ORIGINAL ARTICLES

Correlation between ST Elevation and Q Waves on the PredischARGE Electrocardiogram and the Extent and Location of MIBI Perfusion Defects in Anterior Myocardial Infarction

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Background: The common electrocardiographic subclassification of anterior acute myocardial infarction (AMI) is not reliable in presenting the exact location of the infarct. We investigated the relationship between predischARGE electrocardiographic patterns and the extent and location of perfusion defects in 55 patients with first anterior AMI.

Methods: PredischARGE electrocardiogram was examined for residual ST elevations and Q waves which were correlated with technetium-99m-sestamibi function and perfusion scans.

Results: Patients with ST elevations in V2–V4 and Q waves in leads V3–V5 had worse global perfusion scores. Perfusion defects in the apex inferior segment were significantly less frequent in patients with Q waves in leads I and aVL (11% vs 54%, $P = 0.027$; and 22% vs 60%, $P = 0.011$, respectively). Patients with Q wave in aVF had more frequently involvement of the apex inferior segment (80% vs 40%; $P = 0.035$). Patients with Q wave in lead II had significantly more frequent perfusion defects in the inferior wall. ST elevation in V3 and V4 was associated with perfusion abnormalities of the infero-septal segments. ST elevation in V5 and V6 and Q wave in V5 were associated with regional perfusion defects in apical inferior segment (73% vs 30%, $P = 0.002$), extending into the mid inferior segment (55% vs 18%, $P = 0.005$ for Q wave in V5). Q wave in lead aVL is associated with less apical and inferior involvement. Q waves in leads II and aVF are a sign of inferior extension of the infarction.

Conclusions: Residual ST elevation in leads V3 and V4 are more frequently associated with involvement of the apical-inferoseptal segment rather than the anterior wall. Residual ST elevation and Q waves in V5 are related to a more inferior rather than a lateral involvement.

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ECG is commonly used as an early noninvasive tool for diagnosing acute myocardial infarction because of its widespread accessibility, low cost and simplicity of operation. The classification of anterior wall myocardial infarction (AMI) is based historically on autopsy findings in the subacute and chronic phases of infarction and their corre-

lation with electrocardiographic (ECG) patterns.^{1–4} Acute AMI is commonly classified as septal when Q waves are present in leads V1 and V2; anterior, when Q waves are present in leads V3 and V4; antero-septal, if Q waves are in V1–V4; lateral, when Q waves are in leads V5, V6, I and aVL; and anterolateral, when Q waves appear in leads V3–V6,

and aVL.⁵ Although most AMIs involve the left ventricular apical regions, ECG identification of apical infarction is controversial and has several different definitions.^{6,7} Several studies have shown that although predicting the general location of the infarction, the ECG is not reliable in providing detailed information concerning the exact size and localization of the infarction.⁷⁻⁹ Nevertheless, the traditional ECG classification of anterior AMI is widely used in clinical practice.

Numerous studies assessed the ability to estimate myocardial infarct size and extension by the ECG in the chronic phase of myocardial infarction, using mainly the QRS scoring method.¹⁰⁻¹² However, recently we have shown that when residual ST elevation is present, as is commonly found on the predischARGE ECG, the correlation between the sestamibi perfusion defect size and the QRS score is weak.¹³ Because the duration of hospitalization for AMI has been shortened considerably during the last 10 years, many patients with AMI still have ST elevation upon discharge. Studies concerning predicting the exact location of acute AMI by the ECG are comparatively few, and concentrated mostly on correlations between the various ECG patterns and coronary angiographic morphology.¹⁴⁻²⁶ Fewer reports studied the correlations between various ECG subtypes of AMI and the location of infarction, as assessed by echocardiography^{27,28} and myocardial scintigraphy perfusion defects.^{24,29,30} ST segment elevation and Q waves in the various leads are assumed to have the same significance in localizing the myocardial infarction although Q waves are recorded at the beginning of systole and ST toward the end of systole, and the various leads may not face the same myocardial segments during the beginning and end of systole.

The aim of the present study was to investigate the relationship between ST elevation and the presence of Q waves on each lead of the predischARGE ECG and the location of segmental perfusion defects, global severity perfusion scores, and extension scores, as detected by predischARGE Tc-99m-sestamibi myocardial perfusion scan. In particular, we wanted to assess whether (1) ST elevation and Q waves in leads I and aVL are associated with involvement of the lateral segments; (2) ST elevation and Q waves in the inferior leads are associated with inferior or apical involvement; (3) ST elevation and Q waves in the "anteroseptal" leads are associated with more septal involvement; and (4) ST elevation and Q waves in leads V5-

V6 are associated with more apical or distal lateral involvement.

METHODS

We prospectively included 55 consecutive patients admitted to the cardiac intensive care unit with first ST elevation evolving into Q wave acute AMI between the years 1997-2000 who underwent Tc-99m-sestamibi resting myocardial perfusion scan for 4-12 days (mean $\pm$ standard deviation: 7 ± 3 days) after their admission, before discharge from the cardiac intensive care unit.

The diagnostic criteria of Q wave acute AMI were chest pain of more than 30-minute duration, ST-segment elevation ≥ 0.1 mV in at least two consecutive precordial leads (V1-V4) on admission (≥ 0.2 mV above the TP segment for leads V2 and V3, and ≥ 0.1 mV from the TP segments for leads V1 and V4), development of pathological Q waves in ≥ 2 adjacent precordial leads and elevation of serum level of creatinine kinase. Patients with ECG evidence or history of old myocardial infarction, non-Q-wave AMI, intraventricular conduction defects, left ventricular hypertrophy, arrhythmias or cardiomyopathy were excluded. Clinical data were collected from the patients files concerning age, sex, history of smoking, diabetes mellitus, hypertension, total cholesterol level, peak serum creatinine-kinase, and reperfusion therapy.

Electrocardiography

ECG tracings recorded at the day of the Tc-99m-sestamibi myocardial perfusion scan were analyzed, without awareness of the clinical and imaging data. ST segment was measured manually 60 ms after J point. ST elevation was defined as ≥ 0.1 mV. Abnormal Q waves were defined as ≥ 30 ms in leads I, aVL, V5, V6, and the inferior leads; ≥ 20 ms in lead V4; and any Q wave in leads V1-V3.

The number of leads with residual ST elevation and Q wave were calculated as well as the sum of ST elevation in those leads.

Tc-99m-Sestamibi Myocardial Perfusion Imaging

No patient had severe decompensation during the 24 hours preceding the study.

First pass imaging: Tc-99m-sestamibi at a dose of 25 mCi was infused. First pass acquisition was performed for 30 seconds using one-head gamma

camera (Apex-SPX-4HR, Elscint Inc, Haifa, Israel). Acquisition and processing of left ventricular ejection fraction and volumes were performed as described previously.³¹

Single photon emission computed tomography (SPECT): One hour after the administration of radiopharmaceutical, SPECT images were acquired. Quantitative assessment of segmental Tc-99m-sestamibi activity was analyzed by circumferential profile.^{32,33} The left ventricle was divided into 20 segments consisting of three sets of short axis slices in the apical, mid, and basal portions, each divided into six segments: anterior, antero-septal, infero-septal, inferior, infero-lateral, and antero-lateral. Two additional apical segments: antero-apical and infero-apical were taken from vertical long axis (Fig. 1). The relative activity in each segment was calculated and categorized with scoring from 0 to 3: score 0, normal, with uptake >80%; score 1 or mild defect, with uptake of 65–80%; score 2 or moderate defect, with uptake of 50%–65%; score 3 or severe defect, with uptake of <50%. Perfusion analysis was translated into (1) segmental perfusion defects (severity scores 1–3); (2) global severity per-

fusion score, which is the sum of severity score in all 20 segments; (3) global extension score, which is the sum of the segments with any perfusion defect. The digitized images were evaluated by two experienced readers who were blinded to the clinical outcome and ECG findings of the patients.

Statistics

Means $\pm$ SD were calculated for continuous variables and absolute and relative frequencies were measured for discrete variables. Differences between groups were analyzed with the independent two-tailed Student's *t*-test for continuous variables and by chi-square test for discrete variables. In cases of a small number of patients per category, Fisher's exact test was used. Pearson's correlation coefficient and significance was calculated between continuous variables. P-value ≤ 0.05 was considered statistically significant.

RESULTS

Demographic and clinical characteristics of the 55 patients are shown in table 1. The percentage

Figure 1. Schematic presentation of the 20 myocardial segments. The left ventricle is divided into three short axis view slices (apical, mid, and basal). Each slice is divided into 6 segments: anterior, antero-septal, infero-septal, inferior, infero-lateral, and antero-lateral. Additional two apical segments: anteroapical and inferoapical are taken from vertical long axis view.

Table 1. Characteristics of the Patients

Total number of patients	55
Mean age (years)	58.6 ± 13.8
Men	41 (74.5%)
Women	14 (25.5%)
Smokers	24 (43.6%)
Diabetes mellitus	11 (20%)
Hypertension	22 (40%)
Hyperlipidemia	35 (60.3%)
Mean maximal creatinine kinase (IU/L)	1979 ± 1732

of patients with ST elevation (≥ 0.1 mV and ≥ 0.2 mV) in each lead on the predischage ECG is presented in figure 2a. Most patients had ST elevation in leads V2 (51 patients, 93%) and lead V3 (50 patients, 91%). ST elevation was noted in leads V1 and V4 in only 22 patients (40%) and 37 patients (67%), respectively. The percentage of patients with predischage Q wave in each lead is presented in Figure 2b. Most patients had Q wave in lead V2 (49 patients, 89%) and V1 (35 patients, 64%). Twenty-two patients (40%) had Q waves in lead V5 and 10 patients (18%) had Q waves in lead V6. Nine patients (16%) had Q waves in lead I and 18 patients (32.7%) had Q waves in lead I aVL. Q wave was found in 4 (7%) patients in lead II, 10 (18%) patients in lead III, and 10 (18%) patients in lead aVF.

The incidence of the perfusion defects in each of the 20 segments is presented in figure 3. Perfusion defects were most prevalent in the antero-apical and infero-apical segments (long axis) as well as in the apex anterior and antero-septal segments. Only 12 patients (22%) had perfusion defects in the basal anterior segment. In contrast, perfusion defects in the apex anterior and mid anterior segments were found in 54 patients (98%) and 46 patients (84%), respectively.

No significant correlations were found between the number of leads with ST segment elevation, the number of leads with Q wave or the sum of ST elevation in the various leads on the predischage ECG and the global severity perfusion score, extension score, peak creatinine kinase, left ventricular ejection fraction, or end-systolic or end-diastolic volumes.

ST Segment Elevation and Q Waves in the Anterolateral Leads (I and aVL)

There was no significant difference in global severity perfusion score or extension score between

patients with and without ST elevation or Q waves in leads I and aVL. There were no differences in the distribution of segmental perfusion defects between patients with and without ST elevation in these leads (Table 2). Patients with Q wave in leads I and aVL had significantly less frequently perfusion defects in the apex inferior segment (Table 3, Fig 4). In addition, those with Q wave in lead aVL had perfusion defects less frequently in the apex antero-septal segment. Forty-four patients had a perfusion defect in the apex antero-septal segment. Thirteen of them (29%) had Q wave in lead aVL. In contrast, 7 patients did not have a perfusion defect in the apex antero-septal segment, 5 of them (71%) had Q waves in aVL. None of the 18 patients with Q wave in aVL had Q waves in the inferior leads.

ST Segment Elevation and Q Waves in the Inferior Leads (II, III and aVF)

There was no significant difference in global severity perfusion score or extension score between patients with and without ST elevation or Q waves in leads II, III and aVF on the predischage ECG. There was no difference in the distribution of segmental perfusion defect between patients with and without ST elevation in the inferior leads (Table 2). However, patients with Q wave in lead II had significantly more perfusion defects in apex inferior, mid inferior, and basal inferior segments than patients without Q wave in this lead (Table 3). Patients with Q wave in lead aVF had significantly more perfusion defects in the apex-inferior segment (Table 3, Fig. 4). There were no significant differences in the distribution of perfusion defects between patients with or without Q wave in lead III.

ST Segment Elevation and Q Waves in the Precordial Leads (V1–V6)

Patients with ST elevation ≥ 0.1 mV in leads V2, V3, and V4 had significantly worse global severity perfusion score than patients without ST elevation in those leads (Lead V2: 21.5 ± 8.9 vs 7.2 ± 3.9 , $P = 0.003$. Lead V3: 21.7 ± 8.8 vs 8 ± 4.1 , $P = 0.001$. Lead V4: 22.5 ± 8.5 vs 16.2 ± 9.8 , $P = 0.017$). Patients with ≥ 0.1 mV ST elevation in leads V3 and V4 had significantly worse extension score (Lead V3: 9.7 ± 3.2 vs 4.6 ± 2.1 , $P = 0.001$. Lead V4: 9.9 ± 3.2 vs 7.9 ± 3.6 , $P = 0.048$). Patients with ≥ 0.1 mV ST elevation in leads V2 and V4 had in addition significantly lower ejection fraction (Lead

Figure 2. The percentage of patients with (a) ST-elevation (≥ 0.1 mV and ≥ 0.2 mV) (b) Q-waves in each lead on the predischarge electrocardiogram.

V2: $37.3 \pm 9.1\%$ vs $47.3 \pm 10.7\%$, $P = 0.042$. Lead V4: $36.1 \pm 10.0\%$ vs $41.8 \pm 7.1\%$, $P = 0.038$.

Patients with predischarge Q wave in leads V3, V4, and V5 had significantly worse global severity perfusion score (Lead V3: 22.3 ± 8.7 vs 16.5 ± 9.8 , $P = 0.033$. Lead V4: 23.0 ± 9.4 vs 18.0 ± 8.8 , $P = 0.045$. Lead V5: 23.5 ± 8.9 vs 18.5 ± 9.3 , $P = 0.053$)(Fig. 4). Patients with predischarge Q wave

in leads V3 and V4 had significantly worse extension score (Lead V3: 10.0 ± 3.4 vs 7.6 ± 3.2 , $P = 0.015$. Lead V4: 10.2 ± 3.7 vs 8.4 ± 3.1 , $P = 0.056$). There was no significant correlation between the presence or absence of Q waves in leads V1, V2, or V6 and the size of either the global severity perfusion score or extension score. Patients with Q wave in leads V3, V4, and V5 had significantly lower

Figure 3. The incidence of the regional perfusion defects in each of the 20 segments.

ejection fraction (Lead V3: $36.3 \pm 8.9\%$ vs $41.8 \pm 9.8\%$, $P = 0.044$. Lead V4: $35.3 \pm 8.7\%$ vs $40.7 \pm 9.5\%$, $P = 0.033$. Lead V5: $34.6 \pm 8.7\%$ vs $40.2 \pm 9.4\%$, $P = 0.032$). There was no significant correlation between the presence or absence of Q waves in leads V1, V2 or V6 and left ventricular ejection fraction.

There were no significant differences in the distribution of segmental perfusion defects between patients with and without ST elevation in leads V1 and V2. Patients with ≥ 0.2 mV ST elevation in lead V3 tended to have perfusion defects more frequently in the apex infero-septal segment than patients with < 0.2 mV ST elevation in this lead (Table 2). Patients with ST elevation in lead V4 had significantly more perfusion defects in the apex in-

fero-septal, apex inferior and mid infero-septal segments (Table 2, Fig. 4).

There was no difference in the distribution of segmental perfusion defects between patients with and without ≥ 0.1 mV ST elevation in leads V5 or V6. However, ST elevation ≥ 0.2 mV in those leads was associated with mid and apexinferior segments [Lead V5: 6/8 patients (75%) vs 12/47 patients (25%), $P = 0.011$. Lead V6: 4/4 patients (100%) vs 22/51 patients (43%), $P = 0.044$]. Patients with ST elevation ≥ 0.2 mV in lead V6 had in addition significantly fewer perfusion defects in the basal antero-septal segment [0/4 patients (0%) vs 31/51 patients (61%), $P = 0.031$].

Patients with Q wave in lead V1 had significantly more perfusion defects in the basal-infero-septal

Table 2. Correlation Between ST-Elevation and Segmental Perfusion Defect

Lead	Perfusion Segment	ST elevation (Present vs absent)	Significance (P)
I, aVL (≥ 0.1 mV)	—	—	NS
II, III, aVF (≥ 0.1 mV)	—	—	NS
V1 (≥ 0.1 mV)	—	—	NS
V2 (ST ≥ 0.2 mV)	—	—	NS
V3 (ST ≥ 0.2 mV)	Apex Inferoseptal	25/35 (71%) vs 9/20 (45%)	0.052
V4	Apex Inferoseptal	27/37 (73%) vs 7/18 (39%)	0.015
	Apex Inferior	21/37 (57%) vs 5/18 (28%)	0.043
	Mid Inferoseptal	22/37 (60%) vs 5/18 (28%)	0.027
V5, V6 (≥ 0.1 mV)	—	—	NS

(Table 3). There were no differences in the incidence of segmental perfusion defects in all 20 segments between patients with and without Q wave in leads V2 and V6. Patients with Q wave in lead V3 had significantly more perfusion defects in the apex-inferior and the basal-antero-septal segments. Patients with Q wave in lead V4 had significantly more perfusion defects in the apex-inferior and apex, mid, and basal segments of the inferolateral wall (Table 3). Patients with Q wave in lead V5 had significantly more perfusion defects in apex-inferior, mid-inferoseptal, and mid-inferior segments (Table 3).

DISCUSSION

The ECG localization and definitions of AMI are historically based on correlations between ECG patterns in the subacute and chronic stages of infarction and autopsy findings.¹⁻⁴ Many studies have demonstrated the unreliability in separating the subdivisions of anterior wall infarcts by ECG patterns.⁷⁻⁹ It is usually assumed that ST segment elevation has a similar significance in localizing the infarction as the distribution of Q waves in the subacute and chronic phases of infarction. In the present study we assessed the correlation between

Table 3. Correlation Between Q Waves in Each Lead and Segmental Perfusion Defect

Lead	Perfusion Segment	Q-wave (Present vs Absent)	Significance (P)
I	Apex Inferior	1/9 (11%) vs 25/46 (54%)	0.027
aVL	Apex Anteroseptal	13/18 (72%) vs 35/37 (95%)	0.032
	Apex Inferior	4/18 (22%) vs 22/37 (60%)	0.011
II	Apex Inferior	4/4 (100%) vs 22/51 (43%)	0.044
	Mid Inferior	4/4 (100%) vs 14/51 (27%)	0.009
	Basal Inferior	2/4 (50%) vs 1/51 (2%)	0.012
III	—	—	NS
aVF	Apex Inferior	8/10 (80%) vs 18/45 (40%)	0.035
V1	Basal Inferoseptal	20/35 (57%) vs 5/20 (25%)	0.021
V2	—	—	NS
V3	Apex Inferior	22/38 (58%) vs 4/17 (24%)	0.018
	Basal Anteroseptal	25/38 (66%) vs 6/17 (35%)	0.035
V4	Apex Inferior	18/27 (67%) vs 8/28 (29%)	0.005
	Apex Inferolateral	4/27 (15%) vs 0/28 (0%)	0.051
	Mid Inferolateral	4/27 (15%) vs 0/28 (0%)	0.051
	Basal Inferolateral	4/27 (15%) vs 0/28 (0%)	0.051
V5	Apex Inferior	16/22 (73%) vs 10/33 (30%)	0.002
	Mid Inferoseptal	15/22 (68%) vs 12/33 (36%)	0.021
	Mid Inferior	12/22 (54%) vs 6/33 (18%)	0.005
V6	—	—	NS

Figure 4. (a). ECG tracing showing anterior infarction with Q-wave and residual ST-elevation in lead aVL. There is QS-wave in lead V1, but there are no Q-waves and the ST is isoelectric in leads V2–V6. The perfusion images (3 short axis slices, and vertical long axis) demonstrate small infarct with global severity score of 14. The infarction involves the apex-anterior, mid-anterior and mid-antrolateral segments on the short axis views and the anteroapical segment in the long axis view. The perfusion in the apex-inferior, apex-inferoseptal, and apex-antroseptal segments on the short axis view, and the inferoapical segment on the long axis are intact. (b). ECG tracing showing anterior infarction with Q-waves in leads I, aVL, V1–V3, and ST-elevation in leads aVL, V2–V4. The perfusion images (as above) demonstrate moderate infarct size with global severity score of 24. The infarction involves the apex-, mid- and basal-anterior segments, as well as apex-antroseptal segment on the short axis views, and the anteroapical and inferoapical segments on the long axis view. The apex-inferoseptal and apex-inferior segments are spared. (c). ECG tracing showing anterior infarction with Q-waves in leads III, aVF, V1–V3 and ST-elevation in leads V1–V4. The perfusion images (as above) demonstrates large infarct with global severity score of 31. The infarct involves the anterior (basal, mid and apical) and antroseptal (basal, mid and apical) segments, as well as the apex-inferior segment on the short axis views. There is a partial perfusion defect in the infero-septal segments. There are perfusion defects also in the anteroapical and inferoapical segments on the long axis view.

residual ST elevation and Q waves in the various leads of the predischarge ECGs and the patterns of global and regional perfusion defects measured by myocardial perfusion imaging with Tc-99m-sestamibi in patients with first acute AMI.

Number of Leads and Sum of ST Segment Elevation

In the present study we did not find any correlation between the number of leads with ST

elevation or the sum of ST elevation in the pre-discharge ECG and global severity perfusion score, extension score, ejection fraction, or peak serum creatinine-kinase levels.

ST Segment Elevation and Q Wave in Leads I and aVL

ST segment elevation and Q waves in leads I and aVL are considered to represent high antero-lateral involvement.^{5,6} Previous studies correlating ECG patterns and coronary angiography reported that ST segment elevation in leads I and especially aVL on admission of acute AMI indicates occlusion of the left anterior descending (LAD) coronary artery proximal to the first diagonal branch.^{14–18} However, the sensitivity of this finding for proximal LAD occlusion is relatively low, because in cases where the occluded LAD wraps the cardiac apex and the inferior segments are involved, the electrical vector of the inferior wall and the high lateral wall oppose each other and no ST deviation is detected in either the lateral leads (I and aVL) or the inferior leads (II, III, and aVF).¹⁹ A recent report correlating admission ECG with echocardiographic assessment of regional wall motion, found that ST elevation in lead aVL is associated with the mid-lateral regional wall motion abnormality.²⁷ In the present study, patients with Q wave in leads I and aVL on the pre-discharge ECG had significantly less perfusion defects in the apex inferior and apex antero-septal segments, a finding that supports involvement of a short LAD that does not extend beyond the cardiac apex and supplies the inferior segments.¹⁹ However, there was no significant difference in the prevalence of regional perfusion defects in the anterolateral segment between patients with and without ST elevation or Q wave in leads I and aVL. Thus, ST elevation in leads I and aVL during the acute stage of AMI and residual ST elevation or Q waves on the pre-discharge ECG may not have the same significance concerning the exact extension of the infarction. We did not find differences in global severity perfusion score or extension score between patients with and without ST elevation or Q waves in leads I and aVL, contrary to what we would have expected if ST elevation in those leads indicates occlusion of proximal LAD artery with more extensive involvement of the infarction. However, others also found that ST elevation in I and aVL is not associated with worse global left ventricular function,²⁷ probably because ST ele-

vation in these leads is found only when a proximal occlusion occurs in a short LAD.¹⁹

ST Segment Elevation and Q Wave in the Inferior Leads (II, III and aVF)

Inferior ST elevation during acute AMI is common in patients with distal occlusion of a long LAD coronary artery that wraps the cardiac apex, and has a relatively good in-hospital prognosis.^{19,20} We did not find a difference in global severity perfusion score or extension score between patients with and without residual ST elevation in the inferior leads, supporting Porter et al.²⁷ who did not find differences in echocardiographic global wall motion score between patients with and without ST elevation on the admission ECG in these leads. In contrast, Sasaki et al.¹⁹ reported that patients without ST elevation on admission in the inferior leads had worse echocardiographic global wall motion score than patients with ST elevation in the inferior leads.

There was no difference in the percentage of patients with perfusion defects in the inferior and posterior segments between patients with and without residual inferior leads ST elevation. We found significantly more perfusion defects in the apex inferior segment in patients with Q wave in leads II and aVF on the pre-discharge ECG. Moreover, patients with Q wave in lead II had significantly more involvement of the mid- and basal inferior segments. Our results are compatible with the angiographic findings of more apical and inferior regional involvement in patients with ST elevation in the inferior leads after AMI, probably due to occlusion of a wrapping LAD.^{19,20}

ST Elevation and Q Wave in the Precordial Leads

Leads V1 and V2

It is commonly believed that Q waves in leads V1–V2 represent involvement of the interventricular septum. The significance of ST elevation in leads V1 and V2 was investigated in several studies, with conflicting results. Usually the magnitude of ST elevation in lead V1 is less than in V2 and fewer patients with AMI have ST elevation in V1 than in V2.^{25,26} It has been suggested that patients with AMI without ST elevation in lead V1 have a large conal branch of the right coronary artery that supplies the interventricular septum, whereas patients

with significant ST elevation in lead V1 have a small conal branch.^{25,26} Porter et al. found that ST elevation in lead V1, early after admission of acute AMI, is associated with a high incidence of regional wall motion abnormality in the basal septal, antero-septal, and anterior regions, whereas ST elevation in lead V2 is associated with more extensive apical-inferior regional dysfunction, emphasizing the difference between leads V1 and V2, and the importance of lead V1 as a sign of basal-anterior and septal regional involvement in the acute phase of acute AMI.²⁷ Shalev et al. disputed the conventional definition of antero-septal acute myocardial infarction. Correlating ECG, echocardiographic, and cardiac catheterization findings in 80 patients they found that 92% of the patients with ST elevation in leads V1–V3 had an antero-apical infarct and a normal septum. They concluded that the ECG pattern traditionally termed antero-septal infarction should be called an antero-apical infarction typically resulting from the occlusion of the mid-to-distal LAD.²⁸ Engelen et al. strengthened this finding by showing that ST elevations in leads V1–V3 were associated with LAD occlusion distal to the first and second septal perforator branches (in 57% and 23% of their patients, respectively). However, ST elevation > 2.5 mm in lead V1 was the only parameter that strongly predicted LAD occlusion proximal to the first septal branch.¹⁷

In the present study, there were no differences in the distribution of regional perfusion defects between patients with and without ST elevation in lead V1. However, patients with Q wave in lead V1 had perfusion defects more frequently in the basal inferoseptal segment.

Leads V3 and V4

ST elevation and Q waves in leads V3 and V4 are considered to represent "pure" anterior infarction when they appear alone, antero-septal as they accompany changes in leads V1 and V2, or antero-lateral (or lateral apical) when ischemic changes in lead V4 occur together with alterations in leads V5 and V6.^{5,6}

We found that patients with ≥ 0.2 mV ST elevation in lead V3 had a larger infarct size, related to more frequently extension of the AMI toward the apical portion of the inferoseptal wall. Similarly, patients with ≥ 0.1 mV ST elevation in lead V4 had larger infarct size, associated with more extension toward the apex-inferior, apex inferosep-

tal and mid-inferoseptal segments. Patients with Q wave in lead V3 had significantly more perfusion defects in apex-inferior and basal-antero-septal segments, while patients with Q wave in lead V4 had regional perfusion defects more frequently in the apex-inferior and the apex/mid/basal inferolateral segments. Our results show that residual ST elevation and pathological Q waves on the predischARGE ECG in leads V3 and V4 are not related to "anterior" location of the infarction, but to apical and inferior involvement.

Leads V5 and V6

ST elevation and Q waves in leads V5 and V6 are considered to indicate more extensive lateral or apical involvement of acute AMI.^{5,6} Engelen and coauthors found that in acute AMI, abnormal Q waves in leads V4–V6 were associated with occlusion of the LAD distal to the first septal perforator branch.¹⁷ Our finding corroborates with the previous study, showing that patients with ST elevation in V6 had less frequent involvement of the basal antero-septal segment. In the present study, ≥ 0.2 mV ST elevation in leads V5 and V6 was associated with more mid and apical-inferior extension. Q wave in lead V5 was seen significantly more in relation to apex inferior, mid inferior and mid inferoseptal regional perfusion defects. Moreover, patients with Q wave in lead V5 had significantly worse global severity perfusion score and lower ejection fraction than patients without Q wave in those leads, which can be attributed to more inferior and inferoseptal extension. Thus, residual ST-elevation in leads V5 and V6, and Q wave in lead V5 indicate more inferior and not lateral involvement.

Apical Infarction

In almost all our patients with first acute AMI the apical portion of the anterior and antero-septal segments as well as the antero-apical and infero-apical segments (in the long axis view) were involved (Fig 3). No specific ECG pattern was associated with involvement of these segments. However, ST elevation in lead V3 was associated with the involvement of the apical portion of the inferoseptal wall and both ST elevation in leads V4–V6 and Q wave in leads V3–V5 were associated with the involvement of the apical portion of the inferior wall. Previous studies concluded that apical myocardial infarction does not appear to have specific

electrocardiographic findings and is involved in the majority of the patients with acute AMI.^{3,7}

Limitations

This study was not restricted to patients with one-vessel disease. Therefore, disease in vessels other than the LAD coronary artery might have contributed to ischemic ECG patterns or distribution of perfusion defects.

Additional prospective studies in larger populations are desirable in order to elucidate and confirm present results.

CONCLUSIONS

Residual ST elevation and Q waves on the pre-discharge ECG may not have the same significance concerning the exact location of the infarction as the relations between ST elevation in the acute stage of AMI and the ischemic area at risk. Q wave in lead aVL is a sign of less apical and inferior involvement, probably related to occlusion of a short LAD. Q wave in lead aVF and especially in lead II is a sign of more inferior extension of the infarction. Residual ST elevation in leads V2–V4 and Q waves in leads V3–V5 signifies larger infarct size. ST elevation in leads V3 and V4 are not a sign of "anterior" infarction, but of involvement of the apical portion of the inferoseptal wall, whereas ST elevation and Q wave in lead V5 are a sign of more inferior extension of the AMI, and not lateral involvement.

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