VIA MEDICA

ORIGINAL ARTICLE

Folia Cardiol.
2006, Vol. 13, No. 7, pp. 570–577

ISSN 1507–4145

The value of the initial electrocardiogram in the evaluation of an acutely ischaemic area in anterior myocardial infarction

Małgorzata Czechowska, Zdzisława Kornacewicz-Jach, Jarosław Gorący, Krzysztof Przybycień, Joanna Zielonka, Maciej Lewandowski, Andrzej Wojtarowicz and Irmina Kossuth

Department of Cardiology, Pomeranian Medical University, Szczecin, Poland

Abstract

Background: *The aim of the study was to evaluate the importance of admission electrocardiography (ECG) in predicting the extent of acute ischaemia in anterior acute myocardial infarction (AMI)*

Methods: *In 56 patients with anterior AMI electrocardiographic, echocardiographic and angiographic images, troponin I concentration and CK-MB activity were analysed.*

Results: *In 55 cases the artery responsible for infarction was the left anterior descendent (LAD). In the group with proximal occlusions the number of leads with ST elevation was greater (6.6 ± 1.4* vs. *5.3 ± 1.8; p = 0.02) and the level of ST elevations in all leads was higher (18.3 ± ± 9.9* vs. *11.6 ± 7.2; p = 0.01). The mean height of ST elevation in I, aVL, V3 and V5, ST-segment depression in inferior leads and CK-MB activity, was higher in proximal LAD disease. The height of ST elevation in I and aVL correlated with a low ejection fraction and high CK-MB activity. The higher the total ST-segment elevation in all leads, the higher CK-MB activity level.*

Conclusions: *ECG is useful in identifying the site of a LAD occlusion in an anterior AMI. The total ST-segment elevation correlates with the AMI size measured as the maximal CK-MB activity. The height of the ST-segment elevation in leads I and aVL reflects the degree of left ventricle dysfunction. The traditional terminology used to define the localisation of ST-segment shifts in ECG does not take account of the regional wall motion abnormalities observed in echocardiographic examination in an anterior AMI.* (Folia Cardiol. 2006; 13: 570–577)

Key words: electrocardiogram, anterior myocardial infarction, ST-segment elevation, biochemical markers of myocardial necrosis

Editorial p. 543

Introduction

In recent years, after the publication of a new definition of myocardial infarction by the European Society of Cardiology and the American College of

Address for correspondence:
Dr med. Małgorzata Czechowska
Department of Cardiology
Pomeranian Medical University
Powstańców Wlkp. 72, 70–111 Szczecin, Poland
Tel: +48 91 466 13 78, fax: +42 91 466 79
e-mail: malgorzata.czechowska@neostrada.pl
Received: 28.02.2006 Accepted: 2.10.2006

Cardiology, greater attention has been paid to electrocardiography (ECG) [1].

In acute coronary syndromes (ACS) the ECG is not only used for diagnosis. It helps identify the artery responsible for acute ischaemia and predict the artery occlusion site and the extent of the ischaemic area. It is also useful in estimating the risk of adverse outcomes and in making therapeutic decisions.

In anterior acute myocardial infarction (AMI) determination of the exact site of left anterior descending (LAD) coronary artery occlusion is important because the prognosis depends upon this [2, 3]. If the LAD is occluded proximal to the first septal perforator (S1) and/or the first diagonal branch (D1), the extent of cardiac damage is greater. It is an independent risk factor of cardiogenic shock and heart failure [2, 3].

The aim of the study was to evaluate the importance of admission ECG in predicting the extent of acute ischaemia in anterior AMI and especially to demonstrate the relationship between ECG and the echocardiographic and angiographic localisation of acute ischaemia and to compare the extent and type of ECG changes to the markers of the level of cardiac necrosis.

Methods

A study population consisted of 56 patients admitted to the Department of Cardiology of the Pomeranian Medical Academy in 2002–2003 with the diagnosis of anterior AMI. Patients with a pacemaker, a past myocardial infarction, significant valvular heart disease, cardiomyopathy, left ventricular hypertrophy or ion disorder were excluded. All the patients underwent a clinical examination, a 12-lead ECG and coronary angiography. Basic laboratory examinations (including CK-MB activity and troponin I concentration) were performed on each patient immediately after admission. CK-MB and troponin I levels were also examined in the 6^{th} and 12^{th} hours of hospitalisation. CK-MB activity was measured every day up to day four.

ST-segment changes were analysed in all 12 leads of the admission ECG. The TP segment was used as the iso-electric line. ST-segment shifts were measured at 60 ms after the J point to an accuracy of 0.5 mm.

A total of 53 patients underwent an echocardiographic examination before coronary angiography and the remaining three within 12 hours of hospitalisation. Regional wall motion was assessed using the 16-segment method, and categorised as follows: 1 — normal, 2 — hypokinetic, 3 — akinetic, 4 — dyskinetic. The wall motion score index (WMSI) was calculated as the sum of the scores in all 16 segments, divided by 16. The ejection fraction (EF) was estimated using the Simpson method.

Coronary angiography was carried out using the Seldinger method (by the femoral artery approach). The culprit lesions were assessed with respect to their localisation and the extent of stenosis.

For statistical analyses, values were expressed as mean ± standard deviation (SD), minimum and maximum. The normality assumption was verified using the Shapiro-Wilk test. Snedecor's F and the Brown-Forsythe tests were used to test for homogeneity of variance in the groups compared. Owing to the lack of the normality assumption and of homogeneity of variance, non-parametric methods (the Kruskal-Wallis test and the Mann-Whitney U test) were used to standardise analyses when comparing mean values. For comparison of categorical variables Pearson's χ^2 test or the Fisher exact test were used. In order to analyse the relationship between continuous variables the linear correlation and regression methods were used with non-parametric Spearman's rank correlation. A p value of ≤ 0.05 was considered statistically significant.

Results

The study population consisted of 56 patients (of whom 75% were male) of a mean age of 54 years and with a mean body mass index (BMI) of 28 kg/m^2.

ST-segment elevation was found in 53 (95%) patients in lead V2 and in 55 (98%) patients in lead V3. ST-segment elevation ≥ 1 mm in lead V1 was present in 41 patients (73%) and ≥ 2 mm in 20 (36%). ST elevation ≥ 1 mm was found in leads V5 and V6 in 28 (50%) and 15 (27%) patients, respectively. In I and aVL such an elevation was observed in 20 (36%) and 27 (48%) cases, respectively. ST-segment elevation in the inferior leads was found in 4 (7%), 3 (5%) and 2 (4%) patients in leads II, III and aVF, respectively. The percentage of patients with ≥ 1 mm and ≥ 2 mm ST-segment elevation in each lead is presented in Figure 1.

All the patients but one had regional wall motion abnormalities (WMAs) in at least one of the anterior segments (either basal, mid or apical). Regional dysfunction was most prevalent in the apical anterior segment (93% patients). Regional WMAs in the other segments were present as follows: in the apical septal (92%), in the mid-anterior (82%) and in both mid-septal segments (66% and 73% in the anterior and posterior respectively).

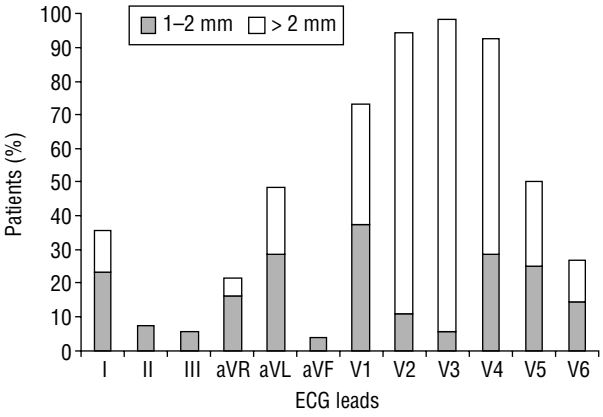

Figure 1. Percentage of patients with ≥ 1 mm and ≥ 2 mm ST-segment elevation in each lead.

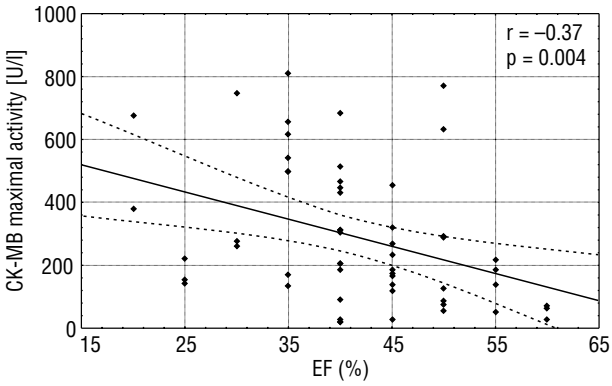

Figure 3. Correlation between ejection fraction (EF) and maximal CK-MB activity.

Only in 22% of patients was regional dysfunction observed in the basal anterior segment and in 17% in the basal septal segments. It was not associated with any ECG changes in comparison with the other patients. In half the study group the regional WMAs were found in the apical inferior and apical lateral segments. The percentage of patients with regional WMA > 1 (hypokinetic) and > 2 (akinetic and dyskinetic) in each of the 16 myocardial segments is presented in Figure 2.

A low EF and high WMSI were associated with high maximal CK-MB activity ($r = –0.37$; $p = 0.004$ and $r = 0.26$; $p = 0.05$ for EF and WMSI, respectively) (Fig. 3).

There were no differences in echocardiographic examination (EF, WMSI and regional WMAs) between the group of patients with and that without ST-segment elevation in leads I and aVL. However, the higher the aforementioned elevation, the worse the EF ($r = –0.30$; $p = 0.03$ and $r = –0.30$; $p = 0.02$ for I and aVL, respectively). Figure 4 shows this relationship.

Maximal CK-MB activity also depended on ST-segment elevation in lead I and was the highest in the group of patients with elevation ≥ 2 mm. The differences were significant and are presented in Table 1.

The ST-segment elevation in lead V1 correlated with the height of the ST-segment elevation in

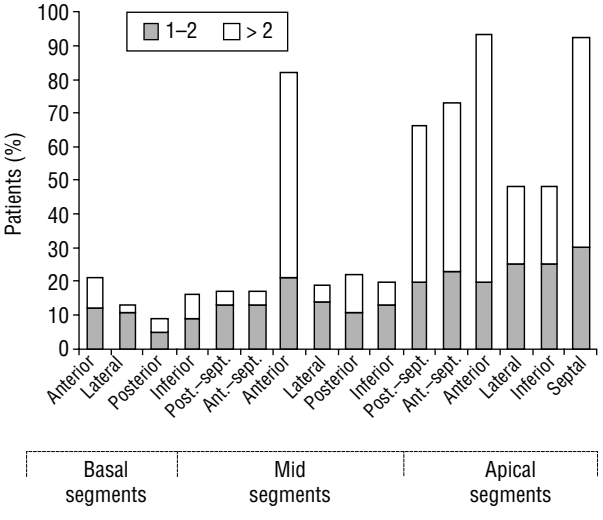

Figure 2. Percentage of patients with regional WMAs > 1 (hypokinetic) and > 2 (akinetic and dyskinetic) in each of the 16 myocardial segments; 1–2 — hypokinetic; > 2 — akinetic or dyskinetic.

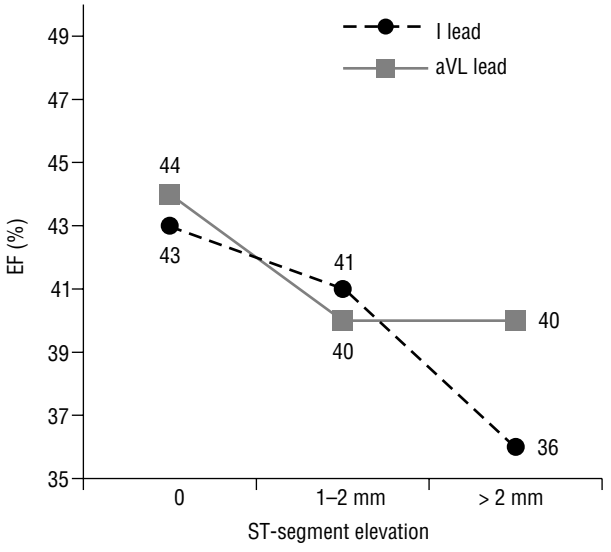

Figure 4. The left ventricle ejection fraction (EF) according to the height of the ST-segment elevation in leads I and aVL.

Table 1. Maximal CK-MB activity according to the height of ST-segment elevation in lead I.

ST-segment elevation height in lead I	Maximal CK-MB activity	
	Mean±SD	Min–max
Without ST elevation[0]	232.5 ± 188.1	19.0–677.9
ST elevation 1 mm ≤ ST < 2 mm[1]	354.9 ± 217.9	119.4–748.1
ST elevation ≥ 2 mm[2]	456.3 ± 281.2	26.1–811.1

$p^{0-1} = 0.04$; $p^{0-2} = 0.04$

aVR (r = 0.3; p = 0.03), which was observed in 12 (21%) patients. There were no significant differences in echocardiographic and angiocardiographic examinations and in the level of maximal CK-MB activity between the group of patients with and that without ST-segment elevation in leads V1 and aVR.

ST-segment elevation in lead V2 was found in 95% of patients. Regional WMAs in the apical inferior and lateral segments were more prevalent in the 47 patients with ST-segment elevation in lead V2 ≥ 2 mm (55%) than in patients with an elevation < 2 mm (0%) and patients with no ST-segment changes (33%). EF, WMSI and maximal CK-MB activity were comparable in the group with and without ST-segment elevation in V2.

It was impossible to make a comparison between groups of patients with and without ST-segment elevation in leads V3 and V4 because of to the low number of patients without any changes in these leads.

There were no significant differences between the patients with and those without ST-segment elevation in leads V5 and V6. However, the maximal CK-MB activity correlated with the height of ST-segment elevation in leads V4 and V5.

The greater the total ST-segment elevation in all 12 ECG leads, the larger the area of myocardial injury as measured by maximal CK-MB activity level (r = 0.28; p = 0.04) (Fig. 5).

There were no differences between groups of patients related to the various kinds of ST-segment elevation (straight, convex or concave) presented.

Among patients with ST-segment depression in leads II, III and aVF the average ST-segment elevation in leads I and aVL was higher than that in patients without ST-segment depression in the inferior leads (Table 2).

The deeper the ST-segment depression in leads V5 and V6, the more segments with WMAs, including akinetic segments, were found in echocardiographic examination (r = 0.33, p = 0.01 and r = 0.34, p = 0.009 for V5 and V6, respectively).

Complete right bundle branch block (RBBB) occurred in four patients, whereas incomplete

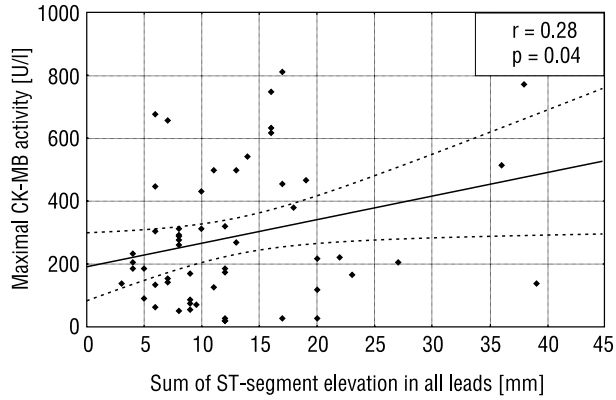

Figure 5. Correlation between the total ST-segment elevation in all leads and maximal CK-MB activity.

RBBB occurred in one patient. There were no differences between this group and the group without any conduction disturbances.

In 55 cases the artery responsible for infarction was the LAD, while in one case it was D1. The culprit lesion was found proximal to the S1 in 12 patients, while in the remaining patients it was distal to S1. There were significant differences between proximal and distal lesions. In the group with proximal occlusions the number of leads with ST-segment elevation was greater (6.6 ± 1.4 *vs.* 5.3 ± 1.8; p = 0.02) and the level of ST-segment elevation in all 12 leads was higher (18.3 ± 9.9 *vs.* 11.6 ± ± 7.2; p = 0.01). In addition, the number of leads with any ST-segment shift (elevation or depression) was greater for the proximal lesions (9.4 ± 2.1 *vs.* 7.0 ± 2.9; p = 0.01). The mean height of ST-segment elevation in leads I, aVL, V3 and V5, as well as ST-segment depression in inferior leads, was greater in proximal LAD disease (Table 3). The area of myocardial injury measured by peak CK-MB activity was significantly higher in the group with the culprit lesion situated proximal to the S1 (413.0 ± 242.4 *vs.* 253.5 ± 198.4; p = 0.04). The subgroups were comparable with regard to the number of segments with regional

Table 2. Height of ST-segment elevation in leads I and aVL in the groups with and without ST-segment depression in leads II, III and aVF.

	ST-segment elevation in leads I and aVL in the group of patients:								
	With ST-segment depression in II	Without ST-segment depression in II	p	With ST-segment depression in III	Without ST-segment depression in III	p	With ST-segment depression in aVF	Without ST-segment depression in aVF	p
I	0.7 ± 0.8	0.3 ± 0.6	0.03	0.8 ± 0.8	0.2 ± 0.5	0.001	0.8 ± 0.7	0.2 ± 0.5	0.002
aVL	1.1 ± 0.7	0.4 ± 0.8	0.0005	1.3 ± 0.8	0.2 ± 0.5	0.000004	1.3 ± 0.7	0.06 ± 0.2	0.000004

Table 3. Electrocardiographic, echocardiographic and biochemical data of patients according to location of the culprit left anterior descending artery lesion in relation to the first septal branch.

Evaluated parameter	Proximal to S1 occlusion		Distal to S1 occlusion		p
	Mean±SD	Min–max	Mean±SD	Min–max	
Number of leads with ST-segment elevation	6.6±1.4	4–9	5.3±1.8	2–9	0.02
Number of leads with ST shifts (↑ or ↓)	9.4±2.1	5–12	7.0±2.9	2–12	0.01
Total ST-segment elevation [mm]	18.3±9.9	5–38	11.6±7.2	3–39	0.01
ST elevation in I	1.2±0.8	0–2	0.3±0.6	0–2	0.003
ST elevation in aVL	1.6±0.9	0–3	0.5±0.6	0–2	0.0007
ST elevation in V3	4.3±2.3	2–10	2.8±1.6	0–8	0.02
ST elevation in V5	2.5±3.0	0–10	0.8±1.1	0–4	0.03
ST depression in III	2.2±1.1	0–4	0.5±0.8	0–3	0.0001
ST depression in aVF	1.3±0.8	0–2	0.4±0.6	0–2	0.002
Maximal CK-MB activity	413.0±242.4	26.1–772.3	253.5±198.4	19.0–811.1	0.04
Left ventricular ejection fraction	37.9±8.1	25–50	42.4±8.8	20–60	0.2
Wall motion score index	1.802±0.445	1.250–2.500	1.631±0.372	1.000–2.563	0.3

S1 — first septal perforator; ↑ST — ST segment elevation; ↓ST — ST segment depression

WMAs, WMSI and EF in echocardiographic images (Table 3).

ST-segment elevation ≥ 1 mm in leads I and aVL was present in 75% and 83% of the occlusions proximal to S1 and in 31% and 39% of the occlusions distal to S1, respectively (I: $p = 0.01$; aVL: $p = 0.005$). Additionally, the ST-segment depression in leads II, III and aVF was more often observed in proximal LAD lesions (65 *vs*. 35%, $p = 0.05$; 92 *vs*. 35%, $p = 0.0005$; 83 *vs*. 35%, $p = 0.003$, respectively).

There were no differences between the group with the occluded LAD proximal to S1 and that with it proximal to D1. The group with a totally occluded artery was comparable to the group with a significantly narrowed artery.

The maximal troponin I concentration was between 0.8–10 μg/l in 22%, between 10–20 μg/l in 18%, between 20–50 μg/l in 20% and over 50 μg/l in 40%. In patients with distal LAD disease, the maximal troponin I concentration was less often over 50 μg/l (17%) when compared to the proximal group (58%) ($p < 0.05$).

Discussion

ST-segment elevation was most prevalent in leads V2 and V3 and, in order of decreasing frequency, in V4, V1, V5, I, V6, aVR, II, III and aVF. Maximal ST-segment elevation (15, 10, 8 mm) was found in V2–V4.

These observations are in good agreement with those reported by other investigators, although ST elevation is usually more frequent in V2 [4].

In echocardiographic examination all patients but one had regional WMAs in at least one of the anterior segments. Regional dysfunction was the most prevalent in the apical anterior and the apical septal segments. Regional WMAs in the mid segments (the anterior and both septal segments) were found in

a significant percentage of patients. Only a small percentage of patients presented regional dysfunction in the basal segments (the anterior and the septal).

Despite the fact that echocardiography is essential in the diagnostic of ACS, there are no large trials to compare regional WMA with the presence of ST-segment shifts in various leads during the acute phase of anterior AMI [4–6].

Porter et al. [4], in a group of 132 patients with anterior AMI, observed similar proportions of patients with regional WMA in individual segments of the left ventricle.

Lead I faces the lateral region of the left ventricle along with lead aVL, which faces the basal portion of the anterolateral free wall of the left ventricle [7]. ST elevation in these leads, accompanying ST elevations in leads V5 and V6, has traditionally been defined as high lateral [7]. Previous studies have shown that the aforementioned ECG changes accompanying ST shifts in precordial leads were highly predictive of LAD occlusion proximal to D1 [8–10].

Porter et al. [4] concluded that there was no significant difference in the occurrence of regional WMA in the lateral segments between patients with and without ST-segment elevation in leads I and aVL. However, ST elevation in lead aVL was associated with regional dysfunction in the basal anterior segment [4].

The results of this study indicate that, even though the ST-segment elevation in leads I and aVL was associated with proximal LAD occlusion, there were no differences in echocardiographic images between the groups with and without ST elevation in these leads. However, if there was ST elevation in I and aVL, its height correlated with low EF and high maximal CK-MB activity, which shows a more extensive myocardial necrosis area in this group.

Leads V1 and V2 are thought to face the interventricular septum [5–7]. In traditional terminology, which has been based on reports comparing autopsy findings with the distribution of pathological Q waves, anterior AMI was classified as septal if Q waves were present in leads V1 and V2 [5–7]. Previous studies about the correlation between ST-segment elevation in leads V1, V2 and regional WMAs in septal segments are not unanimous. Shalew et al. [6] observed that ST-segment elevation in leads V1–V3 correlated with anterior (94% of patients) and apical (100% of patients) regional dysfunction but rarely with septal regional WMAs (19% of patients), which was confirmed by the observations of Bogaty et al. [8]. In two studies performed by Porter et al. [4, 5] the authors showed that patients with ST elevation in V1, when compared to those without ST shifts, more often had regional WMA in the basal septal segments (57% *vs*. 16%; $p = 0.003$; 43% *vs*. 13%, $p = 0.003$, anterior and posterior respectively) and in the basal anterior segment (43% *vs*. 11%, $p = 0.01$). However, ST elevation in lead V2 was associated with regional dysfunction in the apical inferior segment. The authors emphasise the differences between leads V1 and V2 which, although adjacent, face different myocardium areas.

In our study, we found no difference in the echocardiographic images of patients with and patients without ST-segment elevation in lead V1. However, our observations about the group with ST-segment elevation in V2 are similar to these presented by Porter et al. The high (≥ 2 mm) ST elevation in V2 was associated with regional dysfunction in the apical inferior and apical lateral segments.

ST-segment elevation in V1 correlated with ST-segment elevation in aVR. This fact may suggest that, although they lie on perpendicular planes, they are adjacent and represent ischaemia of the same area.

The myocardial area thought to be represented by lead V1 is supplied by septal branches of the LAD [9, 10]. However, in many cases the septum is supplied doubly, additionally by the conal branch of the right coronary artery or by branches of the circumflex coronary artery [10]. Many authors consider ST-segment elevation in lead V1 as indicative of LAD occlusion proximal to S1 [11, 16–18]. These observations, however, did not find confirmation in the studies of Ben-Gal et al. [9]. Our findings are with accordance with the Ben-Gal findings. We found no difference between the group with and that without ST elevation in V1 with respect to LAD site occlusion.

In our study, we found a correlation between the total ST-segment elevation and AMI size, expressed as a peak of CK-MB activity. There is no agreement in the literature about the usefulness of the total ST-segment elevation for the evaluation of AMI size [11–14]. Aldrich et al. [12] demonstrated that the number of leads with ST elevation and the total ST elevation are closely correlated with infarction size. They proposed a formula based on quantitative measurements of ST elevation on the initial ECG to predict the final AMI size. Clemmensen et al. [13], testing the usefulness of a modified Aldrich formula, claimed that both the height of the ST-segment elevation and the number of leads with ST elevation correlated with AMI size ($r = 0.63$; $r = 0.65$). In contrast, Christian et al. [14]

reported that the number of leads with ST-segment elevation in an anterior AMI were not predictive of the technetium 99m-sestamibi.

Sadanandan et al. [11] compared the left ventricle EF and the number of segments with regional WMAs with maximal CK-MB activity in three groups of patients with different numbers of leads with ST-segment elevation and different sums of ST elevation: those with ST elevation in both the anterior and inferior leads, those with ST-segment elevation in the anterior leads only and those with ST-segment elevation in the anterior leads and ST-segment depression in the inferior leads. Of these the first group was found to have the best left ventricular EF (53% *vs*. 49% *vs*. 45%, $p = 0.0001$), the lowest number of segments with regional WMAs (21% *vs*. 32% *vs*. 40%, $p = 0.0001$) and the lowest peak of CK-MB activity (1370 *vs*. 1670 *vs*. 2381 IU, $p = 0.0001$). The authors emphasise that, despite a greater ST elevation, patients with combined anterior and inferior ST elevation have limited AMI size and better left ventricle function. This paradoxical result is explained by the distal occlusion of wrap-around LAD. In these cases the AMI size is smaller.

The angiographic image and the influence of the occlusion site of LAD on the ECG changes in ACS have been subjects of numerous studies in the last few years [11, 15–23]. In the present study, the culprit lesion in the LAD was found proximal to the S1 in 12 patients. Only one of these patients had the D1 branch take-off from the LAD proximal to the S1. In 14 cases the site of LAD occlusion was localised between two main LAD branches, while in the remaining cases it was distal to both of them. In all patients the take-off of S1 from the LAD was very close to D1. This fact may explain why no differences were found between the groups with regard to the D1 branch. Only with reference to the S1 were differences observed. Patients with an occlusion site proximal to S1 were characterised by a greater number of leads with ST-segment elevation and a greater total ST-segment elevation. In the proximal group ST elevation in leads I and aVL were presented more frequently. Its mean height and the mean ST-segment depression in leads III and aVF were significantly higher.

Similar findings have recently been published by many other investigators [15, 16, 18, 19]. When comparing patients with proximal LAD disease with those with a distal localisation of the lesion, Birnbaum et al. [16] observed a higher mean ST-segment elevation in I and aVL in the former ($0.86 \pm \pm 1.27$ *vs*. 0.31 ± 0.77; $p < 0.01$ and 2.01 ± 4.79 *vs*. 0.40 ± 0.81; $p < 0.03$, respectively) as well as a deeper mean ST-segment depression in leads II, III, aVF (1.00 ± 0.88 *vs*. 0.1 ± 0.76; 1.48 ± 1.32 *vs*. 0.34 ± 0.84 and 1.33 ± 1.17 *vs*. 0.25 ± 0.73; $p < 0.0001$, respectively). Tamura et al. [18] demonstrated that ST-segment elevation ≥ 1 mm in aVL was more frequent among patients with the culprit lesion situated proximal to the S1 than in those with a distal location (66 *vs*. 47%; $p < 0.05$). The same was true of ST-segment depression in the inferior leads (81% *vs*. 27%, 85% *vs*. 54% and 87% *vs*. 47% in II, III and aVF, respectively, $p < 0.01$). Engelen et al. [15] proved that ST-segment elevation in lead aVR, RBBB, ST-segment depression in V5 and ST--segment elevation greater than 2.5 mm in V1 were characteristic of a LAD occlusion site proximal to S1.

Our study did not confirm this relationship. These results can be explained by the aforementioned changeable vascularisation of the intraventricular septum and concomitant significant lesions in other coronary arteries.

Engelen et al. [15] and Vasudevan et al. [19] drew attention to the higher maximal CK-MB activity in cases with lesions proximal to S1 (3948 *vs*. 2238 IU; $p = 0.01$) and to D1 (3333 *vs*. 2239 IU; $p = 0.05$). Our observations are in accordance with those of these authors. In the present study the area of myocardial injury measured as maximal CK-MB activity, predominated in the group with proximal LAD dysfunction. In 58% of these patients the troponin I concentration was over 50 μg/l.

Conclusions

1. ECG is useful in identifying the site of an LAD occlusion in an anterior AMI.
2. The total ST-segment elevation correlates with the size of the AMI as measured by maximal CK-MB activity.
3. The height of ST-segment elevation in leads I and aVL reflects the degree of left ventricle dysfunction measured as EF.
4. Deep ST-segment depression in leads V5 and V6 identifies patients with great regional WMAs.
5. Traditional terminology used to define the localisation of ST-segment shifts in ECG does not take account of regional WMAs observed in echocardiographic examination in an anterior acute myocardial infarction.

References

1. From The Joint European Society of Cardiology/ /American College of Cardiology Committee. Myocardial infarction redefined. A consensus document of The Joint European Society of Cardiology/American College of Cardiology Committee for Redefinition of Myocardial Infarction. Eur Heart J, 2000; 21: 1502–1513.
2. Miller WL, Sgura FA, Kopecky SL et al. Characteristics of presenting electrocardiograms of acute myocardial infarction from a community-based population predict short- and long-term mortality. Am J Cardiol, 2001; 87: 1045–1050.
3. Karha J, Murphy SA, Kirtane AJ et al. Evaluation of the association of proximal coronary culprit artery lesion location with clinical outcomes in acute myocardial infarction. Am J Cardiol, 2003; 92: 913–918.
4. Porter A, Wyshelesky A, Strasberg B et al. Correlation between the admission electrocardiogram and regional wall motion abnormalities as detected by echocardiography in anterior acute myocardial infarction. Cardiology, 2000; 94: 118–126.
5. Porter A, Strasberg B, Vaturi M et al. Correlation between electrocardiographic subtypes of anterior myocardial infarction and regional abnormalities of wall motion. Coron Artery Dis, 2000; 11: 489–493.
6. Shalew J, Fogelman R, Oettinger M et al. Does the electrocardiographic pattern "anteroseptal" myocardial infarction correlate with the anatomic location of myocardial injury? Am J Cardiol, 1995; 75: 763–766.
7. Surawicz B, Uhley H, Borum K et al. Task force I: standardization of terminology and interpretation. Am J Cardiol, 1978; 41: 130–145.
8. Bogaty P, Boyer L, Rousseau L et al. Is anteroseptal myocardial infarction an appropriate term? Am J Med, 2002; 113: 37–41.
9. Ben-Gal T, Herz I, Solodky A et al. Acute anterior wall myocardial infarction entailing ST-segment elevation in lead V1: electrocardiographic and angiographic correlations. Clin Cardiol, 1998; 21: 399–404.
10. Ben-Gal T, Sclarovsky S, Herz I et al. Importance of the conal branch of the right coronary artery in patients with acute anterior wall myocardial infarction: electrocardiographic and angiographic correlation. J Am Coll Cardiol, 1997; 29: 506–511.
11. Sadanandan S, Hochman JS, Kolodziej A et al. Clinical and angiographic characteristics of patients with combined anterior and inferior ST-segment elevation on the initial electrocardiogram during acute myocardial infarction. Am Heart J, 2003; 146: 653–661.
12. Aldrich HR, Wagner RB, Boswick J et al. Use of initial ST-segment deviation for prediction of final electrocardiographic size of acute myocardial infarcts. Am J Cardiol, 1998; 61: 749–753.
13. Clemmensen P, Grande P, Aldrich HR et al. Evaluation of formulas for estimating the final size of acute myocardial infarcts from quantitative ST-segment elevation on the initial standard 12-lead ECG. J Electrocardiol, 1991; 24: 77–83.
14. Christian TF, Gibbons RJ, Clements IP et al. Estimates of myocardium at risk and collateral flow in acute myocardial infarction using electrocardiographic indexes with comparison to radionuclide and angiographic measures. J Am Coll Cardiol, 1995; 26: 388–393.
15. Engelen DJ, Gorgels AP, Cheriex EC et al. Value of the electrocardiogram in localizing the occlusion site in the left anterior descending coronary artery in acute anterior myocardial infarction. J Am Coll Cardiol, 1999; 34: 389–395.
16. Birnbaum Y, Sclarowsky S, Solodky A et al. Prediction of the level of left anterior descending coronary artery obstruction during anterior wall acute myocardial infarction by the admission electrocardiogram. Am J Cardiol, 1993; 72: 823–826.
17. Yasuhiro A, Hiroatsu Y, Toshiya F et al. Electrocardiographic diagnosis of the coronary artery culprit site in ischaemic heart disease. Circ J, 2003; 67: 775–780.
18. Tamura A, Kataoka H, Mikuriya Y, Nasu M. Inferior ST segment depression as useful marker for identifying proximal left anterior descending artery occlusion during acute anterior myocardial infarction. Eur Heart J, 1995; 16: 1795–1799.
19. Vasudevan K, Manjunath CN, Srinivas KH et al. Electrocardiographic localization of the occlusion site in left anterior descending coronary artery in acute anterior myocardial infarction. Indian Heart J, 2004; 56: 315–319.
20. Porter A, Sclarovsky S, Ben-Gal T et al. Value of T-wave direction with lead III ST-segment depression in acute anterior wall myocardial infarction: electrocardiographic prediction of "wrapped" left anterior descending artery. Clin Cardiol, 1998; 21: 562–566.
21. Schmitt C, Lehmann G, Schmieder S et al. Diagnosis of acute myocardial infarction in angiographically documented occluded infarct vessel. Limitations of ST-segment elevation in standard and extended ECG leads. Chest, 2001; 120: 1540–1546.
22. Sasaki K, Yotsukura M, Sakata K et al. Relationship of ST-segment changes in inferior leads during anterior wall acute myocardial infarction to length and occlusion site of the left anterior descending coronary artery. Am J Cardiol, 2001; 87: 1340–1345.
23. Arbane M, Goy J. Prediction of the site of total occlusion of the left anterior descending coronary artery using admission electrocardiogram in anterior wall acute myocardial infarction. Am J Cardiol, 2000; 85: 487–491.