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Full Length Research Paper

ST Segment Elevation in Leads V5 and V6 for Predicting Culprit Artery in Acute Inferior Wall Myocardial Infarction

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ST elevation (ST↑) in the leads V5 and V6 to predict the culprit and furthermore, comparing the degree of ST ↑ in lead V6 with that in lead III. Patients were first divided according to the presence (n-62) and absence (n-98) with ST ↑ of ≥2 mm in leads V5 and V6, the patients were then further subdivided into the 2 groups according to the degree of ST ↑ in lead III and in lead V6 and were compared with coronary angiography. In patients with ST ↑ in leads V5 and V6, the infarct related artery was right coronary artery (RCA) in 66% and left circumflex artery (LCx) in 31%. The RCA was found significantly higher proportion in ST ↑ in leads V5 and V6 with ST ↑ in lead III>V6 (n-37; 82%) (*p=0.0001)*, whereas the LCx disease in ST ↑ in leads V5 and V6 with ST ↑ in III≤V6 (n-12; 71%) (*p-0.0001)*. The sensitivity, specificity, positive and negative predictive value in ST ↑ in leads V5 and V6 with ST ↑ in lead III>V6 for RCA, and ST ↑ in lead III≤V6 for LCx to predict culprit artery were 90%, 63%, 84%, 75% and 63%, 90%, 75%, 84% respectively. ST elevation in leads V5 and V6 suggests a greater risk area in patient with acute inferior wall myocardial infarction. It also signifies a larger perfusion territory and demands for more aggressive reperfusion therapy.

Keywords: Infarct related artery (IRA); ST elevation myocardial infarction (STEMI); Right coronary artery (RCA); Left circumflex artery (LCX); Electrocardiogram (ECG).

INTRODUCTION

The inferior wall and inferolateral wall of the left ventricle are supplied by the right coronary artery (RCA) and left circumflex (LCx) artery. Inferior wall acute myocardial infarction (I-AMI) is caused by an occlusion of either the RCA or LCx. Identification of RCA occlusion is very important because it is associated with the right ventricle extension and the clinical syndrome of hypotension and shock (Braat Set al., 1984). Multiple ECG criteria have been studied to predict the culprit artery based on ST segment elevation and ST segment depression in different leads (Kosuge et al., 1998; Chia et al., 2000; Herz et al., 1997; Zimetbaum et al., 1998; Bairey et al., 1987; Hasdai et al., 1995; Assali et al., 1999; Fiol et al.,

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2004; Fiol et al., 2004). The present study examines an electrocardiographic algorithm based on ST segment changes in leads V5 and V6 that has a very high sensitivity, specificity and predictive value to predict the culprit artery in an inferolateral acute myocardial infarction.

METHODS

The present study was approved by our local hospital Institutional Review Board (IRB). We retrospectively analyzed 390 patients having an acute inferior wall MI, admitted and underwent coronary angiography in the cardiovascular department of our hospital within a year-period. Only 160 patients (male 132 and female 28) met our inclusion criteria of chest pain for ≥ 30minutes before hospital admission. If the elevation of creatinine kinase (CK-MB) is greater than twice the upper limit (Normal:0-3.5 ng/ml), the ECG shows ST segment ≥0. 1mV (1mm) in at least 2 of 3 inferior leads (II, III, aVF). Coronary angiography showing total occlusion or critical stenosis >70% in single vessel either RCA or LCX. Whereas, exclusion criteria include lack of ST elevation ≥0.1 mV (1mm) in the inferior leads (II, III, aVF), previous history of acute myocardial infarction, coronary artery bypass surgery or percutaneous coronary intervention prior to current hospitalization, evidence of recent left bundle branch block or left ventricular hypertrophy on ECG, and significant stenosis in both LCX and RCA or triple vessel disease so that a single infarct related artery could not be defined. A 12 leads ECG was recorded in all patients at a paper speed of 25mm/s and voltage 10mm/mV. The ST-elevation deviation was measured manually to the nearest 0.5mm, 80ms after the J point by a single cardiologist who was unaware of all clinical and angiographic findings. ST segment depression in aVR of at least 0.1mV (1mm) was the most important criterion to distinguish between LCx and RCA as presumptive predictor of the culprit artery. Coronary angiography was performed during hospitalization in all patients. The results of this test were compared with those obtained from ECG to distinguish the real culprit artery. Data are expressed as the mean ± SD for continuous variable and percentages for categorical variables. Statically significance ($p<0.05$) was assessed by chi-square test or fisher exact test for categorical variables. The sensitivity, specificity, positive predictive value and negative predictive value were evaluated in the 2 coronary arteries.

RESULTS

A total of 160 patients (male 132 and female 28) was included in the study. Patients were divided according to the presence (n -62) and absence (n -98) of ST elevation of ≥2 mm in leads V5 and V6 on admission. Patients with ST elevation in leads V5 and V6 were further subdivided into the 2 groups according to the degree of ST elevation in lead III and in lead V6. Patients with ST elevation in lead III greater than in V6 were 45 (73%), and ST elevation in lead III equal to or less than in V6 were 17 (27%) as shown in the Figure 1.2. The baseline characteristics of groups are summarized in the Table 1.1. There were no significant differences between 3 groups in baseline clinical characteristics and prevalence of risk factors. ST elevation in leads II, III and αVF with ST elevation in leads V5 and V6 was more common in male than in female (81% vs 19%).

The clinical, electrography and echocardiography findings on admission are listed in the Table 1.2. ST elevation in leads V5 and V6 with ST elevation in lead III>V6 was associated with greater ST elevation in leads II, III and αVF, in particular lead III. In the ECG of ST elevation in inferior leads (II, III and αVF), ST elevation in lead III>II was frequently associated with ST elevation in leads V5 and V6 and ST elevation in lead III>V6 than in the ST elevation in leads V5 and V6 with ST elevation in lead III ≤ V6 (84% vs 53%)(*P=0.027*), whereas ST elevation in lead II >III was more associated with ST elevation in leads V5 and V6 with ST elevation in lead III ≤V6 than in the ST elevation in V5 and V6 with ST elevation in lead III > V6(46% vs 15%)(*P=0.027)* as shown in the Table 1.2. However, ST segment depression in V1-V3 was greater in both patients with ST elevation in leads V5 and V6 with ST elevation in lead III >V6 and ST elevation in leads V5 and V6 with ST elevation in lead III≤V6.

There was no much difference in creatinine kinase (CK-MB) and ejection fraction between three groups. Coronary angiography findings are listed in the Table 1.3. Among 62 patients with ST elevation in leads V5 and V6, the infarct related artery was right coronary artery in 41 patients (66%) and left circumflex artery in 19 (31%). No ST elevation in leads V5 and V6 and ST elevation in leads V5 and V6 with ST elevation in lead III >V6 groups were strongly associated with RCA occlusion; 73% and 82% respectively. However, ST elevation in leads V5 and V6 with ST elevation in lead III ≤ V6 was associated with LCX; 71% occlusion. TIMI 0 coronary flow was found in 145 patients (91%). No differences were found between

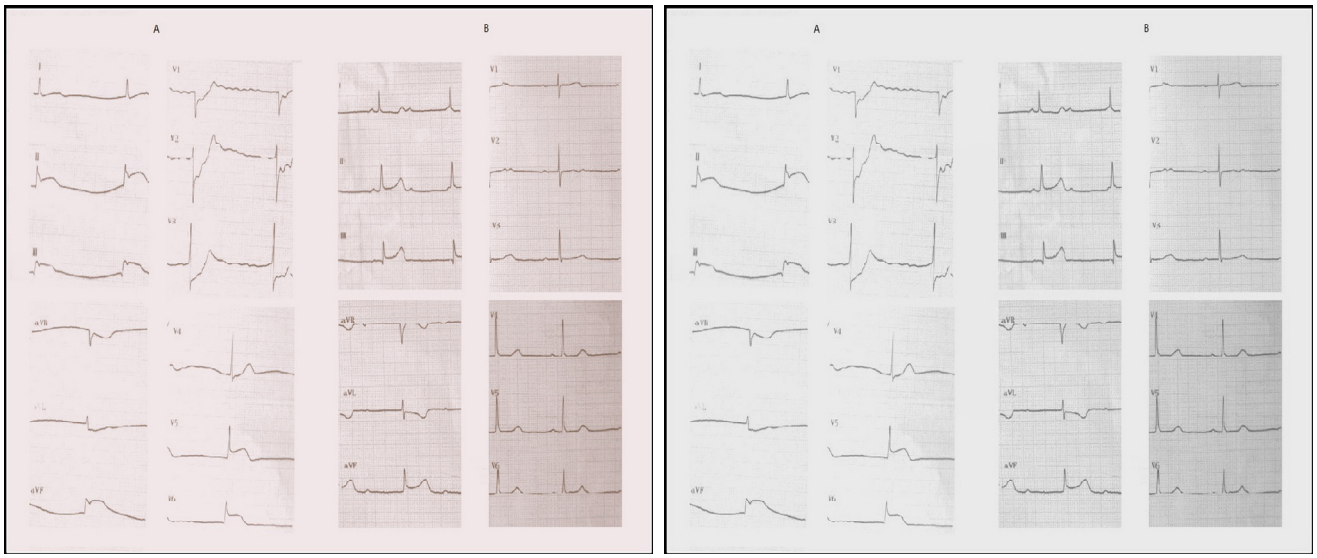

Figure 1.1 (A). Electrocardiogram of a patient with Inferior wall acute myocardial infarction with ST-segment elevation in leads II, III and aVF and in precordial leads V5 and V6 (≥2mm). ST segment elevation in leads V5 and V6 with ST elevation in lead III greater than V6. The coronary angiography later confirmed right coronary artery. (B) Electrocardiogram of a patient with Inferior wall acute myocardial infarction with no ST-segment elevation in precordial leads V5 and V6.

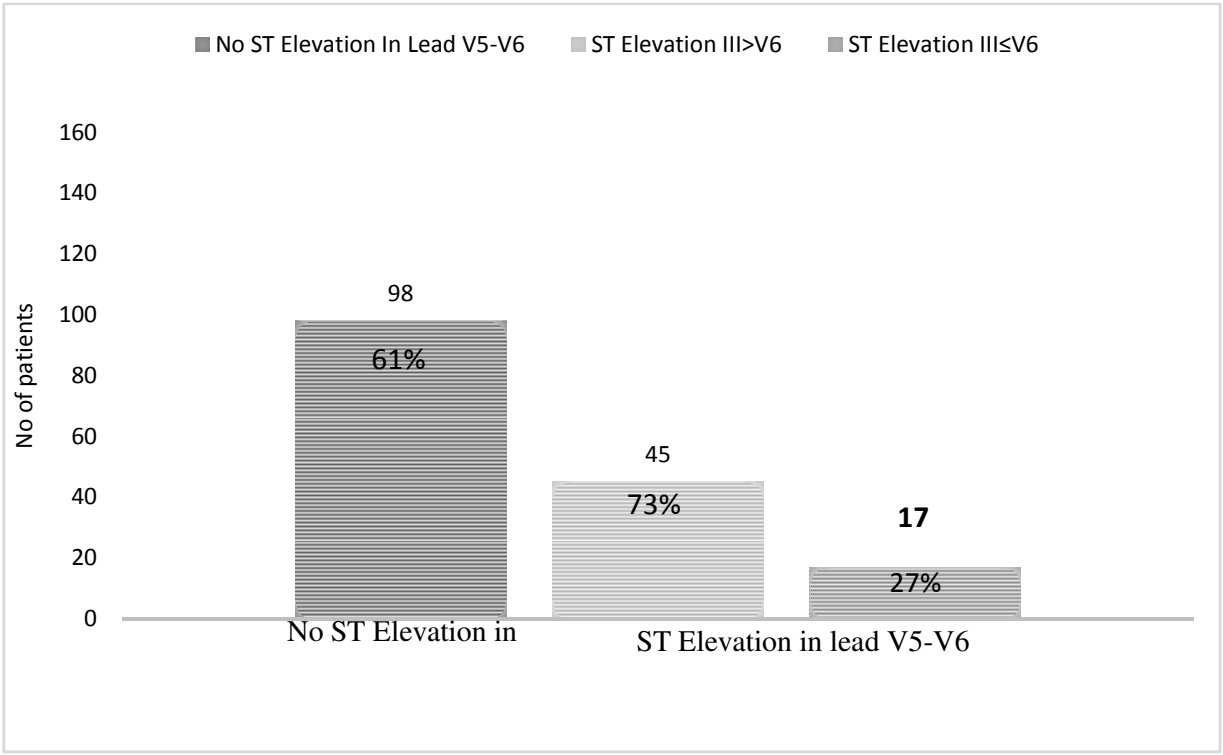

Figure 1.2. Shows no. of patients in inferior wall myocardial infarction with and without ST segment elevation in leads V5-V6.

Table 1.2. Clinical, Biochemical, ECG and ECHO

Variable	No ST ↑ in Lead V5-V6 (n-98)	ST ↑ in lead V5-V6		*p* value
		ST ↑ in lead III>V6 (n-45)	ST ↑ in lead III≤ V6 (n-17)	
KILIP 1	93 (94%)	41 (91%)	17 (100%)	0.37
ST ↑ in				
II>III	21 (21%)	7 (15%)	8 (47%)	0.027*
III>II	77 (79%)	38 (84%)	9 (53%)	0.027*
ST in lead V1-V3				
ST depression	36(37%)	32 (71%)	12 (70 %)	0.0001***
Ejection Fraction	55.78±7.33	54.15±6.41	54.35±6.24	0.091
CKMB	42.26±40.69	52.43±33.05	59.35±27.61	0.178

Table 1.3. Coronary angiography (CAG)

Variable	No ST ↑ in Lead V5-V6 (n-98)	ST ↑ in lead V5-V6		*P* value
		ST↑ in lead III>V6 (n-45)	ST↑ in lead III≤ V6 (n-17)	
RCA	72 (73%)	37 (82%)	4 (23%)	<0.0001***
LCX	22 (22%)	7 (16%)	12 (71%)	< 0.0001***
MULTIVESSELS	4 (4%)	1 (2%)	1 (5%)	0.76
TIMI 0	87 (89%)	41 (91%)	17 (100%)	0.33

Table 1.4. According to Coronary angiography; to predict the culprit artery in ST ↑ in lead V5-V6 in related to ST ↑ in lead III>V6 and ST ↑ in lead III≤ V6 (RCA or LCX): Sensitivity, Specificity, PPV and NPV

ECG	CAG	Sensitivity	Specificity	PPV	NPV
ST ↑ in lead III>V6	RCA	90%	63%	84%	75%
ST↑ in lead III≤ V6	LCX	63%	90%	75%	84%

PPV-Positive Predictive Value, NPV-Negative Predictive Value, CAG-Coronary angiography.

three groups regarding TIMI O flow score. Multivessels disease were not significant.

The right coronary artery (RCA) disease was found significantly higher proportion in ST elevation in leads V5 and V6 with ST elevation in lead III > V6 (n-37; 82%) than in ST elevation in leads V5 and V6 with ST elevation in lead III ≤V6 (n-4; 23%)(*p=0.0001)*, whereas the left circumflex artery (LCX) disease was found very frequently in ST elevation in leads V5 and V6 with ST elevation in III ≤V6 (n-12; 71%) than in ST elevation in leads V5 and V6 with ST elevation in lead III >V6 (n-7; 16%) (*p-0.0001)*.

According to coronary angiography findings the sensitivity, specificity and positive predictive value and negative predictive value in ST elevation in leads V5 and V6 with ST elevation in lead III>V6 to predict right coronary artery (RCA) as a culprit artery were 90%, 63%, 84% and 75% respectively. And, the sensitivity, specificity, positive predictive value and negative predictive value in ST elevation in leads V5 and V6 with ST elevation in lead III≤V6 to predict left circumflex artery (LCX) as a culprit artery were 63%, 90%, 75% and 84% respectively as shown in the Table 1.4.

DISCUSSION

Abnormalities on the electrocardiogram (ECG) are frequently used to predict the infarct related artery (IRA) in patients with acute myocardial infarction (AMI). Acute myocardial infarction of the inferlateral wall is due to occlusion of the right coronary artery (RCA) or the left circumflex (LCX) coronary artery and the outcome depends on the culprit artery involved. The presence of significant ST elevation >2mm in leads V5 and V6 with inferior wall AMI is a sensitive and specific sign of mega-artery- related infarction with expected high ischemic risk (Assali et al., 1998). Thus, the prediction of a culprit artery based on electrocardiography is of clinical importance. Our findings have important clinical implications to predict the infarct related artery (RCA or LCX) by comparing the degree of ST elevation in lead V6 with that of lead III in inferior wall STEMI.

Leads V5 and V6 face the posterolateral wall adjacent to the apex of the left vertical (Cooksey et al., 1977). This region of the left ventricle is usually supplied by the larger obtuse marginal branch of the left circumflex artery (LCX), the atrioventricular branch of the right coronary artery, or the diagonal branch of the left anterior descending coronary artery. Therefore, the concurrent ST elevation in leads V5 and V6 during inferior AMI presenting with ST elevation in the inferior leads might reflect transmural ischemia extending to the posterolateral wall in addition to the inferior wall, which could be induced by either the right coronary artery (RCA) or the left circumflex artery (LCX) occlusion.

Fewer studies have suggested that ST elevation in leadsV5 and V6 suggests the presence of the left circumflex artery (LCX) occlusion (Herz et al., 1997; Bairey et al., 1987; Assali et al., 1998; Kontos et al., 1997). In our study, among 62 patients with ST elevation in leads V5 and V6, the infarct-related artery was the right coronary artery in 41 patients (66%; *p-0.0001)* and left circumflex artery (LCX) in 19 patients (31%; *p-0.0001).* The ST elevation in leads III and V6 was strongly related to the infarct related artery. The ST elevation in lead III > V6 indicated right coronary artery (RCA) occlusion in 37 patients (82%) with the sensitivity and specificity of 90% and 63%, respectively. Whereas the ST elevation in lead III≤V6 indicated left circumflex artery (LCX) occlusion in 12 patients (71%) with sensitivity and specificity of 63% and 90% respectively. These findings are consistent with the results of Kosuge M et al. (Kosuge et al., 2012). In the presence of ST elevation in leads V5 and V6 in inferior wall AMI, we believe that these simple ECG criteria are useful for predicting the infarct related artery (IRA) in the clinical practice to evaluate the high-risk. Lead III faces the right inferior wall, and is usually supplied by the right coronary artery distribution (Kosuge et al., 1998; Herz et al., 1997; Zimetbaum et al., 1998; Hasdai et al., 1995; Kontos et al., 1997; Zimetbaum and Josephson, 2003; Wagner et al., 2009). We, therefore, selected lead III as an illustrative inferior lead in our study. ST segment elevation in lead III > V6 might suggest that the severity of transmural ischemia as evaluated by the degree of ST-segment elevation was comparatively greater in the right inferior wall to the posterolateral wall. This might suggest involvement of the mega-artery right coronary artery occlusion, irrespective of the site of occlusion, as shown in the present study. A similar findings were seen in Kosuge M.et al. (Kosuge et al., 2012). In contrast, ST elevation in lead III≤V6 indicates the similar in the right inferior wall and the posterolateral wall or greater than in the latter. In the present study, 12 patients (71%) had LCX occlusion with ST elevation in lead III≤V6. The ECG changes during LCX artery occlusion is highly variable because of its anatomical distribution. The LCX supplies blood to the posterior, posterolateral, posteroinferior, or posteroinferolateral myocardium (Dunn et al., 1984). During LCx occlusion, posterolateral wall involvement is associated with proximal occlusion because this region is supplied, at least in part, by the obtuse marginal branch of the LC artery (Hasdai et al., 1995; Dunn et al., 1984). When occlusion of the LCx artery is more distal, the posterolateral wall will not be involved, and the ST-segment vector will be oriented more inferior. Thus, the ST-segment will not be elevated in leads V5 and V6, and ST elevation in inferior leads could occur (Dunn et al., 1984). Thus, when assessing the greater risk area in patients with inferior wall AMI, the clinician should consider the presence of ST segment elevation in leads V5 and V6, which implies a large perfusion area of the left ventricle and the need for aggressive therapeutic strategies to improve myocardial reperfusion. ST-segment depression in the anterior leads V1-V3 in patients with inferior acute wall STEMI (ST elevation in II, III and αVF) have been shown to be useful for evaluating the infarct size and outcomes (Berger and Ryan, 1990; Evans et al., 1998; Peterson et al., 1996). In our study, the presence of ST-segment depression in leads V1-V3 with ST elevation in leads V5 and V6 with ST elevation in lead III>V6 and ST elevation in lead III≤V6 was not of discriminatory value.

Tierala et al proposed that in inferior AMI, right coronary artery (RCA) might be the culprit artery in the ST elevation in lead III>II and left circumflex artery (LCX) if the ST elevation in lead II>III (Tierala et al., 2009). In our study, we reported that in patients with inferior AMI, the degree of ST elevation in the inferior leads, mainly leads III>II, was significantly greater in right coronary artery (RCA) occlusion than in left circumflex artery occlusion (81% vs 16%). Importantly, ST elevation in lead III>II frequently supported ST elevation in leads V5 and V6 with ST elevation in lead III>V6 than the ST elevation in leads V5 and V6 with ST elevation in lead III≤ V6 (n-38; 84% vs n-9; 53%; *p-0.027).* However, ST elevation in lead II>III favored ST elevation in leads V5 and V6 with ST elevation in lead III≤V6 than the ST elevation in lead

III>V6 (n- 8; 47% vs n-7; 15%; *p-0.027).*

The present study had several limitations that need to be considered when interpreting the clinical implications of our findings. This was a small sample size, with retrospective study in a single center. The study didn't deal in predicting the lesion proximal or distal coronary artery occlusion and involvement of the mega artery. The study lacked the follow-up and complications like infection, postinfarction,angina, arrhythmias and mortality. The electrocardiographic findings in our study might differ considerably from those patients with previous episodes of myocardial infarction, late presentation. Thus, our findings cannot be generalized to all patients with inferior wall AMI and large scale studies are needed to confirm the present findings.

CONCLUSION

In conclusion, ST segment elevation in leads V5 and V6 suggests a greater risk area in patient with acute inferior wall myocardial infarction. The right coronary artery occlusion was most common in ST elevation in leads V5 and V6 with ST elevation in lead III>V6 and the left circumflex artery occlusion in ST elevation in leads V5 and V6 with ST elevation in lead III≤V6. And, while evaluating risk in an acute inferior wall myocardial infarction, we should also look for ST elevation in leads V5 and V6, as it signify a larger perfusion territory and demands for more aggressive reperfusion therapy.

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