral T inversions (defined as T inversion in $\geq$2 leads out of lateral leads I, aVL, V_5, and V_6) were seen in 44 (59%) patients in group I.

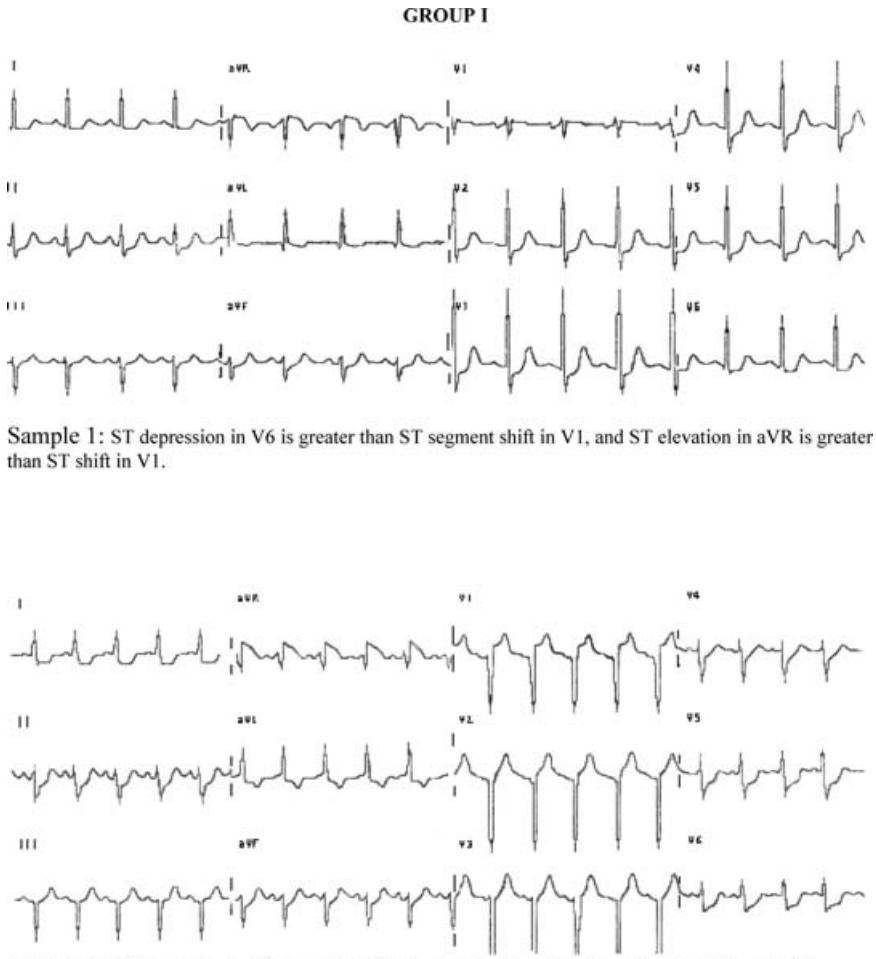
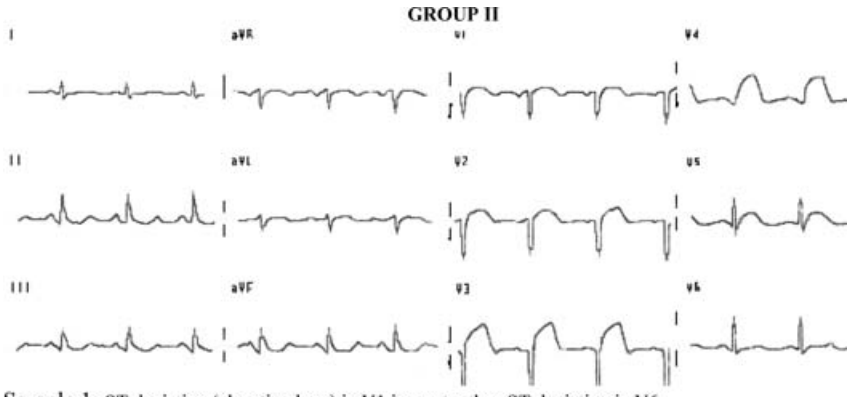
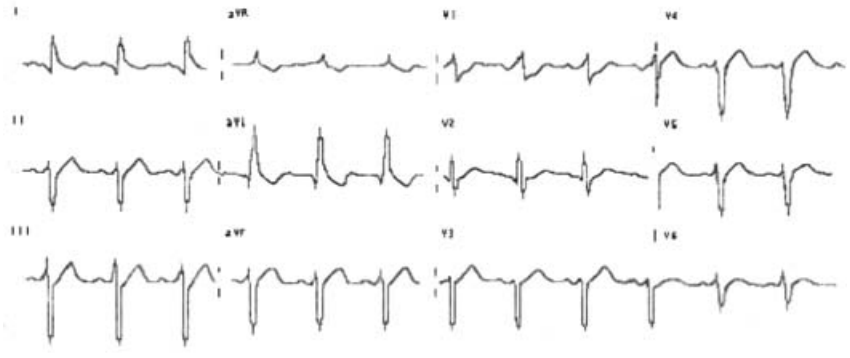


Figure 1. Representative 12-lead electrocardiogram tracings at admission in the two groups.



Sample 1: ST deviation (elevation here) in V1 is greater than ST deviation in V6.



Sample 2: ST deviation (depression here) in V1 is greater than ST deviation in V6.

Figure 1. Continued.

Table 3 summarizes the results of univariate comparison among ST segment deviations in leads V_6, V_1, and aVR between group I and group II. Statistically significant differences were found for leads V_6 and aVR ($P < 0.001$) but not for lead V_1. ST segment deviation in V_6 was greater than ST segment deviation in lead V_1 in group I and vice-versa in group II. A *t*-test was done to determine whether there was a difference between ST segment deviations in leads V_1 and V_6. The results are statistically significant (Table 4). ST segment deviation in V_6 is significantly greater than ST segment deviation in V_1 in group I ($P < 0.001$). In group I, the mean ST segment deviation in lead aVR is greater than mean ST segment deviation in lead V_1. In group II, ST segment deviation in lead aVR is less than ST segment deviation in V_1. To determine whether there is a difference between groups for the ratios of ST deviations in leads V_6,V_1 (V_6/V_1) and leads aVR,V_1 (aVR/V_1), a *t* test was conducted to assess the difference between the ratios. We also calculated the reliability of $V_6/V_1 \geq 1$ and aVR/$V_1 \geq 1$ for predicting LMCA disease in patients where ST segment in V_1 was not isoelectric. The results (Table 5) are statistically significant ($P < 0.001$ for both analyses). The electrocardiographic findings are similar in subgroups IA and IB.

The reliability of the different criteria (V_6-$V_1 \geq 0$, aVR-$V_1 \geq 0$, $V_6/V_1 \geq 1$, and aVR/$V_1 \geq 1$) for predicting LMCA disease is shown in Figure 4. The criteria using ST segment deviation in V_6 greater than or equal to ST segment deviation in V_1 (V_6-$V_1 \geq 0$) had 81% sensitivity, 57% specificity, and 64% accuracy for predicting LMCA as culprit vessel. The finding of ST deviation in aVR greater than or equal to ST deviation in V_1 (aVR-$V_1 \geq 0$) had 75% sensitivity, 59% specificity, and 63% accuracy for predicting LMCA as culprit vessel. The criterion using $V_6/V_1 \geq 1$ was associated with a sensitivity of 74%, a specificity of 89%, and an accuracy

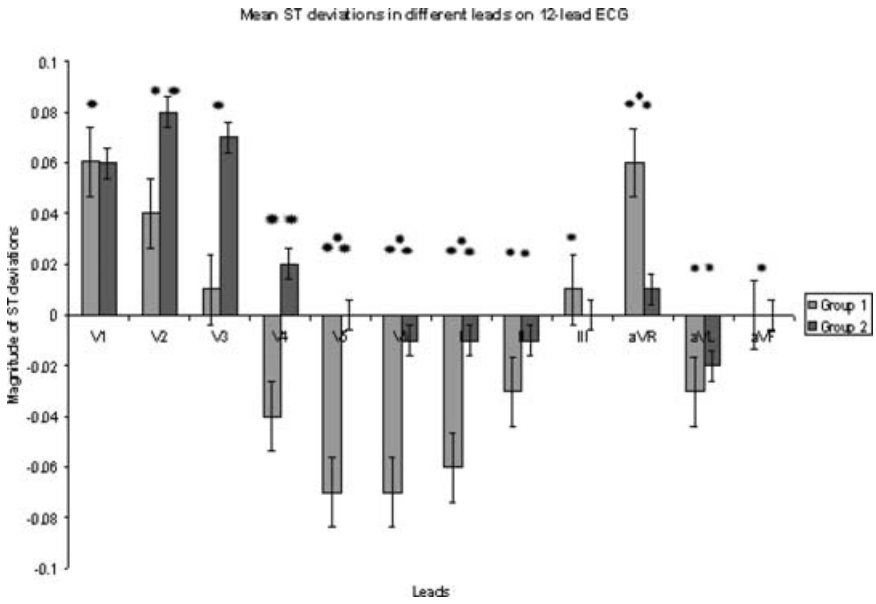


Figure 2. Mean of actual ST segment deviations (mean ± standard error) in the two groups (*P = not significant, **P < 0.05, ***P < 0.0001).

of 82%. The finding of aVR/$V_1 \geq 1$ had a sensitivity of 63%, a specificity of 89%, and an accuracy of 85%.

The stepwise logistic regression (shown in Table 6) involving leads V_1, V_6, and aVR led to a model that successfully discriminated group I from group II with a sensitivity of 75%, a specificity of 74%, and an accuracy of 74% (shown in Table 7).

DISCUSSION

This study showed that greater ST segment deviation in lead V_6, compared to ST segment deviation in lead V_1, was a useful predictor of ACS resulting from culprit LMCA lesion. It (V_6-$V_1 \geq 0$ and $V_6/V_1 \geq 1$) is more sensitive in predicting LMCA disease than previously reported aVR$\geq V_1$.

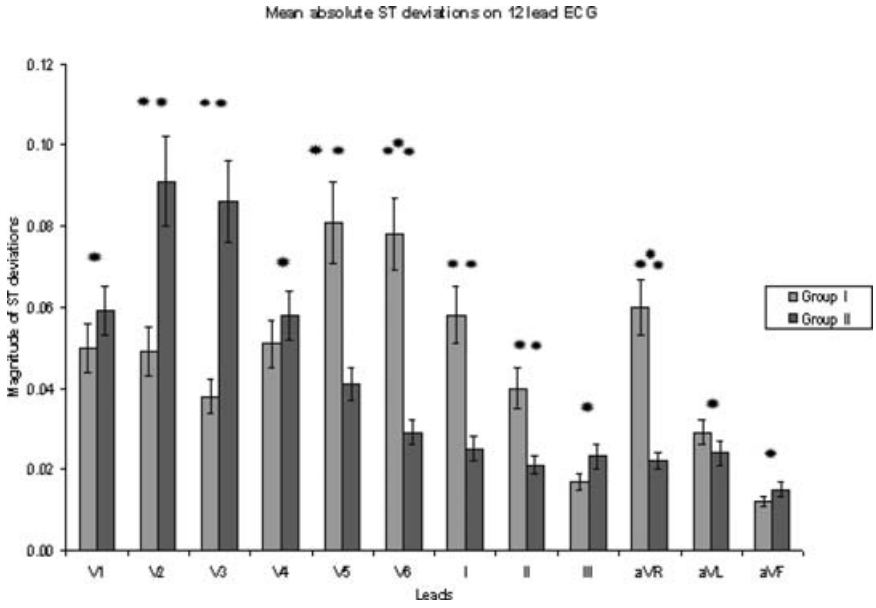


Figure 3. Mean of absolute ST segment deviations (mean ± standard error) in the two groups (*P = not significant, **P < 0.05, ***P < 0.0001).

Table 3. Results of Univariate Analyses Assessing the Difference Between ST Segment Deviations in Selected Leads for the Two Groups

	Mean ST Deviation		Unpaired *t*-Test for Equality of Means					
							95% Confidence Interval of the Difference	
Lead	Group I	Group II	*t*	P (2-Tailed)	Mean Difference	Standard Error Difference	Lower	Upper
V_1	0.057	0.059	−0.169	0.866	−0.002	0.009	−0.020	0.017
V_6	0.074	0.029	5.629	0.000	0.045	0.008	0.029	0.061
aVR	0.059	0.022	5.687	0.000	0.037	0.006	0.024	0.050

ST Segment Deviation in Lead V_6 Versus Lead V_1

Disruption of blood supply to the LAD causes ischemia in the anteroseptal wall. This ischemia manifests on a 12-lead ECG as ST elevation in anterior precordial leads with reciprocal ST depression in inferior leads (total occlusion) or ST depression in anterior precordial leads with reciprocal ST elevation in posterior leads (subtotal occlusion). LMCA obstruction causes reciprocal changes in anterior precordial leads resulting from posterior and lateral wall ischemia (LCX territory), and this tends to counteract the ST segment deviations in anterior precordial leads produced by ischemia in the LAD territory. The etiology of reciprocal electrocardiographic changes has always been a matter of debate. Reciprocal electrocardiographic changes are postulated to result from (a) ischemia of the opposite wall; (b) electrophysiologic phenomenon in which the ST depression is the "mirror-image" of the classic ST elevation; and (c) extension of the infarct beyond the territory of the culprit vessel.[18] Many studies to date have provided evidence in support of ischemia of opposite wall and "mirror-image" phenomenon.[19–21]

ST Segment Elevation in Lead aVR

The present study found lead aVR ST segment elevation in 62% (46/75) patients in group I. By convention, lead aVR is considered a negative lead that reflects the inverse of changes in other leads (ST segment elevation is analogous to ST segment depression in other leads). The electric current in ST elevation of aVR is directed toward the right shoulder.[22] ST segment elevation in aVR is not specific for acute LMCA occlusion. Soler-Soler et al. found that ST segment elevation in aVR $\geq$0.5 mm correlated with culprit lesion in LMCA in only 8% of patients in their study population.[11] In acute LMCA occlusion, ST segment elevation in aVR is reflective of transmural ischemia of the basal part of interventricular septum (due to the dominance of basal ventricular mass),[22,23] or it may be reflective of ST segment depression in lateral leads.[9]

Our study found greater ST segment deviation in aVR compared to V_1, a useful predictor of LMCA disease, supporting the findings of Yamaji et al.[16] We found a significantly greater ST segment deviation in V_6 as compared to ST segment deviation in V_1 in patients with ACS from culprit LMCA lesion. The finding of ST deviation in V_6 greater than or

Table 4. Results of *t*-Test to Check Equality of Means of ST Segment Deviations in Leads V_1 and V_6 in Group I

	Mean ST Deviation Difference		Paired *t*-Test for Equality of Means					
							95% Confidence Interval of the Difference	
ST Deviation Difference	Group I	Group II	*t*	P (2-Tailed)	Mean Difference	Standard Error Difference	Lower	Upper
V_1-V_6	−0.017	0.030	−4.881	0.000	−0.046	0.010	−0.065	−0.028

Table 5. Results of Univariate Analyses Assessing the Ratios Between ST Segment Deviations in Selected Leads for the Study Groups

	Mean Ratio of ST Deviation		Paired *t*-Test for Equality of Means					
							95% Confidence Interval of the Difference	
Ratio of ST Deviation	Group I N = 54	Group II N = 55	*t*	P (2-Tailed)	Mean Difference	Standard Error Difference	Lower	Upper
V_6/V_1	1.362	0.406	6.074	0.000	0.955	0.157	0.644	1.267
aVR/V_1	0.992	0.301	5.968	0.000	0.691	0.116	0.462	0.921

equal to ST deviation in V_1 had higher sensitivity, as compared to using ST deviations in aVR and V_1, and comparable specificity and accuracy for predicting LMCA as culprit vessel.

Nikus et al. have reported that ST depression with T inversion in V_4-V_6 is a predictor of LMCA disease.[9] Our study found T inversions in lateral leads in 44 (59%) patients in group I. This finding may help in detecting a subgroup of patients with ACS from culprit LMCA lesion.

CONCLUSIONS

This is the largest published series of ECG analysis in patients with ACS resulting from isolated significant LMCA disease. This study shows that the majority of patients with culprit LMCA lesions present with angina followed by MI. Focusing on ST-T changes in lateral leads with respect to anterior precordial leads may help in identifying such patients, as will greater ST segment elevation in lead aVR compared to ST segment elevation in V_1. ST segment deviation in V_6 greater than or equal to ST segment deviation in V_1 (V_6-$V_1 \geq 0$ and $V_6/V_1 \geq 1$) is more sensitive in predicting LMCA as culprit vessel than comparing ST changes in aVR and V_1. The use of ratio of $aVR/V_1 \geq 1$ as predictor of LMCA disease is associated with best specificity of 89%. The model looking at ST segment deviations in V_1, V_6, and aVR gives a sensitivity of 75%, a specificity of 74%, and an accuracy of 74%. This is a retrospective study. The results need to be validated in a prospective study.

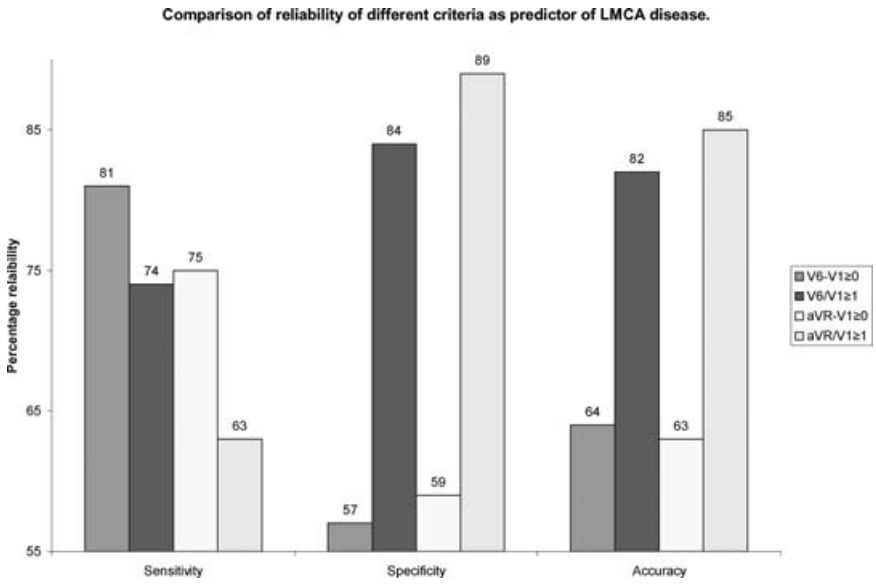


Figure 4. The comparison of the reliability of different criteria for predicting LMCA disease* (*Accuracy—positive predictive value).

Table 6. Results of Stepwise Logistical Regression with Group as Criterion

				95% Confidence Interval	
Lead	**Beta**	**P (2-Tailed)**	**Standard Error**	**Lower**	**Upper**
V_1	11.3615	0.004	3.901	3.654	19.069
V_6	−15.7063	0.003	5.252	−26.083	−5.329
aVR	−18.4264	0.002	6.010	−30.301	−6.552

Limitations

The angiographic determination of degree of narrowing of LMCA as well as other major epicardial arteries is prone to intraobserver and interobserver errors.[25] All the films were reviewed in multiple projections to minimize this error. It is hoped that careful patient selection and analysis by multiple methods may have partly compensated for this limitation. All the cases with discordant evaluation by reviewing cardiologists on the extent and severity of LMCA lesion were excluded. The limitations of angiography in the determination of the degree of LMCA narrowing have been confirmed by recent studies documenting the presence of significant left main atherosclerosis by intravascular ultrasound, despite a minimal or insignificant angiographic appearance.[26] The cases in group 1 were selected retrospectively over a period of 10 years. The strict inclusion criteria alone can be a source of selection bias, even though adequate steps were taken to blind the reviewers to ECG findings at the time of reviewing the angiograms. It remains to be seen whether these ECG changes are also seen in patients with left main equivalent disease. The influence of magnitude of ST segment deviation in lateral leads on mortality needs to be ascertained, although published literature suggests higher in-hospital mortality in patients with ST depressions in lateral leads.[27]

Table 7. Prediction of Groups Using Leads V_1, V_6, and aVR as Multivariate Predictors

Observed		**Predicted Group**	
Group I vs Group II		I	II
	I	52	23
	II	17	64
Overall accuracy			

Sensitivity = 75%; Specificity = 74%; and Accuracy (positive predictive value) = 74%.

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