Original Article

Acute Inferior Wall MI

Clinical Significance of ST Segment Depression in Lead aVR on ECG as a Predictor of Left Circumflex (LCx) Artery Involvement in Patients of Acute Inferior Wall Myocardial Infarction

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ABSTRACT

Objective: To determine the diagnostic accuracy of ST depression in aVR lead on electrocardiogram (ECG) in detecting left circumflex artery involvement in inferior wall myocardial infarction taking coronary angiography as gold standard.

Study Design: Descriptive / cross-sectional study

Place and Duration of Study: This study was conducted at the Department of Cardiology, Ch. Pervaiz Elahi Institute of Cardiology, Multan from 13-Aug-2017 to 12-Feb-2018.

Materials and Methods: 191 patients with diagnosis of inferior wall MI having age 30-70 years were included in this study. Diagnosis of inferior wall myocardial infarction was made on the basis of ECG, and underwent coronary angiography. Data was analysed through computer software SPSS 20.0. 2×2 contingency table was used to calculate sensitivity, specificity, positive predictive value, negative predictive value of ST depression in aVR taking coronary angiography (CA) as gold standard.

Results: Mean age of study patients was 50.49±7.10 years. There were 156 (81.68%) male patients and 35 (18.32%) female patients. There were 79 (41.36%) smokers, 18 (9.42%) patients had positive family history, 111 (58.12%) were hypertensive and 17 (8.90%) patients were dyslipidemic. Sensitivity of ST segment depression in determining the LCx taking coronary angiography as gold standard was 91.8%, specificity 89.4%, positive predictive value (PPV) 75.0% and negative predictive value (NPV) was 96.9%.

Conclusion: ST depression in lead aVR has a good sensitivity and specificity (91.8% and 89.4% respectively) in determining the left circumflex artery (LCx) artery involvement in patients of acute inferior wall myocardial infarction.

Key Words: Inferior wall myocardial infarction, Lead aVR, Left circumflex artery.

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INTRODUCTION

Ischemic heart disease (IHD) is the leading cause of death and disability worldwide. Despite global reductions in age standardized incidence of acute myocardial infarction (MI) and in the prevalence of angina since the early 1990s, growing populations of aging high-risk individuals led to an increase in the global burden of IHD.[1] Slow normal turnover of cardio myocytes may be responsible for modest persistent elevations of troponin levels in certain normal individuals.[2,3]

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Although the majority of patients with MI have significant obstructive coronary disease, occasionally plaque rupture and ulceration can occur in the absence of an angiographic ally obstructive lesion[4].

ROS scavengers significantly attenuate postischemic dysfunction, supportingthe role of ROS in the pathophysiology of myocardial stunning[5,6]. Specific mechanisms involved in systolic dysfunction may include decreased responsiveness of sarcomeric proteins to calcium, and calcium overload-induced protease activation, followed by proteolysis of contractile proteins (such as troponins and α- actinin)[7].

The effects of ROS in myocardial stunning may involve alterations in calcium homeostasis, as free radicals are known to induce calcium overload in the heart and may reduce responsiveness of sarcomeric proteins to calcium[8]. ECG is an easy, cheap and universally accepted tool for the diagnosis, early risk factor stratification, and triage for guidance of therapy in patients with suspicion of acute MI.[9] The relative prevalence of inferior wall myocardial infarction is 21% among all MIs.[10] Therefore the prediction of

culprit artery in inferior wall myocardial infarction is often difficult because of relative dominance of right coronary or circumflex artery among patients.[11] Inferior wall myocardial infarction is usually manifested as presence of ST segment elevation in inferior leads (Lead II, III and aVF), along with ST changes in other leads that represent concomitant ischemia in other areas of heart or just reciprocal changes.[12]

Due to these ''cancellation'' effects, the relation between ECG changes and the location and extent of myocardial injury is very complex, which is detrimental to the proper identification of the culprit artery by means of ECG parameters.[13] ECG helps to predict the culprit artery and locate the lesion within the infarct related artery (IRA), thus providing clinically important information to augment clinical decision making and tailor reperfusion therapy.[14,15] ST segment changes in lead aVR are usually ignored in ECG interpretation.

Recently Gupta et al have concluded that ST depression > 1 mm in aVR lead predict involvement of left circumflex artery in patients of inferior wall myocardial infarction with sensitivity of 69% and 85% specificity.[16] Another study found 53% sensitivity and 86% specificity of ST segment depression in aVR lead to diagnose left circumflex artery involvement in inferior wall myocardial infarction.[17]

MATERIALS AND METHODS

After approval from ethical review committee of the hospital, total number of 191 patients who presented in Department Of emergency, were selected. This descriptive, cross-sectional study undergone in, Ch. Pervaiz Elahi Institute of Cardiology, Multan from 13-Aug-2017 to 12-Feb-2018 .Non-probability, consecutive sampling was done. All patients with diagnosis of inferior wall MI at presentation, both male and female and with age 30 to 70 years were included. Patients with MI other than inferior wall myocardial infarction, previous myocardial infarction, assessed on ECG findings (i.e. those having ST elevation in leads other than lead II, III and aVF, or presence of q waves on ECG) were excluded from this study.

Diagnosis of inferior wall myocardial infarction was made on the basis of 12 lead electrocardiogram (ECG). After taking informed consent, all patients underwent coronary angiography and findings were noted for presence or absence of involvement of left circumflex artery.Data regarding confounder variables such as family history, diabetes, smoking, dyslipidemia and hypertension will also be collected. Diagnosis of these confounder variables were made on the basis of previous history of patients or on the basis of new diagnosis of any of these co-morbidities during hospital stay (diagnosed on the basis of laboratory findings) was noted as presence of the disease. All this data was recorded on a specially designed Performa.

Collected data was analysed through computer software SPSS 20.0.

Mean and standard deviation were calculated for quantitative variables i.e. age. Frequency and percentage will be calculated for qualitative variables i.e. gender, family history, smoking, diabetes, hypertension, dyslipidemia, ST depression in aVR on ECG and presence of left circumflex artery involvement on coronary angiography. 2×2 contingency table was used to calculate sensitivity, specificity, positive predictive value, negative predictive value and diagnostic accuracy of ECG in diagnosing left circumflex artery involvement taking coronary angiography (CA) as gold standard. Effect modifiers were controlled by stratification. Post-stratification sensitivity and specificity was calculated again to see the effect of confounder variables on diagnostic accuracy and p-value ≤0.05 was considered as significant.

RESULTS

A total number of 191 patients with diagnosis of acute inferior wall myocardial infarction were included in this study. Mean age of study patients was 50.49±7.10 years. Maximum age was 70 years and minimum age was 32 years.

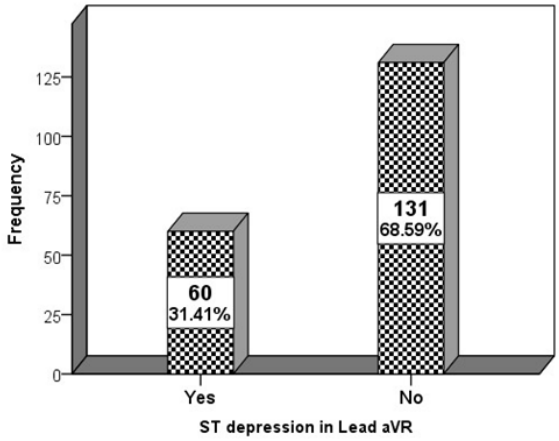

Figure No.1. Frequency of ST segment Depression in Lead aVR.

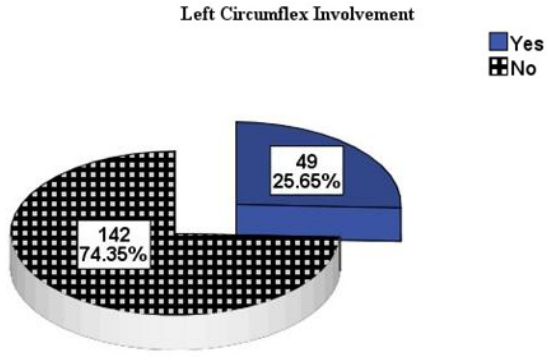

Figure No.2: Frequency of Left Circumflex Involvement on Coronary Angiography.

There were more males as compared to females who presented with acute inferior wall myocardial infarction. There were 156 (81.68%) male patients and 35 (18.32%) female patients. There were 79 (41.36%) smokers and 112 (58.64%) non-smoker patients in this study. There were 18 (9.42%) patients who were having positive family history of coronary artery disease. While in remaining 173 (90.58%) there was no history of coronary artery disease in 1st degree relatives.

Table No.1: Stratification of Age to determine the effect of age on sensitivity and specificity.

(i) Age ≤50 Years

		Left Circumflex Involvement on CAG		Total
		Yes	No	
ST depression in Lead aVR on ECG	Yes	22	13	35
	No	1	58	59
Total		23	71	94

Sensitivity = 95.7% Specificity = 81.7%

(ii) Age <50 Years

		Left Circumflex Involvement on CAG		Total
		Yes	No	
ST depression in Lead aVR on ECG	Yes	23	2	25
	No	3	69	72
Total		26	71	97

Sensitivity = 88.5% Specificity = 97.2%

Table No.2: Stratification of gender to determine the effect of gender on sensitivity and specificity.

(i) Male Gender

		Left Circumflex Involvement on CAG		Total
		Yes	No	
ST depression in Lead aVR on ECG	Yes	40	9	49
	No	3	104	107
Total		43	113	156

Sensitivity = 93.0% Specificity = 92.0%

(ii) Female Gender

		Left Circumflex Involvement on CAG		Total
		Yes	No	
ST depression in Lead aVR on ECG	Yes	5	6	11
	No	1	23	24
Total		6	29	35

Sensitivity = 83.3% Specificity = 79.3%

There were 111 (58.12%) hypertensive patients. There were 17 (8.90%) patients in whom dyslipidemia was diagnosed, while remaining 174 (91.10%) having normal blood cholesterol levels. On electrocardiograph (ECG), ST segment depression was noted in 60 (31.41%) patients. while remaining 131 (68.59%) patients with inferior wall myocardial infarction were not having ST segment depression on ECG (Fig 01). On coronary angiography, left circumflex involvement was diagnosed in 49 (25.65%) patients and LCx was normal in remaining 142 (74.35%) patients (Figure 2).

Stratification of gender was done. In male patients, sensitivity of ST segment depression was 93.0% and specificity was 92.0%. In female patients, sensitivity of ST segment depression was 83.3% and specificity was 79.3% (Table 2).

Stratification of family history of coronary artery disease was done. In patients having positive family history, sensitivity was 100% and specificity was 92.9%. In patients having negative family history, sensitivity was 91.1% and specificity was 89.1%.

Stratification of smoking was done. In patients having positive smoking history, sensitivity of ST segment depression was 88.2% and specificity was 90.3%. In patients having negative smoking history, sensitivity was 93.8% and specificity was 88.8% .

Stratification on the basis of hypertension was done. In hypertensive patients, sensitivity was 88.9% and specificity was 90.3%. In non-hypertensive patients, sensitivity was 93.5% and specificity was 88.8% .

Stratification on the basis of dyslipidemia was done. In dyslipidemic patients, sensitivity was 50% and specificity was 80%. In patients with normal cholesterol levels, sensitivity of ST depression was 97.7% and specificity was 92.2% .

DISCUSSION

Inferior wall MI due to RCA occlusion frequently presents with STE in leads

II, III, and aVF, with reciprocal ST-segment depression (STD) in leads I and aVL. The ECG changes in LCX occlusions are highly variable. Approximately 30–50% of patients present with STE, usually in the inferior leads II, III, and aVF. Others show STD in leads V1–V4, or occasionally a tall R wave in lead V1. In up to 38% of patients, there is no discernible STE.[18]

In an I-AMI, the prognosis of patients depends on identification of the occluded artery (RCA or LCx). Patients with occlusion in the RCA (approx. 80% of cases) is frequently associated with right ventricle involvement and have a poorer outcome.[19,20] The identification of arteries and the size of infarct plays an important role in guiding the reperfusion therapy. The display of lead aVR (−150°) in an inverted format as lead −aVR (+30°) lies between lead I (0°) and lead II (60°). Thus, aVR depression means −aVR elevation, which represents the infarct of the apical and inferolateral walls, usually supplied by the posterolateral branch of either the RCA or LCx itself. The aVR depression suggests the involvement of LCx or a large RCA with a large posterolateral branch

occlusion. This finding is consistent with the observation that 60% of both dominant RCA and dominant LCx infarctions had aVR depression.[21]

Several studies have considered the clinical importance of ST-segment depression in lead aVR in an I-AMI to identify the culprit artery. Tierala et al.[22] in the HAAMU trial, a non- randomized prospective study of an acute I-AMI, proposed a new algorithm and compared it with the prior algorithm from Fiol et al.[23] to predict the culprit artery. Among 98 patients included in their study, they found with ECG and angiography a positive and negative predictive value for the prediction of LCx or RCA as an IRA of 75% and 94%, 92% and 75% respectively.

Kanei et al, with the main aim to review all algorithms and compare them to ST depression in the lead aVR to predict the culprit artery in an I-AMI found the sensitivity and specificity of ST segment depression in lead aVR to predict the LCx as a culprit artery were 53% and 83% respectively. RCA as the culprit artery, the sensitivity and specificity were, 86% and 55%, respectively.[24]

Nair and Glancy[25] found that quantifying ST depression in lead aVR distinguished a culprit LCX (≥1 mm) from a culprit RCA (<1 mm or no depression), in a small retrospective analysis. However, Baptista et al.[25] found that lead aVR ST depression showed limited use in differentiation between the RCA and the LCX. Sensitivity and specificity to predict the infarct related artery were 33% and 71%, respectively.

Gupta et al.[26] conducted a similar study and found 69% sensitivity, 85% specificity of ST segment depression in determining LCx involvement in patients of acute inferior wall myocardial infarction. The negative predictive value was 87% and positive predictive value was 66%.

CONCLUSION

ST depression in lead aVR has a good sensitivity and specificity (91.8% and 89.4% respectively) in determining the left circumflex artery (LCx) artery involvement in patients of acute inferior wall myocardial infarction.

Author's Contribution:

Concept & Design of Study:	Abubakr Ali Saad
Drafting:	Tariq Abbas
Data Analysis:	Saadat Hussain Khan Khakwani
Revisiting Critically:	Abubakr Ali Saad, Muhammad Amin
Final Approval of version:	Tariq Abbas

Conflict of Interest: The study has no conflict of interest to declare by any author.

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