

A Simple Non-invasive ECG Technique to Localize Culprit Vessel Occlusion Site in ST-Elevation Myocardial Infarction (STEMI) Patients

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Received date: November 15, 2017; Accepted date: November 22, 2017; Published date: November 27, 2017

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Abstract

Background: Electrocardiograms (ECGs) are essential in identifying the type and location of acute myocardial infarction. In the setting of inferior wall myocardial infarction (IWMI), identification of the right coronary artery (RCA) as the culprit artery is important because of the potential complications associated with its involvement.

Objectives: The study was conducted to identify the culprit artery responsible for STEMI from ECG tracings. To validate Fiol's algorithm in Eastern Indian population and to compare the ECG findings with coronary angiogram.

Methods: The cross-sectional study was conducted in 100 patients with acute STEMI. Patients presenting with acute ST-elevation myocardial infarction within 12 h of symptom onset were included in the study. In this study, we focused on Fiol's algorithm and some other pre-specified criteria for prediction of occluded vessels. Cohen's kappa statistical method was used to correlate ECG localisation of culprit artery with that of coronary angiogram.

Results: Out of 100 STEMI patients, 73 patients had left anterior descending as the culprit artery, 24 patients had right coronary artery as the culprit artery and 3 patients had left circumflex artery as the culprit artery (73%, 24% and 3% respectively). Following Fiol's algorithm, we have found that ECG has: high specificity (86%) but low sensitivity of 29% for an occlusion proximal to D1. Moderate sensitivity and moderate specificity (62% and 69% respectively) was noted for an occlusion distal to D1. Similarly, high sensitivity (90%) but low specificity (33%) was noted for lesion proximal to S1. Cohen's kappa $\kappa=1$ suggests excellent agreement between ECG and coronary angiogram for inferior wall MI (both RCA and LCx occlusion).

Conclusion: It can be concluded that ECG can reliably predict the culprit artery in STEMI patients. The Fiol's algorithm is validated and considered as a simple tool to localize the infarct related artery in anterior wall myocardial infarction (AWMI) and inferior wall myocardial infarction (IWMI).

Keywords ECG; Angiogram; Right coronary artery; STEMI; Fiol's algorithm

Introduction

The myocardium is frequently provided by three coronary arteries, although there are several variations in the number, origin, course, and distribution of coronary arteries [1]. A significant contribution to left ventricular myocardial blood flow is by left anterior descending coronary artery (LAD) (50%), rest is equally contributed by right coronary artery (RCA) and left circumflex artery (LCx). Also, most of the right ventricle is supplied by RCA. The need for a rapid reperfusion therapy is widely determined by how close the occlusion site is to the origin of the coronary artery, which corresponds to the area of ischemic myocardium. ST-elevation in precordial leads in patients with acute coronary syndrome (ACS) symptoms indicates ST-segment elevation myocardial infarction (STEMI) involving the area perfused by the left anterior descending coronary artery (LAD) [2]. In acute anterior myocardial infarction (AMI), the site of occlusion in the left anterior descending (LAD) coronary artery is related to the extent of the myocardial necrosis and prognosis [3-7].

Patients who presented with ECG criteria of proximal occlusion (any ST-depression in III+aVF>0.5 plus sum of ST-deviation in aVR +V1-V6 ≥ 0) constitute a high-risk group. This high-risk group had lower ejection fraction, higher peak of creatine phosphokinase (CPK) and its isoenzyme MB (CPK-MB), and worse Killip class, and included more patients with major adverse cardiac events compared to the patients who did not meet all these criteria [1]. ECG can help in identifying the location of occlusion of the coronary arteries in acute MI, thereby contributing to predict the amount of myocardium at risk and guide decisions regarding the urgency of revascularization. It is better to apply an easy to use, a sequential algorithm based on deviations of ST in 12-lead ECG rather than to assess the ECG criteria separately [1]. Thus, an early prediction of proximal LAD occlusion is important not only from an academic standpoint but also from a clinical point of view. Changes in ST-segment in different leads of surface ECG can identify patients with proximal or distal LAD occlusion [2].

In this study, we focused on Fiol's algorithm and some other pre-specified criteria for prediction of occluded vessels. The study was conducted in eastern population of India and ECG criteria's are all validated in those population. So far, very few studies are done in Indian population to characterize the infarct-related coronary artery

and current study is an attempt to do the same. The primary objectives of the study were an identification of the culprit artery responsible for STEMI from ECG tracings, localization of the site of lesion in the artery to proximal, mid or distal part, validation of the different algorithms, to identify the infarct-related artery and correlation with coronary angiogram, identification of the STEMI patients who need urgent intervention. This study will help in the routine clinical practice to categorize and optimize STEMI patients by artery occluded into either right coronary artery or left coronary artery or left circumflex coronary artery as the infarct related culprit vessel and predict the possible complication. The study will depict the importance of knowing culprit artery responsible for STEMI before putting the patient to invasive coronary angiography and in some cases; it can categorize the group patients who are at higher risk of considerable damage to the myocardium.

Patients and Methods

The cross-sectional study was conducted in 100 patients with acute STEMI. Informed consent was obtained from all patients. The enrolled patients were distributed depending on their age, gender and risk factors. The study was approved by the local ethical committee and has been performed by the ethical standards in the Declaration of Helsinki. The patients presented with acute STEMI admitted to cardiology department of RGKAR Medical College and Hospital, from February 2014 to end of August 2015. All patients within 12 h from the onset of symptoms were included, the criteria for diagnosis of acute ST-elevation myocardial infarction was as follows: a) Ischemic symptoms, and b) New ST elevation at the J-point in two contiguous leads with the cut-off points: ≥ 0.2 mV in men or ≥ 0.1 mV in women in leads V2-V3 and/or ≥ 0.1 mV in other leads. Patients who were excluded from the study were those having acute STEMI but not willing to undergo a coronary angiogram, non-ST elevation MI (NSTEMI), patients with previous myocardial infarction, patients with previous coronary bypass or angioplasty, implanted pacemaker, previous bundle branch block in ECG, known case of congenital heart disease.

Demographic profile (age, sex), risk factor profile (smoking, diabetes, hypertension) were noted. Patients were divided into three groups: Groups I, II and III according to the localization of occlusion site in left anterior descending (LAD), right coronary artery (RCA) and left circumflex (LCx) coronary arteries, respectively. Group I was further divided into four subgroups: Ia, Ib, Ic and Ib+c according to whether occlusion in LAD was proximal to both first septal (S1) and first diagonal (D1) branches, proximal to S1 but distal to D1, distal to S1 but proximal to D1 branch, distal to both S1 and D1 branches respectively. Group II was further divided into two subgroups: IIa and IIb according to whether occlusion in RCA was proximal or distal to right ventricular (RV) branch, respectively. Each patient was subjected to electrocardiogram with the use of special leads. Culprit artery was identified using different algorithms. Next, every patient was subjected to coronary angiogram to confirm the electrocardiographic prediction of the infarct-related artery. The extent of coronary artery disease and the morphology of all coronary artery lesions were determined.

Electrocardiography evaluation

A detailed surface ECG with special leads was performed when required, eg. Right-sided leads in inferior wall MI and if necessary serial ECG to observe the changes within 12 h of presentation to localize the culprit artery.

Coronary angiographic evaluation

Coronary angiography (with or without PCI) was performed to detect the culprit lesion within 1 week after the admission of the patient. Two independent observers evaluated the CAG findings. Images of the coronary tree of all patients were obtained using standardized projections with the digital quantitative Siemens Axiom Artis Zee system® (Siemens, the Germany).

- Significant lesion/stenosis was defined in the coronary angiogram by the presence of total occlusion, $>70\%$ stenosis, acute thrombosis or dissected plaque in the coronary artery.
- Proximal LAD was defined as the part of left anterior descending artery proximal to and/or including the origin of first septal perforator, Mid LAD as the part distal to the first septal perforator up to second diagonal branch and distal LAD after the origin of second diagonal branch.
- Proximal RCA was defined as the part of right coronary artery proximal to and including the origin of first major RV branch.
- Mid-RCA as the region distal to the origin of first major RV branch up to the last acute marginal branch and distal RCA from last acute marginal branch to the origin of posterior descending artery.

Statistical Analysis of Data

The Chi-square test for categorical variables was used for comparisons between groups. An unpaired t-test was used for comparison of two different patient groups. Standard methods were used to calculate sensitivity, specificity, positive predictive value (PPV) and negative predictive value (NPV); 95% confidence intervals (95% CIs) were calculated. Cohen's kappa statistics is calculated as a measure of inter-rater reliability by SPSS software version 20. Statistical analysis was performed using SPSS 20 (Chicago, IL, USA). A probability value of $p < 0.05$ was considered significant.

Results

In the present study, 100 patients, consisting of 10 female and 90 male participants, with a maximum number of patients (34%) in the age range of 51-60 years were evaluated (Table 1). The median age of the STEMI patients was 51.7 ± 13 years. Highest percentages (34%) of patients were present in 51-60 years age group followed by ≥ 61 (24%), 41-50 (22%) and 31-40 (15%) years age group respectively. Lowest percentages of patients were present in 21-30 years age group (5%). The rate of previous MI was 100% (90% male and 10% females). None of the patients were excluded from the primary analysis. The patients were distributed as per the baseline demographic, clinical, laboratory findings and angiographic characteristics, listed in Table 1. 40% of the patients had hypertension, 33% were diabetic and 30% were smokers. The relationship between the risk factors and STEMI patients was statistically not significant (Table 1).

Variables	Patients	p value
	N=100	
Distribution of patients by age (%)		
21-30	5	
31-40	15	

41-50	22	
51-60	34	
≥ 61	24	
Age, mean ± SD		
STEMI patients	51.7 ± 13 years	
With anterior wall MI (%)	52.7 ± 13 years	
With inferior wall MI (%)	48.4 ± 12.6 years	
Gender: Male (%), Female (%)	90, 10	
Male: With anterior wall MI % (n), with inferior wall MI % (n)	73.3 (66), 26.7 (24)	p=0.88
Female: With anterior wall MI (%) with inferior wall MI (%)	70 (7); 30 (3)	
Patients with risk factors		
Diabetes	33	
Hypertension (%)	40	
Smokers (%)	30	
STEMI (%)		
Relationship of diabetes with STEMI: With anterior wall MI (%), with inferior wall MI (%)	Yes: 67.5 and 32.5 No: 76.7 and 23.3	p=0.60
Relationship of hypertension with STEMI: With anterior wall MI (%), with inferior wall MI (%)	Yes: 69.7 and 30.3 No: 74.6 and 25.4	p=0.09
Relationship of smoker with STEMI: With anterior wall MI (%), with inferior wall MI (%)	Yes: 67.5 and 32.5 No: 76.7 and 23.3	p=0.31
Diabetic and smoker with STEMI: With anterior wall MI (%), with inferior wall MI (%)	16.4 and 14.8	p=0.62
Hypertensive and Diabetic with anterior wall MI (%), with inferior wall MI (%)	15 and 11.1	
Hypertensive and smoker with STEMI: With anterior wall MI (%), with inferior wall MI (%)	16.4 and 18.5	
Hypertensive, diabetic and smoker with STEMI: With anterior wall MI (%), with inferior wall MI (%)	6.84 and 7.41	

Table 1: Demographic and clinical characteristics of study population.

Electrocardiography analysis to identify the culprit artery involved in STEMI patients

100 STEMI patients underwent ECG followed by coronary angiogram. In our study 73% patients had left anterior descending as the culprit artery, 24% patients had right coronary artery as the culprit artery and 3% patients had left circumflex artery as the culprit artery (Figure 1).

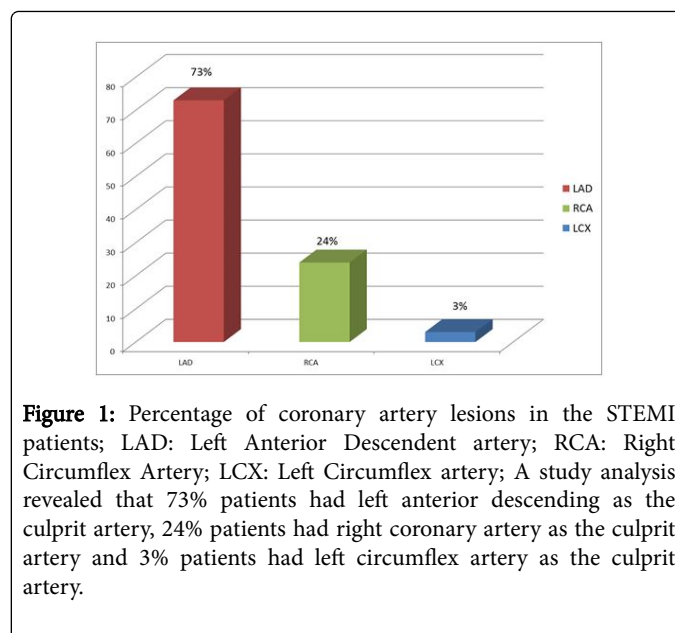


Figure 1: Percentage of coronary artery lesions in the STEMI patients; LAD: Left Anterior Descending artery; RCA: Right Circumflex Artery; LCX: Left Circumflex artery; A study analysis revealed that 73% patients had left anterior descending as the culprit artery, 24% patients had right coronary artery as the culprit artery and 3% patients had left circumflex artery as the culprit artery.

Distribution of occlusion in LAD about S1 and D1 as a culprit artery

The LAD has two major branches S1 & D1. The order of emergence may be S1 followed by D1 or D1 followed by S1. In our patients LAD occlusion was observed in 73 patients. The level of occlusion was as follows: 39 patients (53.4%) had lesion proximal to both S1 and D1, 10 (13.7%) patients had lesion proximal to S1 but distal to D1, 13 patients (17.8%) had lesion distal to S1 but proximal to D1 while 11 patients (15.10%) had lesion distal to both S1 and D1.

Angiographic analysis as per the ECG criteria for distribution of lesion to LAD-D1 branch

In our study, 73 patients having LAD as culprit artery, 24% of the patients (n=18) had Σ ST depression in III+aVF ≥ 2.5 , 20% (n=15) of the patients had proximal lesion, and 4% (n=3) of the patients had distal lesion to D1. Of the 29 patients who had ST-segment isoelectric or elevated in III and aVF, 55% (n=16) of the patients had proximal lesion and 44% (n=13) of the patients had distal lesion to D1. Of the 26 patients who had Σ ST depression in III+aVF ≤ 2.5 and ≥ 0.5 , 80% (n=21) of the patients were found to have proximal lesion and 19% (n=5) patients had distal lesion to D1 (Table 2).

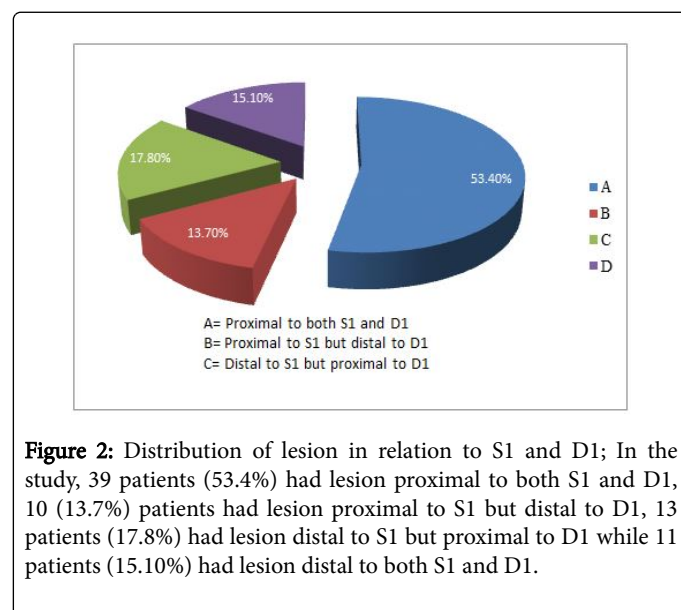
ECG criteria	Occlusion site	Sensitivity (%)	Specificity (%)	PPA (%)	NPA (%)
Σ ST depression III+aVF ≥ 2.5	Proximal to D1 (24%)	29	86	83	33
ST-segment in III and aVF isoelectric or elevated	Distal to D1 (40%)	62	69	45	82
Sum of aVR+V1-V6 ≥ 0	Proximal to S1 (44%)	90	33	73	61.5
Sum of aVR+V1-V6 < 0	Distal to S1 (8%)	33	90	61.5	73

Table 2: ECG Criteria for distribution of lesion with respect to LAD; NPA: Negative Predictive Accuracy; PPA: Positive Predictive Accuracy.

Angiographic analysis as per the ECG criteria for distribution of lesion to LAD-S1 branch

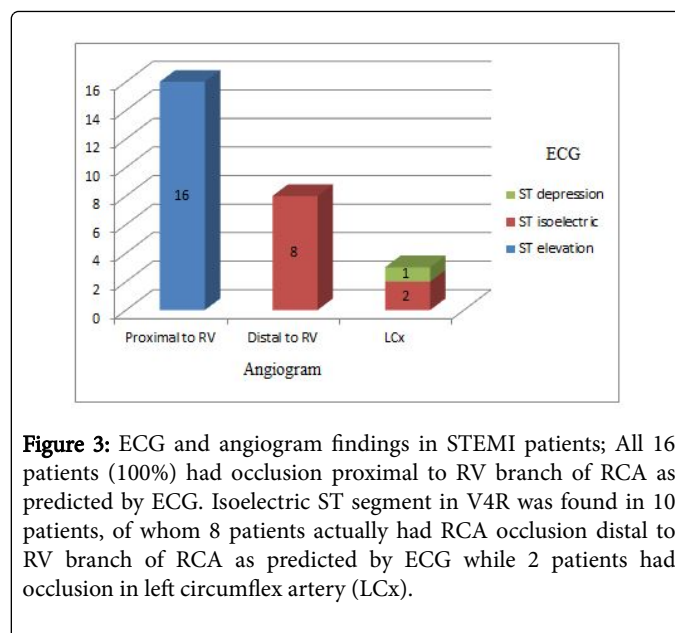
In our study group of 73 patients having LAD as culprit artery, 60 patients (82%) met the ECG criterion of Σ ST deviation in $aVR+V1-V6 \geq 0$. Of these 60 patients, only 44 patients (73%) were found to have occlusion proximal to S1 as predicted and the remaining 16 patients (27%) had occlusion distal to S1. Of the 73 patients having LAD as the culprit artery, 13 patients (18%) met the ECG criteria of Σ ST deviation in $aVR+V1-V6 < 0$. Out of them actually only 8 (62%) patients had occlusion distal to S1 as predicted, the remaining five patients (38%) had occlusion proximal to S1 (Table 2).

The D1 and S1 analysis showed that best criteria to identify the culprit artery were marked ST depression in III, aVF (Sum of ST depression in III and aVF ≥ 2.5 mm) was found to have high specificity (86%) for an occlusion proximal to D1 but with a low sensitivity of 29%. On the other hand an elevated or isoelectric ST segment in III, aVF has moderate sensitivity and moderate specificity (62% and 69% respectively) for an occlusion distal to D1. ST deviation in $aVR+V1-V6 \geq 0$ was found to have high sensitivity (90%) but low specificity (33%) for lesion proximal to S1. On the other hand sum of ST deviation in $aVR+V1-V6 < 0$ was found to have high specificity for occlusion distal to S1 (90%) but low sensitivity (33%) (Figure 2).



RCA as the culprit artery

Out of the 16 patients having ST elevation in V4R, all 16 patients (100%) had occlusion proximal to RV branch of RCA as predicted by ECG. Isoelectric ST-segment in V4R was found in 10 patients, of whom 8 patients had RCA occlusion distal to RV branch of RCA as predicted by ECG while 2 patients had occlusion in LCx. One patient had ST depression in V4R, and the patient actually had occlusion in LCx artery as predicted by the ECG. Therefore out of the 3 patients with occlusion in LCx, 2 patients had isoelectric ST-segment while 1 patient had depressed ST-segment in V4R (Figure 3).



Distribution of lesion to RCA and LCx

In our study, there is high sensitivity (100%) for RCA occlusion in ECG while specificity is low (33.33%). On the other hand, there is high specificity (100%) for LCx occlusion in ECG while sensitivity is very low (33.33%) (Table 3).

Total no. of patients fulfilling ECG criteria	RCA occlusion	LCx occlusion	Sensitivity (%)	Specificity (%)	PPA (%)	NPA (%)
ECG suggestive of RCA occlusion	24	2	100%	33.33%	92.30%	100%
ECG suggestive of LCx occlusion	0	1	33.33%	100	100	92.3

Table 3: ECG Criteria for distribution of lesion with respect to RCA and LCx; NPA: Negative Predictive Accuracy; PPA: Positive Predictive Accuracy; *Findings as per angiogram.

Correlation between ECG and coronary angiogram

Cohen's kappa statistics ($\kappa=0.55$) (between 0.41–0.60) for lesion with respect to D1 and indeterminate lesion are taken together while lesion proximal to D1 is considered separately. On the other hand, Cohen's kappa statistics ($\kappa=0.280$) (0.21–0.40) for lesion with respect to D1 when lesion proximal to D1 and indeterminate lesion are taken together while lesion distal to D1 is considered separately. Cohen's kappa statistics ($\kappa=0.607$) (between 0.41 to 0.80) for lesion with respect to S1. Cohen's kappa statistics ($\kappa=1$) (>0.81) for inferior wall MI (both RCA and LCx occlusion) (Table 4) (Figure 4).

Site of lesion	Cohen's Kappa (κ)	Interpretation
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AWMI: LAD	0.55 (proximal and distal+intermediate)	Moderate agreement between ECG and coronary angiogram
D1	0.280 (distal and proximal+intermediate)	Fair agreement between ECG and coronary angiogram
S1	0.607	Substantial agreement between ECG and coronary angiogram
IWMI: RCA & LCx	1	Excellent agreement (almost perfect agreement) between ECG and coronary angiogram

Table 4: Correlation between ECG and coronary angiogram; AWMI: Anterior Wall Myocardial Infarction; IWMI: Inferior Wall Myocardial Infarction; RCA: Right Coronary Artery; LCx: Left Circumflex coronary arteries; LAD: Left Anterior Descending.

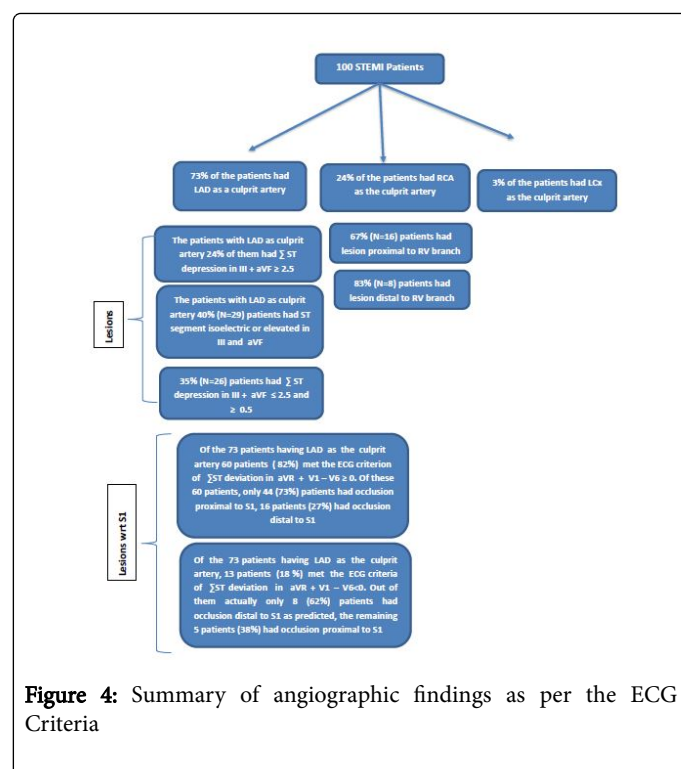


Figure 4: Summary of angiographic findings as per the ECG Criteria

Discussion

Although coronary angiography is the gold standard for determining the infarct-related artery in acute myocardial infarction, the ECG can be a clinically valuable tool in identifying the culprit artery [8]. The early and accurate identification of the infarct-related artery on the ECG can help predict the amount of myocardium at risk and guide decisions regarding the urgency of revascularization. Accurate localization of infarct-related artery from surface electrocardiogram is crucial in formulation of management and need for early thrombolysis or primary percutaneous coronary intervention. The recent findings of Masoudi et al. suggest that the failure to identify high-risk ECG patterns in patients with acute myocardial infarction (AMI) results in lower quality care in the emergency room and highlights the importance of system changes to enhance the accuracy of ECG interpretation [9]. Knowing that the occlusion is proximal or

distal to D1 or S1 may be crucial for deciding on the best approach to treatment: to start fibrinolytic treatment and keep the patient in the hospital because the risk of a large AMI is low, or, independently of fibrinolytic treatment, make the decision to send the patient immediately to a referral center for a PCI, since there is ECG evidence that the risk of a large AMI is high.

Occlusion of the LAD artery may lead to a very extensive anterior MI, or only septal, apical-anterior or mid-anterior according to the site of occlusion. Proximal LAD occlusion has been documented as an independent predictor of poor outcome related to higher mortality and recurrent MI and distal LAD occlusion is considered to have a better outcome [10,11]. Ischemia provoked by LAD occlusion is reflected by ST-elevation in precordial leads. The different ECG criteria for predicting the site of occlusion are based on the direction of the injury vector. Using Fiol's algorithm, we demonstrated an approach to identify and ascertain the culprit artery (right coronary versus circumflex artery) in STEMI with ST-segment elevation in inferior leads. Different criteria have been used to differentiate between proximal and distal LAD using the deviation of ST in inferior leads as the most useful standard. However, we predict the location of an occlusion in LAD STEMI with high accuracy. In our study hypertension, diabetes mellitus and smoking were taken into consideration as risk factors for coronary artery disease and myocardial infarction. No other study to our knowledge has compared the risk factors among the various types of STEMI although Fiol et al. in their study have divided patients into high risk (61 patients) and low risk (39 patients) groups using age, ejection fraction, killip class, CK value, LV failure, ventricular arrhythmia, etc., however, no such grouping was done in our study [12]. Of the 100 STEMI patients enrolled in the study, all underwent ECG followed by coronary angiogram. Of the 73 patients having left anterior descending as the culprit artery, 18 patients had additional lesions in the left circumflex and 11 in right coronary artery. Of the 24 patients having right coronary artery as the culprit artery, 6 patients had additional lesions in the left anterior descending artery and 3 patients had lesions in the left circumflex. The secondary lesions were all <50% stenosis, hence not significant. Therefore, culprit artery localization was as follows: 73 patients had LAD as the culprit artery while 24 patients had RCA as the culprit artery and 3 patients had LCx as the culprit artery. During the LAD culprit artery analysis at D1, marked ST depression in III, aVF (Sum of ST depression in III and aVF ≥ 2.5 mm) was found to have high specificity (86%) for an occlusion proximal to D1 but with a low sensitivity of 29%. An elevated or isoelectric ST segment in III, aVF has moderate sensitivity and moderate specificity (62% and 69% respectively) for an occlusion distal to D1. However, lesions in the S1 terminal of the LAD culprit artery, ST deviation in aVR+V1-V6 ≥ 0 was found to have high sensitivity (90%) but low specificity (33%) (PPV-32%, NPV-100%) for lesion proximal to S1 and ST deviation in aVR+V1-V6 < 0 was found to have high specificity for distal occlusion to S1 (90%) but low sensitivity (33%) (PPV-61.5%, NPV-73%). Present study findings examining sensitivity, specificity, PPV, NPV as per the Fiol et al. criteria. The results obtained were in an agreement with the original study population of Fiol et al. we also find that the results are broadly similar both for lesions proximal and distal to S1. Additionally, the ability of ST-segment deviation to predict RCA occlusion (proximal/ distal) had a high sensitivity of 100% and a low specificity of 33.33% (PPV-92.3%, NPV-100%). The ability of ST-segment deviation to predict LCx occlusion had a low sensitivity of 33.33% and a high specificity of 100% (PPV-100%, NPV-33.33%). The comparison

of our study results is in agreement with that of Fiol et al. and Tierala et al. [13].

The minor differences in the results of our study and those of Fiol et al. (for LAD occlusion-D1 and S1 branches) and those of Fiol et al. and Tierala et al. (for RCA/LCx occlusion) are because of differences in the composition of the study population, differences in location and severity of the lesions in the arteries, time between ECG recording and coronary angiogram, etc. Our current study is an attempt to localize the culprit artery from ECG in STEMI following the study by Fiol et al. and perhaps it is the single study which has been conducted in Eastern Indian population. Our study results are broadly similar to the original study by Fiol et al. suggesting that this algorithm of ECG can be reliably used to localize the infarct-related artery in our study.

However, our study is unique because we have calculated Cohen's kappa (κ) in addition to estimation of validity which gives fair to substantial agreement between ECG and coronary angiogram for localizing the culprit artery in STEMI patients with reliable correlation.

$\kappa=0.55$ for lesion to D1 suggests a moderate agreement between ECG and coronary angiogram. $\kappa=0.280$ for lesion concerning to D1 when lesion proximal to D1 and indeterminate lesion are taken together while lesion distal to D1 is considered separately suggests a fair agreement between ECG and coronary angiogram. $\kappa=0.607$ for lesion to S1, which indicates substantial agreement between ECG and coronary angiogram.

Conclusion

ECG can be used in clinical practice to diagnose culprit artery in inferior wall MI as either RCA or LCX as per Fiol's algorithm. ECG can be reliably used to localize the lesion in LAD as culprit artery (to S1) as per Fiol's algorithm, and the reliability of ECG is more for S1 as compared to D1. Although coronary angiography remains the benchmark method for determining the infarct-related artery in acute myocardial infarction, ECG information can help to expect culprit artery involved before angiography, especially in hospitals without angiographic facilities.

Acknowledgement

We are grateful to USV India for the Publication support. The authors want to thank Dr Gandhali Deshpande from CIMS Medica Mumbai, for the scientific writing and editing support.

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