The prognostic value of a prominent Q wave in lead (–)aVR in acute anterior wall myocardial infarction

Akut ön duvar miyokart enfarktüsünde (–)aVR derivasyonunda belirgin Q dalgasının prognostik değeri

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ABSTRACT

Objective: This study aimed to determine the association of a prominent Q wave in lead (–)aVR with clinical, echocardiographic and angiographic findings in anterior ST elevation myocardial infarction (STEMI) and to evaluate the role of this finding in short-term and long-term outcomes.

Methods: During a one-year period, 150 patients with first time anterior STEMI were screened and 121 patients with no other cardiopulmonary and renal comorbid diagnoses were included in the study. Patients were allocated into two groups based on presence or absence of a prominent Q wave in lead (–)aVR. All clinical, electrocardiographic, echocardiographic and angiographic data were recorded and compared between the groups. In-hospital adverse outcomes and mortality as well as two-year survival were also compared.

Results: Among 121 patients (mean age: 62.8±12.5 years) 26.4% had a prominent Q wave in lead (–)aVR. The prevalence of multi-vessel disease was higher in patients with a Q wave (76.9% *vs.* 52.8%, p=0.03). ST-segment elevation in lead V6 was significantly more common in those with a Q wave (50% *vs.* 30.3%, p=0.04). Posterobasal region motion abnormality was more common in the Q wave group. (9.4% *vs.* 1.2% respectively, p=0.04). Overall, mortality was higher in the Q wave group; however, it was not statistically significant (15.4% *vs.* 9.3%, p=0.39).

Conclusion: In anterior STEMI, presence of a Q wave in lead (–)aVR is associated with occlusion of multiple arteries. Short- and mid-term mortality are not affected by this ECG finding.

ÖZET

Amaç: Bu çalışma ön duvar ST-segment yükselmeli miyokart enfarktüsünde (STYME) (–)aVR derivasyonunda belirgin Q dalgasıyla ilişkili klinik, ekokardiyografik ve anjiyografik bulguları belirlemeyi ve bu bulgunun kısa ve uzun dönemli sonuçlardaki rolünü değerlendirmeyi amaçlamıştır.

Yöntemler: Bir yıllık dönem boyunca ilk kez ön duvar STYME'si geçiren 150 hasta tarandı, çalışmaya başka bir kardiyopulmoner ve renal komorbidite tanısı olmayan 121 hasta alındı. Hastalar (–)aVR derivasyonunda belirgin Q dalgası varlığı veya yokluğuna göre iki gruba ayrıldı. Klinik, elektrokardiyografik, ekokardiyografik ve anjiyografik veriler kaydedildi ve gruplar arasında karşılaştırıldı. Hastanede yatış sırasında oluşan olumsuz sonuçlar, mortaliteyle birlikte iki yıllık sağkalım oranları da karşılaştırıldı.

Bulgular: Yüz yirmi bir hastanın %26.4'ünde (yaş ortalaması: 62.8±12.5 yıl) (–)aVR derivasyonunda belirgin bir Q dalgası mevcuttu. Çok damarlı hastalığın prevalansı Q dalgası olan hastalarda daha yüksek idi (%76.9 ve 52.8%, p=0.03). V6 derivasyonunda ST-segment yükselmesi, Q dalgası olanlarda anlamlı derecede daha sık görülmekteydi (%50.2 ve %30.3, p=0.04). Posterobasal bölgede hareket anormalliği Q dalgası grubunda daha sık görülmekteydi (%9,42 ve %1.2, p=0.04). Genelde mortalite Q dalgası grubunda daha yüksek olmasına karşın anlamlı bulunmadı (%15.4 ve %9.3, p=0.39).

Sonuç: Ön duvar STYME'de (–)aVR derivasyonunda Q dalgasının varlığı birden çok arterin oklüzyonuyla ilişkilidir. Kısa ve orta vadede mortalite bu EKG bulgusundan etkilenmemektedir.

Standard 12-lead electrocardiography (ECG) has been in use for over 70 years and still plays a crucial role in modern clinical cardiology, especially in the diagnosis of acute myocardial infarction. This study provides information on cardiac electrical function on two separate planes, each consisting of six leads.[1,2] In contrast to the horizontal plane, there are variations in conventional placement of leads on the

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frontal plane due to presentation format. There is a 60° gap between lead I and lead II and a 90° gap between lead III and lead aVR. Moreover, the most substantial inadequacy is the non-comprehensive and inconsistent view of the limb leads grouped on this plane, which results in a puzzling progression in waveform morphology.

Abbreviations:

ECG	*Electrocardiography*
LMCA	*Left main coronary artery*
LV	*Left ventricle*
LVEDD	*Left ventricular end diastolic diameter*
LVEF	*Left ventricular ejection fraction*
ms	*Milliseconds*
RWMA	*Regional wall motion abnormality*

These issues may be resolved by simply inverting the aVR lead ((–)aVR) and changing exposition of the hexaxial lead system to an orderly, sequential position (aVL, I, (–)aVR, II, aVF, and III), resulting in a panoramic view of the frontal plane.[3–5] As a consequence of its atypical mien and position, lead aVR, which obtains information from the right upper section of the heart, has generally been considered a non-informative lead in the interpretation of electrocardiograms in a clinical setting. It is thought to provide only repetitious information from the left lateral side, already covered by other leads.[6,7] This new position and appearance of lead (–)aVR contains many advantages, including mitigating all the above-mentioned shortcomings.[5,8] Recently, researchers have shown increasing interest in lead aVR and (–)aVR with regard to diagnosis[9,10] and risk stratification of acute coronary syndrome.[4,11,12] In one study, Kotoku et al.[13] found that a prominent Q wave in lead (–)aVR— defined as a Q wave of 20 milliseconds (ms) or longer—in patients with anterior ST elevation myocardial infarction (STEMI), is related to severe regional wall motion abnormality in the apical and inferior regions, with the left anterior descending artery (LAD) wrapping around the apex. This is the only study in the literature to investigate the value of a Q wave in lead (–)aVR in anterior STEMI.

The present study evaluated the effects of a prominent Q wave in lead (–)aVR on treatment course, echocardiographic and angiographic findings and two-year mortality in anterior STEMI.

METHODS

The study protocol was approved by the Committee on Research Ethics at Tabriz University of Medical Sciences and registered as thesis project No. 89.1-11.18. The study was waived from obtaining informed consent due to its nature and minimal risk to the participants. Participant privacy was maintained throughout the study period.

Study population and design

Between March 2010 and March 2011, 150 patients admitted to Madani Cardiovascular University Hospital with a diagnosis of anterior STEMI were retrospectively reviewed and included in the study based on the criteria of[1] typical ischemic chest pain lasting 20 minutes or longer and[2] ST-segment elevation in 2 or more adjacent precordial leads (≥2 mm). Twenty-nine patients were excluded because of[1] previous history of MI,[2] electrocardiographic findings of bundle branch block, intraventricular conduction disturbance, Wolff-Parkinson-White syndrome, or ventricular rhythm,[3] any other associated cardiac or lung disease affecting ECG findings, and[4] history of pace maker implantation. Electrocardiograms from the remaining 121 patients were investigated. Since electronic inversion of lead avR at the time of recording was not possible with the available 12-lead ECG machines, (–)aVR was obtained by vertical inversion of a scanned image of convention al lead avR. A prominent Q wave in (–)aVR was defined as a wave of 20 ms or longer.[13] Figure 1 shows an example of ECG with and without a prominent Q wave in lead (–)aVR. Electrocardiograms were studied for the presence of a prominent Q wave in lead (–)aVR and were assigned to either one of the two groups based on presence of a prominent Q wave (Group A) or absence of a Q wave (Group B). Demographic data, cardiovascular risk factors, results of findings in physical exam, course of treatment, electrocardiographic, as well as laboratory, echocardiographic and angiographic findings were collected and recorded. The patients were stratified for risk of 30-day and 2-year mortality using the Killip classification and risk class was recorded for each individual patient as follows:

- Killip class I: No clinical signs of heart failure.
- Killip class II: Rales or crackles in the lungs, an S3, and/or elevated jugular venous pressure.
- Killip class III: Frank acute pulmonary edema.
- Killip class IV: Cardiogenic shock or hypotension (measured as systolic blood pressure lower than 90 mmHg), and evidence of peripheral vasoconstriction (oliguria, cyanosis or sweating).

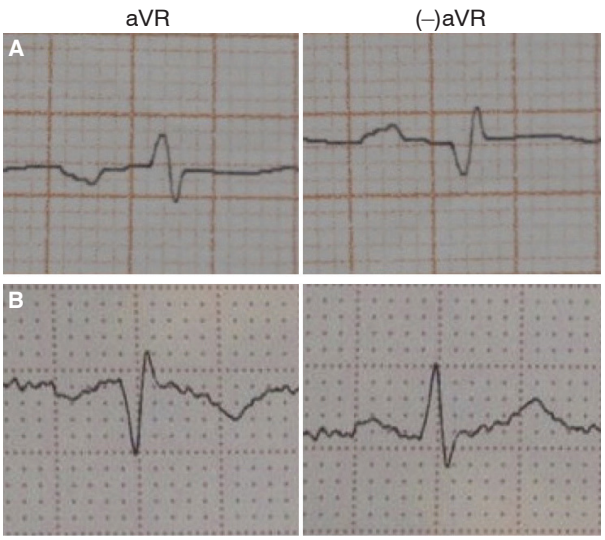

Figure 1. (A) Illustrates a case with prominent q wave in lead (–)aVR after anterior myocardial infarction, whereas **(B)** shows a patient without q wave in lead (–)aVR.

In-hospital occurrence of any adverse event including death was recorded. Additionally, the patients or their family members were contacted by telephone for 2-year follow up.

Electrocardiogram

The standard 12-lead ECG was recorded at a paper speed of 25 mm/s and a standardization of 10 mm = 1 mV. The magnitude of ST-segment elevation or depression was measured at the J point. The J point was determined for each lead independently. Both ST-elevation and ST-segment depression were measured at 80 ms after the J point in all leads. All tracings were evaluated separately by 2 investigators blinded to the echocardiographic and angiographic findings and final outcome. According to the universal definition of myocardial infarction, an abnormal Q wave is defined as any Q wave of 20 ms or longer in leads V2–3, or 30 ms or longer and 1 mm or more in depth in other leads.[14] A prominent Q wave in (–)aVR was defined as a wave of 20 ms or longer.[13]

Coronary angiography

Coronary angiography was performed during first week of admission and in most cases it was a delayed angiography. Multi-vessel disease was defined as the presence of luminal diameter stenosis of greater than 50% in at least 2 major coronary arteries. In addition to multivessel disease, left main coronary artery (LMCA) involvement and proximal LAD stenosis were recorded. The angiographic data were evaluated by consensus between two observers blinded to other data.

Echocardiography

Various clinicians performed two-dimensional echocardiography for most of the patients according to the guidelines for echocardiography reporting at our institutions. Regional wall motion abnormality (RWMA), left ventricular ejection fraction (LVEF) calculated with modified Simpson's method, left ventricular end diastolic diameter (LVEDD), severity of mitral regurgitation and pericardial effusion were quantified and recorded.

Statistical analysis

All analyses were performed using SPSS 17.0 software (IBM® Inc., Chicago, IL) for Windows. The Kolmogorov-Smirnov test was used to examine the normality of the continuous data and the normality was rejected with a p-value <0.01. Data were expressed as means±SD for continuous variables with (normal distribution), and as median (Interquartile range) for continuous variables where normal distribution was rejected. Numerical values with normal distribution were analyzed with two-tail independent t-tests and those that did not have a normal distribution were compared using Mann-Whitney U tests. Categorical data were presented as N (percent) and compared with 2x2 contingency tables and Fisher's exact tests. Receiver operator characteristic analyses were performed and the sensitivity, specificity, positive and negative predictive values, and diagnostic accuracy were calculated. P-value <0.05 was considered statistically significant.

RESULTS

Demographic characteristics (Table 1)

Eighty-five men and 36 women (age range: 30 to 89 years) were enrolled. Of the 121 patients, 32 had a prominent Q wave in lead (–)aVR (26.4%). The mean age of patients was 62.4±13.2 years in group A and 62.9±12.4 years in group B (p=0.91). Gender distribution was also similar between the groups (71.9% male in group A and 69.7% male in group B; p=0.82). The prevalence of hypertension, diabetes mellitus, hyperlipidemia, smoking and family history of premature coronary heart disease was not significantly different between the groups.

Table 1. Comparison of demographic data

	Total (n=121)	Group A (n=32)	Group B (n=89)	*p*
Age (years), (Mean±SD)	62.8±12.5	62.4±13.2	62.9±12.4	0.91
Sex (male), n (%)	85 (70.2)	23 (71.9)	62 (69.7)	0.83
Hypertension, n (%)	64 (52.9)	15 (46.9)	49 (55.1)	0.42
Hyperlipidemia, n (%)	35 (28.9)	12 (37.5)	23 (25.8)	0.22
Diabetes mellitus, n (%)	31 (25.6)	8 (26)	23 (25.8)	0.99
Family history of CAD, n (%)	16 (13.2)	4 (12.5)	12 (13.5)	0.88
Active smoking, n (%)	48 (39.7)	10 (31.3)	38 (42.7)	0.15

Group A: Patients with Q wave in lead (–)aVR and Group B: Patients without Q wave in lead (–)aVR. P-value <0.05 is considered significant.

Electrocardiographic findings (Table 2)

ST segment elevation and ST segment depression in lead (–)aVR on admission ECG were present in 11.6% and 26.4% of all patients respectively. As noted, 26.4% of patients developed a prominent Q wave in (–)aVR. The most common lead with an abnormal Q wave on pre-discharge ECG was lead V2 (78.5%) and the least common lead with an abnormal Q wave was lead V6, in which only 5% of patients had abnormal Q wave. The presence of an abnormal Q wave in any lead other than (–)aVR was similar in both groups.

Comparison of the groups revealed no significant differences between them regarding the incidence of ST elevation ≥1 mm in any lead other than V6. The frequency of ST elevation in lead V6 was higher in Group A than in B (50% vs 30.3% respectively, p=0.04). Five of 32 (15.6%) patients in group A and 9 of 88 (10.1%) patients in group B had ST elevation ≥1 mm in (–)aVR on admission ECG; however, the difference was not statistically significant (p=0.4). The extent of ST-segment deviation in each 12-lead was similar in the two groups (Table 2). The number of leads with significant ST elevation on admission ECG, as well as pre-discharge ECG was similar in the two groups. The number of leads with ST-segment depression on admission ECG was also similar in two groups (Table 2).

Echocardiographic and angiographic findings (Table 3)

Group A and Group B had similar LVEF (38.3 ± 10.3% vs 40.3 ± 8.7% respectively, P=0.21). The presence of RWMA was similar in Group A and Group B (87.5% vs 96.5% respectively; P=0.08). Assessment of distribution of RWMA revealed a higher frequency of posterobasal region involvement in Group A compared to Group B (9.4% vs 1.2% respectively; P=0.04). Involvement of anteroseptal, anterolateral, and apical and inferior walls was not significantly different between the groups. Frequency and severity of mitral regurgitation and elevation of LVEDD were also similar in both groups.

Patients in Group A had a higher prevalence of multi-vessel disease in coronary angiography than patients in Group B (76.9% vs 52.8% respectively, p=0.03). A left main coronary artery lesion was present in 7.7% of patients in Group A and in 5.6% of patients in Group B, with no significant difference (p=0.06). The prevalence of proximal LAD lesions was also similar between Group A and Group B (65.4% vs. 79.2%, respectively, p=0.16). A similar proportion of the patients in each group underwent primary percutaneous intervention (46.2% in group A and 66.7% in group B; P=0.07).

Clinical outcomes and laboratory findings (Table 4)

There was a similar frequency of pulmonary edema, cardiogenic shock, ventricular dysrhythmias and re-infarction in both groups. Based on Killip criteria, severity of myocardial infarction was similar in the two groups, as was therapeutic response to reperfusion therapies. Length of stay in hospital and in the coronary care unit was similar in both groups. In-hospital mortality rates were 3.1% in group A and 5.6% in group B (p=0.99). Two-year mortality rates were 9.7% in group A and 2.4% in group B (p=0.21).

Table 2. Comparison of electrocardiographic (ECG) findings

		Total (n=121)	Group A (n=32)	Group B (n=89)	p
STE ≥1mm on admission ECG, n (%)	aVL	30 (24.8)	8 (25)	22 (24.7)	0.93
	I	29 (24)	9 (28.1)	20 (22.5)	0.53
	(–) aVR	14 (11.6)	5 (15.6)	9 (10.1)	0.42
	II	21 (11.6)	9 (28.1)	12 (13.5)	0.06
	aVF	14 (11.6)	4 (12.5)	10 (11.2)	0.83
	III	20 (16.5)	8 (25.0)	12 (13.5)	0.12
	V1	112 (92.6)	31 (96.9)	81 (91)	0.21
	V2	121 (100)	32 (100)	89 (100)	–
	V3	120 (99.2)	32 (100)	88 (98.9)	0.92
	V4	106 (87.6)	25 (78.1)	81 (91)	0.06
	V5	83 (68.6)	24 (75)	59 (66.3)	0.31
	V6	43 (35.5)	16 (50)	27 (30.3)	0.04*
ST-Deviation on admission ECG (mm), (Mean±SD)	aVL	0.4±0.1	0.4±0.1	0.4±0.1	0.98
	I	0.4±0.1	0.3±0.1	0.4±0.1	0.65
	(–) aVR	0.4±0.1	0.4±0.1	0.4±0.1	0.82
	II	0.5±0.1	0.6±0.1	0.4±0.1	0.21
	aVF	0.4±0.1	0.3±0.1	0.4±0.1	0.53
	III	0.5±0.1	0.6±0.1	0.4±0.1	0.35
	V1	1.6±1.2	1.7±0.3	1.6±0.1	0.52
	V2	2.9±1.7	2.9±1.9	2.9±1.7	0.93
	V3	3.6±2.4	3.8±2.8	3.5±2.2	0.62
	V4	2.6±2.2	2.8±2.3	2.6±2.2	0.64
	V5	1.5±0.1	1.5±0.2	1.5±0.2	0.91
	V6	0.8±0.1	0.8±0.2	0.76±0.12	0.82
Prominent Q wave in other leads, n (%)	aVL	29 (24)	8 (25)	21 (23.6)	0.83
	I	18 (14.8)	4 (12.5)	14 (15.7)	0.62
	II	11 (9.1)	5 (15.6)	6 (6.7)	0.14
	aVF	9 (7.4)	3 (9.4)	6 (6.7)	0.63
	III	12 (9.9)	3 (9.4)	9 (10.1)	0.71
	V1	84 (69.4)	22 (68.8)	62 (69.7)	0.92
	V2	95 (78.6)	26 (81.3)	69 (77.5)	0.64
	V3	60 (49.6)	17 (53.1)	43 (48.3)	0.62
	V4	46 (38)	14 (43.8)	32 (36)	0.65
	V5	21 (17.4)	5 (15.6)	16 (18)	0.72
	V6	6 (5)	3 (9.4)	3 (3.4)	0.12
Number of leads with STE on admission ECG		5 (3–10)	6 (3–9)	5 (3–10)	0.32
Number of leads with STD on admission ECG		0 (0–6)	0 (0–4)	0 (0–6)	0.60
Number of leads with significant STE on admission		4 (0–9)	4 (0–9)	4 (1–9)	0.38
Number of leads with ST deviation on admission		7 (3–11)	7 (3–11)	7 (3–11)	0.38
Number of leads with STE on Pre-discharge ECG		4 (0–9)	4 (0–8)	4 (0–9)	0.47
Number of leads with significant STE on Pre-discharge ECG		2 (0–8)	2 (0–8)	2 (0–8)	0.59

Group A: Patients with Q wave in lead (–)aVR and Group B: Patients without Q wave in lead (–)aVR. Categorical data are shown as n (%), Normal distribution was rejected for the number of the lead with STE (ST segment elevation); STD (ST segment depression) and ST segment deviation, therefore these date were presented as median (min-max). The remaining of the numerical values were presented as mean±standard deviation and tested by 2 independent samples t-tests and p-values <0.05 were considered significant.

Table 3. Comparison of echocardiographic and angiographic data

	Group A (n=32)			Group B (n=89)			*p*
	n	%	Mean±SD	n	%	Mean±SD	
Regional wall motion abnormality	28	87.5		83	96.5		0.08
Anteroseptal wall involvement	25	78.1		66	76.7		0.83
Anteroseptal wall involvement	17	53.1		46	53.5		0.92
Apical involvement	13	40.6		24	27.9		0.14
Inferior wall involvement	2	6.3		6	7		0.82
Posterobasal involvement	3	9.4		1	1.2		0.04*
Pleural effusion	1	3.1		3	3.4		0.93
Left ventricular ejection fraction (%)			38.3±10.3			40.3±8.7	0.21
Mitral regurgitation more than mild	7	21.9		16	17.9		0.72
Left ventricular end diastolic diameter (cm)			4.7±0.9			4.6±0.7	0.55
Percutaneous coronary intervention	12	46.2		48	66.7		0.07
Rescue percutaneous coronary intervention	1	3.1		1	1.1		0.44
Multi-vessel disease	20	76.9		38	52.8		0.03*
Proximal left anterior descending lesion	17	65.4		57	79.2		0.12
Left main coronary artery lesion	2	7.7		4	5.6		0.61
Response to thrombolytic therapy							
<30%	8	25		12	13.5		
30%–70%	8	25		28	31.5		0.55
>70%	2	6.3		7	7.9		

Group A: Patients with Q wave in lead (–)aVR and Group B: Patients without Q wave in lead (–)aVR. P-value <0.05 is considered significant.

Sensitivity and specificity of the Q wave in (–)aVR for predicting multi-vessel disease in coronary angiography was 34% and 85% respectively. It has also a sensitivity of 17% and specificity of 73% for predicting in-hospital mortality. In addition, the sensitivity and specificity of the Q wave in (–)aVR for 2-year mortality was 60% and 76% respectively (Table 5).

DISCUSSION

We did not find any association between prominent Q waves in lead (–)aVR and in-hospital adverse outcomes or mortality in patients with anterior STEMI. However, multi-vessel involvement was more common in patients who evolved Q waves in lead (–)aVR. In addition, posterobasal WMA of the left ventricle (LV) and presence of significant ST-segment elevation in lead V6 on admission ECG were associated with the presence of prominent Q waves in lead (–)aVR.

Recent studies have emphasized the importance of ST-segment deviation in lead aVR or (–)aVR in patients with anterior or inferior STEMI.[9,12,15] However, the significance of a Q wave in this lead remains poorly understood. In the only existing study, by Kotoku et al., presence of a Q wave in lead (–)aVR was proposed as an indicator for involvement of the apical region of the LV and as a sign for having long LAD wrapping the apex, although its presence had a low sensitivity for detecting apical infarctions.[13] In contrast, our results revealed no significant differences in the incidence of apical wall involvement in patients with and those without Q wave in (–)aVR. Nevertheless, posterobasal wall involvement may imply the presence of a lesion in either long LAD wrapping the apex or a lesion in LAD, which supplies collateral flow to an obstructed, left circumflex or right coronary artery.[16–18] Although posterobasal wall involvement occurred in a higher percentage of patients with a prominent Q wave in (–)aVR lead, the overall frequency of this

Table 4. Comparison of laboratory data and adverse clinical outcomes

	Group A (n=32)			Group B (n=89)			*p*
	n	%	Mean±SD	n	%	Mean±SD	
Pulmonary edema	5	15.6		9	10.1		0.42
Cardiogenic shock	1	3.1		2	2.2		0.74
Peak cTnI (ng/mL)			9.2±1.9			11.8±1.3	0.24
Peak CK-MB (ng/mL)			159±120			172±148	0.63
Reinfarction	3	9.4		8	9.0		0.94
Ventricular dysrhythmias	1	3.1		1	1.1		0.45
Killip class							
1	21	65.6		65	73.0		
2	6	18.8		15	16.9		0.82
3	4	12.5		7	7.9		
4	1	3.1		2	2.2		
Hospital length of stay (Days)			7.3±3.1			7.1±2.8	0.73
Coronary care unit							
Length of stay (Days)			4.2±1.8			4.3±2.1	0.75
In-hospital mortality	1	3.1		5	5.6		0.99
Mortality during follow-up	3	9.7		2	2.4		0.21
Overall mortality	4	15.4		7	9.3		0.33

Group A: Patients with Q wave in lead (–)aVR and Group B: Patients without Q wave in lead (–)aVR. P-value <0.05 is considered significant.

Table 5. Sensitivity, specificity, positive predictive value (PPV) and negative predictive value (NPV) of a prominent Q wave in lead (–)aVR

	Sensitivity	Specificity	Positive predictive value	Negative predictive value
	%	%	%	%
Multi-vessel disease	34	85	77	47
Proximal LAD lesion	23	63	65	21
Regional wall motion abnormality	25	43	88	3
In-hospital mortality	17	73	3	94
Mortality during follow-up	60	76	12	97

LAD: Left anterior descending.

anatomic location (4 patients) was too low to strongly support our hypothesis. In line with this, multi-vessel disease was more prevalent in patients who evolved Q waves in (–)aVR. Based on these facts, both anatomical variations may explain development of Q waves in (–)aVR although the exact cause of this finding requires further investigations.

In the present study, presence of Q waves in (–)aVR was not associated with higher occurrence of adverse outcomes. Both groups had a similar LVEF and the occurrence of pulmonary edema, ventricular dysrhythmias and severity of heart failure were comparable in both groups. Considering that the level of peak cardiac enzymes, which implies a poor prognosis in myocardial infarction,[19] was also similar in two groups, the presence of prominent Q waves in (-)aVR

may simply reflect extensive anatomical involvement rather than the intensity of infarction. Contrary to our findings, in the study by Kotoku et al. presence of a Q wave was associated with a slightly lower LVEF, but the difference in CK-MB levels was not statistically significant.[13] Kosuge et al. examined the association of ST-segment deviation in aVR with the risk of clinical outcomes in anterolateral STEMI, and suggested the presence of ST depression in lead aVR as a useful predictor for larger infarct size and LV dysfunction.[15] In another study, Goto et al. investigated the importance of ST-segment deviation in lead aVR in anterior STEMI and found no difference in LVEF in patients with and those without ST-segment deviation in aVR.[20] It should be noted that these two studies only explored the level of ST-segment in aVR, which may not be associated with presence of a Q wave.

The association between location of the culprit lesion and changes in lead aVR is a matter of controversy.[15,21–23] According to our results, the presence of a Q wave was not associated with either location of the LAD lesion or presence of a stenotic lesion in the LMCA. However, patients with multi-vessel disease in angiographic evaluation had an increased incidence of Q wave in (–)aVR. In contrast, Kotoku et al. found no difference in the prevalence of multi-vessel disease in patients with and those without Q in (–)aVR. In their study, having a long LAD was associated with Q waves in (–)aVR, but site of the LAD lesion was not different between the two groups.[13] In one study on patients with STEMI, the presence of ST-segment elevation in lead aVR was higher in men with multivessel disease.[21] Some studies investigating the ST-segment deviation in lead aVR, suggested the changes in this lead as a predictor for the site of LAD occlusion in patients with anterior STEMI.[15,20,23] In the study by Goto et al., occlusion of the proximal LAD was lower in patients with ST depression than in those with either elevated or normal ST-segment. Moreover, having a long LAD was related to ST-segment depression in lead aVR.[20] It is worth mentioning that another study on patients with anterolateral STEMI found no association between site of the culprit lesion with either ST elevation or ST depression in aVR.[15]

In the present study, patients with significant ST elevation in lead V6 on admission ECG had a higher incidence of prominent Q wave in lead (–)aVR on pre-discharge ECG. The elevation of ST-segment in lead V6 in anterior STEMI may indicate involvement of the proximal LAD in a long LAD, distal diagonal branch occlusion or involvement of the left circumflex artery.[18] Regarding the higher frequency of multi-vessel disease in patients with Q-wave, it is plausible that ST-segment elevation in V6 was concurrently higher in this group.

Both short- and mid-term mortality rates for patients who developed a Q wave in lead (–)aVR, was not significantly different from those who did not. This is the first article investigating the role of predischarge Q wave in (–)aVR in anterior STEMI. ST segment elevation in aVR on admission was previously described as a predictor of mortality in patients with anterior STEMI and with Non-STEMI.[9,10] However, in our study the rate and level of ST-segment elevation and depression in lead (–)aVR as well as the number of other leads with ST-segment elevation on admission ECG was not statistically different in patients who evolved a Q wave on pre-discharge ECG. Treatment response and adverse clinical outcomes during hospital stay were also similar in both groups. This may suggest that presence of a Q wave in lead (–)aVR in anterior STEMI cannot prognosticate either short- or mid-term mortality. In conclusion, developing Q wave in lead (–)aVR after anterior STEMI is not a good indicator for extent of myocardial infarction or adverse outcomes. However, it may provide valuable information about the presence of concurrent occlusion in coronary arteries other than LAD. It should be noted that this study was performed on a relatively small sample of patients with first anterior , and although it was sufficient to detect the main primary endpoint, further subgroup analysis was not possible without affecting its statistical power. Further investigations with larger sample populations and with consideration of normal coronary anatomy variants in different patients are essential to ascertain different causes of Q wave development in (–)aVR and to predict future complications in different subgroups of patients.

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