Acute myocardial infarction entailing ST-segment elevation in lead aVL: Electrocardiographic differentiation among occlusion of the left anterior descending, first diagonal, and first obtuse marginal coronary arteries

Yochai Birnbaum, MD, David Hasdai, MD, Samuel Sclarovsky, MD, Izhak Herz, MD, Boris Strasberg, MD, and Eldad Rechavia, MD Tel Aviv, Israel

Acute myocardial infarction with ST elevation in lead aVL may represent involvement of the first diagonal or the first obtuse marginal branch. This study assesses the correlation among different electrocardiographic patterns of acute myocardial infarction with ST elevation in aVL and the site of the infarct-related artery occlusion. Patients who underwent coronary angiography within 14 days of infarction with an unequivocal culprit lesion were included. Fifty-seven patients were evaluated. The culprit lesion was in the left anterior descending coronary artery proximal to the first diagonal, first diagonal, and first obtuse marginal branches, in 38, 8, and 11 patients, respectively. ST elevation in aVL and V2 through V5 signifies left anterior descending artery occlusion proximal to the first diagonal branch (positive predictive value [PPV] and negative predictive value [NPV] of 95% and 94%, respectively). ST elevation in aVL and V2, not accompanied by ST elevation in V3 through V5, favors occlusion of the first diagonal branch (PPV, 89%; NPV, 100%). ST elevation in aVL accompanied by ST depression in V2 predicts obstruction of the first obtuse marginal branch (PPV, 100%; NPV, 98%). (Am Heart J 1996;131:38-42.)

Determining the infarct-related coronary artery during coronary angiography is frequently difficult because of the presence of more than one lesion. Other confounding factors may be related to the large individual variation in the number, size, and location of branches originating from the three main coronary arteries. Thus complete obstruction at the origin of such branches may go unnoticed. In such cases, determining the culprit lesion is usually facilitated by data derived from wall motion imaging (echocardiography, cineventriculography, and radionuclide ventriculography) and the electrocardiographic (ECG) pattern.

Two recent studies described the ECG features of acute myocardial infarction (AMI) caused by occlusion of the first diagonal branch. In both studies ST-segment elevation in lead aVL was the predominant ECG feature. However, ST-segment elevation in lead aVL may be found in some AMIs caused by left circumflex coronary artery occlusion. The ECG pattern accompanying ST-segment elevation in lead aVL from left circumflex coronary artery occlusion is still not well characterized. We postulated that by characterizing unique ECG patterns associated with occlusion of a particular coronary artery, it may be possible to better predict the site of the culprit lesion in the clinical setting. This study assesses patients with AMI with ST-segment elevation in lead aVL. The patterns of ST-segment deviation in the various ECG leads are analysed to characterize unique ECG patterns related to occlusion of each coronary vessel.

METHODS

Patients. Patients admitted to the coronary care unit with AMI from July 1988 to May 1993 were evaluated retrospectively. AMI was diagnosed on the basis of the presence of chest pain lasting more than 30 minutes, evolving characteristic ECG abnormalities that included ST elevation of ≥0.1 mV in lead aVL and ST elevation of ≥0.1 mV in at least one additional lead, and an increase in serum creatine kinase levels to more than twice the upper limit of normal. Only patients who underwent coronary angiography within 14 days of AMI were included. Patients with pacemakers or previous coronary artery bypass surgery and patients who received digitalis before admission or had ECG evidence of left ventricular hypertrophy, bundle branch block, or advanced ECG stages of infarction (inverted T waves or fully evolved Q waves in the involved leads) were excluded.
**ECG evaluation.** All standard 12-lead ECG recordings of the acute phase were evaluated by two investigators blinded to the angiographic findings. The ECGs were recorded at a paper speed of 25 mm/sec at a calibration of 1 mV = 10 mm. ST-segment deviation from the isoelectric line, determined by a line drawn between subsequent TP segments, was measured manually to the nearest 0.5 mm in every lead at 0.06 second after the J point. The ST segment was considered elevated or depressed if it was ≥0.1 mV above or below the isoelectric line, respectively. Otherwise, it was considered isoelectric.

**Coronary angiography.** Selective coronary cineangiography was performed within 14 days of admission by the Judkins technique with multiple projections. Each cineangiogram was analyzed by two observers who were blinded to the ECG findings. When more than one lesion was present, the site of the culprit lesion was determined by the appearance of complete obstruction of the artery or by the angiographic characteristics of the lesion (presence of either residual thrombus or ulcerated plaque). In the case of disagreement between the two investigators, the patient was excluded. The number of vessels with >70% luminal stenosis was recorded.

**Statistics.** Patients were divided into three groups according to the site of the infarct-related artery determined by the angiographic findings. The three groups were compared for a number of variables by the Fisher Exact Test for categoric data. A p value <0.05 was considered statistically significant.

**RESULTS**

Sixty-one patients were evaluated. Four patients were excluded because of inability to identify the exact culprit lesion with certainty. Fifty-seven patients were included (50 men and 7 women aged 55.1 ± 11.3 years). The culprit lesion was in the left anterior descending coronary artery proximal to the first diagonal branch (LAD group), the first diagonal branch (DIAG group), and the first obtuse marginal branch of the first circumflex artery (OM1 group) in 38, 8, and 11 patients, respectively. Seven patients from the DIAG group have been previously described. Single-, two-, and three-vessel disease was present in 32, 21, and 4 patients respectively. Fig. 1 depicts representative 12-lead ECG recordings obtained on the acute phase from one patient from each group.

**ST deviation in the lateral leads.** By definition, all patients had ST elevation in lead aVL. Fifty-five percent, 75%, and 55% of the patients in groups LAD, DIAG, and OM1, respectively, had ST elevation in lead I (p not significant [NS]) (Table I). The LAD group showed a significantly higher prevalence of ST-segment elevation in lead V5 (74%) than the DIAG (0%) and the OM1 (18%) groups (p < 0.0001) (Table I). ST-segment depression in V5 (p < 0.0001) and V6 (p = 0.02) was more prevalent in the DIAG group (Table II).

**ST deviation in the inferior leads.** ST-segment elevation was not found in leads II, III, or aVF in any of the LAD or DIAG groups but was found in lead II in two patients in the OM1 group (p = 0.05) (Table I). ST depression in leads II and aVF was less prevalent in the OM1 group (p = 0.0001 and <0.03, respectively) (Table II).
Table I. Prevalence of ST-segment elevation in three groups

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<tr>
<th>ST elevation</th>
<th>I</th>
<th>II</th>
<th>III</th>
<th>aVR</th>
<th>aVF</th>
<th>V1</th>
<th>V2</th>
<th>V3</th>
<th>V4</th>
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<tr>
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<td>%</td>
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<td>NS</td>
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Table II. Prevalence of ST-segment depression in three groups

<table>
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<th>III</th>
<th>aVR</th>
<th>aVF</th>
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<th>V2</th>
<th>V3</th>
<th>V4</th>
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</tr>
<tr>
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<td>2</td>
<td>5</td>
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<tr>
<td>p Value</td>
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<td>NS</td>
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ST deviation in the anterior precordial leads. ST-segment elevation in lead V1 was found in 87% of the LAD group but in only 25% of the DIAG group. None of the patients in the OM1 group had ST-segment elevation in lead V1 (p < 0.0001) (Table I). Although ST-segment elevation in lead V2 was found in all the LAD and DIAG patients, it was evident in only one patient in the OM1 group (p < 0.0001) (Table I). In contrast, ST-segment depression in lead V2 was found in 64% of the OM1 group, but not in the LAD and DIAG groups (p < 0.0001). Thus when the ST segment in lead V2 is either isoelectric or negative, the positive predictive value for OM1 lesion is 100% (Fig. 2). ST-segment elevation in leads V3 and V4 was seen in 97% and 87%, respectively, in the LAD group but in none of the DIAG group and in only 18% of the OM1 group (p < 0.0001). Thus in patients with ST-segment elevation in leads aVL and V2, ST elevation in V3 has a 95% positive predictive value and 94% negative predictive value for LAD lesion (Fig. 2). Conversely, ST-segment depression in lead V4 was found in all patients in the DIAG group, in 36% of the OM1 group, and in only 5% of the LAD group (p < 0.0001) (Table I). Thus in the context of ST-segment elevation in leads aVL and V2, isoelectric or negative ST segment in lead V3 has a 89% positive predictive value and 100% negative predictive value for DIAG lesion (Fig. 2).

DISCUSSION

This study demonstrates that ST-segment elevation in lead aVL during AMI may be induced by occlusion of either the LAD (proximal to the origin of the first diagonal branch), the first diagonal branch itself, or the first obtuse marginal branch. Lead aVL faces the basal portion of the lateral free wall of the left ventricle. This area is supplied by both the first diagonal and the first obtuse marginal branches. Theoretically AMI with ST-segment elevation in aVL may represent involvement of either of these two branches or a lesion in the LAD or left circumflex arteries, proximal to their origin. Previous studies reported that ST-segment elevation in aVL in anterior wall AMI is highly predictive, albeit with relatively low sensitivity, of LAD occlusion proximal to the first diagonal branch. Recently Iwasaki et al. and Sclarovsky et al. described the ECG pattern of first diagonal branch occlusion. In both studies, all patients showed ST-segment elevation in lead aVL during the acute phase. The prevalence of ST-segment elevation in lead aVL in AMI caused by left circumflex artery occlusion is low but not negligible. Huey et al. found ST-segment elevation in aVL in 16% of 19 patients with left circumflex artery infarction. The most frequent ECG manifestation of left circumflex artery infarction is ST elevation in the inferior leads.
whereas in the majority of patients lateral and posterolateral AMI are electrocardiographically silent. Because inferior leads, especially leads III and aVF, are opposed to lead aVL, ECG changes in leads facing both the inferior and high lateral regions tend to cancel each other. This is probably one of the reasons why ST-segment elevation is seen less often in left circumflex artery–associated AMI than in AMI caused by LAD or right coronary artery occlusion. In our study, ST-segment elevation in inferior leads III and aVF did not occur in patients with left circumflex artery obstruction and ST elevation in aVL (Table I). This is probably because the culprit lesion was not found in the left circumflex trunk itself in any patient. Involvement of the inferior myocardial region probably tends to attenuate ST-segment elevation in aVL or even causes reciprocal ST-segment depression. However, when the first obtuse marginal is occluded, the changes in aVL can be manifested, as in our study.

The differential diagnosis among LAD, DIAG, and OM1 occlusion lies in the ECG changes in the precordial and inferior leads. Although ST-segment elevation in lead V2 was found in all LAD and DIAG patients, it was found in only one patient in the OM1 group (Table I). In contrast, ST-segment depression in lead V2 was observed in 64% of the OM1 patients (Table II). This finding concurs with previous reports. ST-segment depression in lead V2 is associated with posterior involvement of AMI caused by left circumflex (or its marginal branches). Thus ST-segment depression in lead V2 indicates OM1 occlusion, whereas ST-segment elevation in V2 favors LAD or DIAG occlusion.

ST-segment elevation in the precordial leads V3 through V5 was found to be predominantly the result of LAD occlusion. ST-segment elevation in leads V3 through V5 was not found in any patient in the DIAG group or in the population studied by Iwasaki et al. In contrast, we found that ST-segment depression in leads V3 through V5 may be found in either DIAG or OM1 occlusion.

ST-segment depression in the inferior wall is commonly found during anterior AMI caused by proximal LAD or DIAG occlusion. It merely represents reciprocal changes to the anterolateral infarction. In our study, ST-segment depression in the leads facing the inferior wall was seen less often in OM1 infarction (Table II).

Conclusions. ST-segment elevation in lead aVL during AMI indicates either proximal LAD artery, DIAG, or OM1 occlusion. If ST-segment elevation is found in aVL and the precordial leads V3 through V5, the culprit lesion is most probably located in the proximal portion of the LAD artery before the origin of the first diagonal branch. ST-segment elevation in leads aVL and V2, accompanied by either isoelectric or depressed ST segment in leads V3 through V5, predicts obstruction of the first diagonal branch. ST elevation in aVL accompanied by isoelectric or negative ST segment in the precordial leads, including V2, predicts obstruction of the first obtuse marginal branch.

Study limitations. This is a preliminary and retrospective study of a relatively small number of patients. We limited ourselves to patients whose qualifying ECG demonstrated the acute phase of AMI only (ST-segment elevation with positive T waves in the involved leads). We chose this criterion because the ECG in the acute phase is more closely related to the area supplied by the culprit artery than the ECG in more advanced stages of infarction. None of our patients had ramus intermedius branch occlusion. Because the course of the ramus intermedius branch is intermediate between the diagonal and obtuse marginal branches, the ECG pattern of ramus intermedius branch occlusion may probably resemble either diagonal or obtuse marginal branch obstruction. In this study we included only patients who had coronary angiography within 14 days of admission. The decision concerning coronary angiography was based on accepted clinical indications. Thus the real prevalence of ST-segment elevation in lead aVL during left circumflex artery, obtuse marginal
branch, or diagonal branch obstruction remains unknown. However, this study demonstrates that ST elevation in aVL might be found in either proximal LAD artery, DIAG, or OM1 obstruction and that the ECG pattern in the precordial leads might be helpful in differing among the three locations. There is a need for a prospective study to determine the sensitivities and specificities of our findings.

REFERENCES