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The predictive value of aVR in determining the infarct related artery during primary percutaneous coronary intervention

Ayman Gaballa [a], Wala Farid [b], Ahmad Al-Kersh [b], Rajesh Rajan [a,*], Mohammed Al Jarallah [a]

[a] Department of Cardiology, Sabah Al-Ahmad Cardiac Center, Al-Amiri Hospital, Kuwait
[b] Department of Cardiology, Faculty of Medicine, Menoufia University, Egypt

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ABSTRACT

Background: Isolation of infract related artery and timely revascularisation remains vital in the setting of primary percutaneous coronary intervention.
Objectives: To analyse the predictive value of ST-T changes in lead aVR in inferior myocardial infarction in terms of prognosis and timely risk stratification.
Methods: We conducted a prospective analysis of acute inferior wall myocardial infarction patients. One hundred patients were categorised into two groups according to the culprit artery: group I, right coronary artery (RCA) and group II, left circumflex coronary artery (LCX), with 50 patients in each group. A comparative study was performed between the two groups, comprising the following data outputs: electrocardiogram (ECG) changes that could help determine the culprit artery, cardiac enzyme levels, echocardiographic findings, coronary angiography findings and in-hospital complications. The same patients were divided into two groups according to the presence or absence of 1 mm ST depression in lead aVR. A comparison analysis was performed between the two groups including: cardiac enzyme levels, echocardiographic findings, coronary angiography findings and in-hospital complications.
Results: ST depression in aVR ≥ 1 mm predicted the LCX as a culprit artery with sensitivity, specificity, positive predictive values (PPV) and negative predictive values (NPV) recorded at 66%, 84%, 80.5% and 71.2%, respectively. Also, patients with ST depression in aVR ≥ 1 mm showed significantly higher cardiac enzyme levels, indicating larger infarct size, with mean peak creatinine kinase (CK) = 1560 (1057–2375) IU/L versus 970 (613–1683) IU/L, (P value = 0.014), lower ejection fraction (Ef) with mean Ef = 47.93 ± 8.04 versus 54.66 ± 6.52, (P value < 0.001) and more significant mitral regurgitation: 17 (41.5%) patients versus 11 (18.6%) patients (P value = 0.012). Regarding in-hospital complications, there were no significant differences.
Conclusions: ST depression of >1 mm in lead aVR predicts LCX as the infarct related artery and is a predictor of poor outcome in patients with inferior myocardial infarction.

Introduction

The incidence of inferior ST elevation myocardial infarction (STEMI) is approximately 40% when compared to other STEMI presentations. The condition has a lower risk of complication than anterior STEMI [1]. The mortality rate of inferior STEMI is approximately 10% or less. However, about one third of patients with inferior myocardial infarction (MI) may develop in-hospital complications, associated with poor outcomes [2].

For inferior MI, the infarct related artery may be either the RCA or the LCX. The location of the occlusion may determine patient mortality and morbidity [3]. In the presence of multi-vessel disease, where both arteries are diseased, identification of the infarct-related artery (IRA) during a primary percutaneous coronary intervention (PCI) is challenging. To identify the IRA in acute inferior wall myocardial infarction (IWMI), multiple ECG criteria are available [4,5].

Materials and methods

We performed a prospective analysis of 100 IWMI patients admitted to hospital from 2013 to 2016. We included all patients presenting with the following criteria: acute inferior myocardial infarction for the first time and patients who underwent revascularisation within 12 h of the onset of symptoms, by primary PCI. We excluded the following patients: those with a history of myocardial infarction, patients whose coronary angiography showed significant lesions in both RCA and LCX, patients with a history of previous PCI or coronary artery bypass grafts (CABG), patients with a left bundle branch block (LBBB), a right bundle branch block (RBBB), left ventricular hypertrophy (LVH) or paced rhythms

* Corresponding author.
E-mail address: cardiology08@gmail.com (R. Rajan).

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and life-threatening non-cardiac illness. In the presence of CAD with significant lesions, it may be confusing for the interventionist to identify the culprit artery. The culprit lesion is defined by acute total occlusion, significant stenosis ≥ 70%, intraluminal thrombus, or ulcerated plaque. Left ventricular hypertrophy cases were excluded by ECG criteria to avoid changes in the lead I. ST depression was measured 60 ms after J point.

Patients were divided into two groups according to the IRA. Group I consisted of 50 patients with RCA as the culprit artery and Group II was 50 patients with LCX as the culprit artery. A comparative analysis of the two groups was carried out.

Statistical analysis

For statistical analysis, predictive analytics software (PASW Statistics 18) was used. Associations between categorical variables were tested using the Chi-square test. When >20% of cells had expected counts of less than five, corrections for the Chi-square test were conducted using Fisher's Exact test or the Monte Carlo correction. Quantitative data were described using median, minimum and maximum, as well as mean and standard deviation. The distribution of quantitative variables was tested for normality using the Kolmogorov-Smirnov test and the Shapiro-Wilk test. The D'Agostino test was used if there was a conflict between the two previous tests. If this test revealed a normal data distribution, parametric tests were applied. If the data were abnormally distributed, non-parametric tests were used. For normally distributed data, comparisons between two independent populations were performed using independent *t*-tests. For abnormally distributed data, the Mann-Whitney Test (for data distributions significantly deviated from normal) was used to analyse the two independent populations.

Significant test results were quoted as two-tailed probabilities. The significance of all results was judged at the 5% confidence level.

Results

The cohort of 100 patients, presenting with inferior MI, were divided into two groups according to the IRA, as indicated during the primary percutaneous coronary intervention (PPCI). Groups I (RCA) and II (LCX) comprised 50 patients each. There were no statistical differences between the two groups in terms of baseline characteristics and risk factors.

For the 12 lead ECG, the following findings were recorded: S-T segment elevation in lead III > II, S-T segment depression in lead I ≥ 0.5 mm, ST-segment depression in lead aVR ≥ 1 mm, ST segment elevation in lead V4R ≥ 0.5 mm (Table 1). A total of eight patients (16%) in group I had ST segment depression ≥ 1 mm, whereas 42 patients (84%) had either ST segment elevation or isoelectric or depressed < 1 mm in lead aVR. Group II identified 33 (66%) patients with ST segment depression ≥ 1 mm in lead aVR and 17 (34%) patients had either ST segment elevation or isoelectric or depressed < 1 mm in lead aVR, (P value < 0.001).

It was concluded that ST segment depression in lead aVR ≥ 1 mm was a good predictor for identifying LCX as a culprit artery for inferior STEMI in terms of sensitivity, specificity, PPV and NPV, recorded at 66%, 84%, 80.5% and 71.2%, respectively (Table 2). Regional wall motion abnormalities in basal segments of inferoseptal segments, confirmed by echocardiography, can be used as predictors of RCA as IRA with sensitivity, specificity, PPV and NPV for prediction of RCA occlusion, recorded at 100%, 74%, 79.4% and 100%, respectively (Table 3).

This study demonstrates that patients with ST depression in aVR ≥ 1 mm have (i) significantly higher cardiac enzyme levels, indicating larger infarct size with mean peak CK levels = 1560 (1057–2375) IU/L versus 970 (613–1683) IU/L, (P value = 0.014) and mean peak creatinine kinase muscle/brain (CKMB) levels = 132 (67–248) IU/L versus 78.3 (52–135) IU/L, (P value = 0.008) (Table 4), (ii) decreased ejection fractions with mean Ef = 47.93 ± 8.04 versus 54.66 ± 6.52, (P value < 0.001) (Table 5), (iii) increased incidence of significant mitral regurgitation: 17 (41.5%) patients versus 11 (18.6%) patients, (P value = 0.012) (Table 5).

Discussion

This study focused mainly on the identification of IRA in acute IWMI, using ECG. It was evident from the results that ST depression in aVR ≥ 1 mm was a good predictor for LCX as a culprit artery in patients with inferior STEMI. It also had a prognostic role, as patients with ST depression in aVR ≥ 1 mm were shown to have poor outcomes. Regarding the prognostic value of ST depression in aVR, our study demonstrated that patients with ST depression in aVR ≥ 1 mm had significantly higher cardiac enzyme levels, indicating larger infarct size. In patients with ST depression ≥ 1 mm in aVR, patients had lower EF with incidences of moderate mitral regurgitation. Here, we demonstrated that there were no significant relationships between deviation of the ST segment in lead aVR and the extent of CAD.

Menown et al. reported the display of lead aVR (−150°) in inverted format, as lead −aVR (+30°) bridged the gap between lead I (0°) and lead II (60°). In other words, ST-segment depression in lead aVR may have been a reciprocal change, resulting from ST-segment elevation in the apical and inferolateral walls, which is not an issue with the standard 12 leads. Such regions of the left ventricle are usually supplied by the large posterolateral branch of the LCX or the atrioventricular branch of the RCA. Therefore, concurrent ST-segment depression in lead aVR during inferior AMI may reflect transmural ischemia extending to the apical and inferolateral walls, in addition to the inferior wall [6]. Manohara et al. studied the importance of lead AVR in our clinical practice and its utility [7].

With regard to the utility of ST deviation in aVR in predicting culprit lesions, Nair et al. demonstrated that as predictors of LCX as culprit lesions, ST segment depression ≥ 1 mm in lead aVR had sensitivity, specificity, PPV and NPV for the prediction of LCX occlusions at 80%, 96%, 80% and 96%, respectively. As a predictor of RCA as a culprit lesion, the absence of ST segment depression ≥ 1 mm in lead aVR had a sensitivity, specificity, PPV and NPV for RCA occlusion prediction of 96%, 80%, 96% and 80%, respectively [3]. These results were very similar to the data presented here.

Baptista et al. found that lead aVR ST depression showed limited use in differentiation between the RCA and the LCX; the sensitivity, specificity, PPV and NPV of these criteria were 33%, 71%, 31% and 73%, respectively. Although slightly higher in patients with circumflex occlusion (33.3%) when compared to those with right coronary occlusion (28.9%), ST-segment depression in aVR presented low sensitivity

Table 1
ECG changes in group (I) and group (II).

	Group I (RCA)	Group II (LCX)	Test value[a]	P-value
↓ ST in aVR ≥ 1 mm, n(%)	8 (16.0%)	33 (66.0%)	25.837	0.001
↑ ST in III > II, n(%)	36 (72.0%)	21 (42.0%)	9.180	0.002
↓ ST in Lead I ≥ 0.5 mm, n(%)	33 (66.0%)	16 (32.0%)	11.565	0.001
↑ ST in V4R ≥ 0.5 mm, n(%)	12 (24.0%)	2 (4.0%)	8.306	0.003

NS: non-significant; S: significant; HS: highly significant.
[a] Chi-square test.

Table 2
Criteria for predicting LCX occlusion.

	Sensitivity	Specificity	PPV	NPV	Accuracy
↓ ST in aVR ≥ 1 mm	66.0%	84.0%	80.5%	71.18%	75.0%
↑ ST in II ≤ III	58.0%	72.0%	67.4%	63.16%	65.0%
No ↓ ST in Lead I ≥ 0.5 mm	68.0%	66.0%	66.7%	67.3%	67.0%
No ↑ ST in V4R ≥ 0.5 mm	96.0%	24.0%	55.8%	85.7%	60.0%

Table 3
Criteria for predicting RCA occlusion.

	Sensitivity	Specificity	PPV	NPV	Accuracy
No ↓ ST in aVR ≥ 1 mm	84.0%	66.0%	71.18%	80.5%	75.0%
↑ ST in III > II	72.0%	58.0%	63.16%	67.4%	65.0%
↓ ST in Lead I ≥ 0.5 mm	66.0%	68.0%	67.3%	66.7%	67.0%
↑ ST in V4R ≥ 0.5 mm	24.0%	96.0%	85.7%	55.8%	60.0%
Basal inferoseptal RWMA	100.0%	74.0%	79.4%	100.0%	87.0%

(33%) and no more than reasonable specificity (71%). Differences between these two groups did not reach statistical significance (P = 0.05) [8]. These results do not correlate to our study. This may be due to the small number of patients in the study, or the prevalence of ST depression in aVR was very low in both groups.

The study by Sun Tong-Wen et al. supported the study by Nair and Glancy and correlated with this study. This study included 90 patients with acute inferior wall STEMI. Patients were divided into two groups according to the culprit artery. ST depression in aVR ≥ 0.1 mV was found in 14 (70%) patients who had LCX as the IRA, and in four (5.7%, P < 0.001) patients with RCA as the IRA. Using ST segment depression ≥ 0.1 mV in aVR as a criterion, the sensitivity, specificity, PPV and NPV for prediction of LCX occlusion were 70%, 94.3%, 77.8% and 91.6% respectively [9].

Kanei et al. studied ST-segment depression in aVR as a predictor of culprit artery and infarction size in acute inferior wall ST-segment elevation myocardial infarction. The study demonstrated that ST-segment depression in lead aVR ≥ 1 mm can be used as a predictor of LCX IRA, with sensitivity, specificity, PPV and NPV of 53%, 86%, 45% and 91%, respectively [10]. These results were very similar to those presented in this study.

Ravi Sahi et al. studied the clinical implication of ST segment depression in aVR and aVL in patients with acute inferior wall myocardial infarction [10]. This study concluded that ST depression in lead aVR ≥ aVL helped to diagnose left circumflex artery as a culprit IRA, in acute inferior wall MI [11].

Mahmoud et al. studied the significance of ST-segment deviation in lead aVR for the prediction of culprit artery and infarct size in acute inferior wall ST-elevation myocardial infarction [12]. The study of 50 patients with acute inferior myocardial infarction divided patients into two groups; Group A: patients with ST segment depression in lead aVR ≥ 1 mv and Group B: patients with isoelectric ST segment or with ST segment depression in lead aVR < 1 mv. The study demonstrated that ST-segment depression in lead aVR ≥ 1 mm was a predictor of LCX with sensitivity, specificity, PPV and NPV of 67%, 72%, 47% and 85% respectively [12]. These results were consistent with data from our study.

Our study supports the findings of Kosuge et al., where the ST-segment depression in aVR was cited as a useful predictor of impaired myocardial reperfusion and infarct size in patients with inferior acute wall myocardial infarction. This study included 225 patients and reported that ST-segment depression in lead aVR > 1 mm was associated with a larger infarction size (CPK 4.865 ± 1.757, P ≤ 0.001) and impaired myocardial reperfusion (defined as myocardial blush grade 0/1). It was observed in 67% of patients with ST-segment depression in lead aVR > 1 mm, compared with only 2% of patients without ST depression in aVR. Multivariate analysis showed that the degree of ST-segment depression in lead aVR was an independent predictor of impaired myocardial reperfusion (odds ratio 8.41; 95% confidence interval, 2.96 to 23.9; P < 0.001) [13].

Kanei et al. reported that patients with aVR depression had significantly larger infarctions, as estimated by peak creatinine phosphokinase (CPK) levels, when compared to patients without aVR depression (CPK 3151 ± 2682 U/L versus 1933 ± 1954 U/L, P = 0.0169) [10].

Kukla et al. studied the prognostic significance of ST segment changes in lead aVR in patients with acute inferior myocardial infarction with ST segment. The study included 320 consecutive patients with inferior wall STEMI. It was observed that ST segment depression in aVR was present in 26.6% of the patients. Those with ST segment depression in lead aVR had higher CPK levels when compared to patients with no ST segment depression in lead aVR (2375 ± 2012 versus 1563 ± 1422 U/L, P = 0.004) and they had higher death rates (16.5% versus 1.0%), higher composite end-points (27.0% versus 3.2%) and higher VF (12.1% versus 4.8%) when compared to patients with no ST segment changes in lead aVR (P < 0.001 for all) [14].

These findings correlated with our study results regarding the infarct size, as reflected by CKP levels. With regard to in-hospital complications, our study showed no statistical significance between the ST segment deviation and in-hospital complications (P value = 0.18). This difference may have been due to the small number of patients in our study (n = 100) when compared to numbers in other studies, e.g. 320 patients in the study by Kukla et al. Also, in our study there was one method of therapy, primary PCI within 12 h of symptom onset, while in the other study there were 3 different therapy options: primary PCI, fibrinolytic therapy and conservative therapy.

Sohrabi et al. studied 150 patients with inferior STEMI. The aim of this study was to compare prognostic differences between LCX- and RCA-related acute inferior wall STEMI, treated by routine adjunctive angioplasty, after receiving thrombolytic therapy (TLT). RCA and LCX

Table 5
Relation between ST segment deviation in aVR and echocardiographic findings in the 2 groups.

	↓ ST in aVR ≥ 1 mm		Test value	P-value
	Without ↓ ST = 59	With ↓ ST = 41		
Ejection fraction at discharge (mean ± SD)	54.66 ± 6.52	47.93 ± 8.04	4.612[b]	0.001
RV dysfunction, n(%)	11 (18.6%)	4 (9.8%)	1.499[a]	0.221
Basal inferoseptal RWMA, n(%)	46 (78.0%)	17 (41.5%)	13.827[a]	0.001
Lateral RWMA, n(%)	11 (18.6%)	16 (39.0%)	5.098[a]	0.024
Inferior RWMA, n(%)	59 (100.0%)	41 (100.0%)	NA[a]	NA
Moderate MR, n(%)	11 (18.6%)	17 (41.46%)	6.248*	0.012

NS: non-significant; S: significant; HS: highly significant.
[a] Chi-square test.
[b] Independent *t*-test.

Table 4
Relation between ST segment deviation in aVR and cardiac enzymes.

		↓ ST in aVR ≥ 1 mm		Test value	P-value
		Without ↓ ST = 59	With ↓ ST = 41		
Peak CK level (IU/L)	Median (IQR)	970 (613–1683)	1560 (1057–2375)	−2.467	0.014
	Range	289–2943	265–3412.7		
Peak CKMB level (IU/L)	Median (IQR)	78.3 (52–135)	132 (67–248)	−2.656	0.008
	Range	26–375	21–432		

arteries were occluded in 97 (64.7%) and 53 (35.3%) patients, respectively. The two groups were similar in terms of baseline characteristics, except that multiple-vessel disease was more prevalent in the LCX occlusion cohort (P = 0.008). These results demonstrated that patients with LCX IRA had a higher cardiac CKMB release (CKMB, 246.7 ± 132.3 versus 161.0 ± 105.0, P value < 0.001), they had significant mitral regurgitation (18 (34%) versus 16 (16.5), P value = 0.015) and they had lower left ventricular ejection fraction (LVEF) (43.3 ± 6.5 versus 55.8 ± 45.9, P value = 0.01). Multivariate analysis showed cardiac troponin I (cTn-I) release, the occurrence of MR, and lower LVEF as independent factors leading to poor outcomes [15].

Mahmoud et al. studied infarct size in acute inferior wall ST-elevation myocardial infarction. The study included 50 patients with acute inferior myocardial infarction. In this study, patients with ST segment depression in lead aVR ≥ 1 mv had larger infarct size (CKMB = 156.2 ± 91.5 versus 33.5 ± 91.3, P < 0.002) and lower Ef (53.7 ± 8.7 versus 59.2 ± 7.8, P = 0.03) [12]. These results were in correlation with the results of this study. Similarly, Bahram Sohrabi et al. studied 150 patients with inferior STEMI. Patients were divided into two groups according to the culprit lesion RCA or LCX. The two groups were similar in baseline characteristics, except that multiple-vessel disease was more prevalent in the LCX occlusion cohort, with highly significant P values = 0.008 [15].

Kosuge et al. studied ST-segment depression in aVR as a useful predictor of impaired myocardial reperfusion and infarct size in patients with inferior acute wall myocardial infarction. In this study, 225 patients were divided into three groups according to ST-segment deviation in lead aVR: group A, no ST-segment depression; group B, ST-segment depression of ≤1.0 mm; and group C, ST-segment depression of >1.0 mm. The study showed no differences between the three groups for incidences of multi-vessel disease, concomitant left anterior descending coronary artery disease, infarct-related artery, proximal lesion of the infarct related artery, or TIMI flow grade 0 at initial coronary angiography. Patient numbers with multi-vessel disease in groups A, B, C were 26 (25%) 17 (21%) 10 (24%), respectively, with a non-significant P value = 0.819 [13]. Salah et al. studied the association of discordant T waves in lead aVR and its prognostic value in ischemic cardiomyopathy related sudden cardiac arrest [16].

Our results were similar to the study by Khaled Sayed et al., which showed that of 17 patients with ST depression in aVR ≥ 1 mm, six had multi-vessel disease, while in 33 patients without ST depression in aVR ≥ 1 mm, 20 had multi-vessel disease with non-significant P values = 0.22 [10]. It is important to note that aVR, as a predictor of culprit lesions, is comparable to other ECG changes in leads I, II, III V4R as investigated by other studies. However, in our study, it was discovered that regional wall motion abnormality in the basal segment of the inferoseptal wall can be a good predictor of RCA as culprit lesions, with sensitivity, specificity, PPV and NPV for prediction of RCA occlusions at 100%, 74%, 79.4% and 100%, respectively. Unfortunately, this phenomenon has not been extensively studied and therefore requires intensive investigation in large cohort, well-controlled studies.

Conclusions

Our study has shown that ST segment deviation in aVR is a good predictor in determining the infarct related artery in patients with inferior STEMI. ST segment depression in aVR ≥ 1 mm had a prognostic role in patients with inferior STEMI with larger infarct sizes, lower Efs and more significant mitral incompetence.

Limitations

One of the major limitations of this study was its small sample size. The estimated systolic function of all patients was assessed in the first day of infarction, with no follow-up. The study used the CK as a method to assess infarction size, which lacks accuracy. The study focused on presence or absence of ST depression in aVR, neglecting the importance of ST elevation in aVR. There was a lack of long-term follow-up of patients with poor outcome.

Competing interests

The authors declare that they have no competing interests.

Author's contributions

AG participated in data acquisition and manuscript preparation. WF participated in the data analysis and drafting of manuscript. AAK participated in data analysis and manuscript preparation. RR participated in data analysis and manuscript preparation. All authors had access to data and take responsibility for the integrity of data and the accuracy of data analysis. All authors have read and approved the manuscript.

Ethical standards

We performed this study in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki and its later amendments. Informed consent was taken prior to the recruitment.

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