

ST segment depression in aVL: a sensitive marker for acute inferior myocardial infarction

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In a substantial percentage of patients with acute myocardial infarction, especially in those with inferior wall involvement, no ST elevation is detected on the electrocardiogram. In many of them, ST depression is found in leads oriented to remote segments of the heart. The importance of those reciprocal changes for early diagnosis of acute inferior myocardial infarction in patients without ST elevation has not been stressed. In order to find the prevalence of reciprocal ST depression, we evaluated the admission electrocardiograms of 107 consecutive patients with evolving first acute inferior myocardial infarction. Ninety-three patients had ST elevation of at least 0.1 mV in at least one of the inferior leads: II, III or aVF (group A) and in 14 patients ST displacement did not reach 0.1 mV in any of these leads (group B). In both groups, reciprocal ST depression occurred more frequently in aVL than in any other lead. Only three patients had no ST depression in aVL. In eight patients (7.5%) ST depression in aVL was the sole early electrocardiographic sign of the inferior infarction. aVL is the only lead that is facing the superior part of the left ventricle and thus is the only lead that is truly opponent to the inferior wall. It seems that ST depression in aVL, by contrast to that in the precordial leads, is found in the majority of patients with evolving inferior wall myocardial infarction and is not influenced by extension of the infarction to the right ventricle or to the posterior wall. We conclude that transient ST depression in aVL is a sensitive early electrocardiographic sign of acute inferior wall myocardial infarction.

Introduction

Only 18% of the patients admitted for an acute myocardial infarction (AMI), and only 52% of those eligible for thrombolytic therapy, are actually receiving this therapy^[1]. This under-utilization of thrombolysis in AMI is due in part to the requirement of ischaemic ST elevation of at least 0.1 mV on the electrocardiogram (ECG) as a diagnostic criterion of AMI^[2–7]. Ischaemic ST depression is usually not considered as a diagnostic ECG criterion for AMI^[1]. In some patients with AMI, particularly in those with posterior wall AMI, no ST elevation is detected in the standard 12-lead ECG and the electrocardiographic changes consist of ST depression^[8]. This may also apply to a substantial portion of patients with inferior AMI, especially those resulting from a left circumflex artery thrombosis^[9].

The clinical significance of reciprocal changes has been extensively studied, but their importance for the early diagnosis of inferior AMI, particularly in those patients without significant ST elevation, has not been stressed. To determine the diagnostic significance of reciprocal ST depression in the early stage of acute inferior AMI, we analysed the ECG of patients with an evolving first AMI, which proved to be located inferiorly. We examined the prevalence of reciprocal changes in the different leads in patients with and without ST elevation in the inferior

leads at the early stage of an inferior AMI and compared it to the prevalence of ST elevation in the inferior leads.

Patients and methods

PATIENTS

The admission ECG of 107 consecutive patients admitted to the coronary care unit from January 1989 to March 1991 with an evolving first inferior AMI were evaluated. Acute myocardial infarction was diagnosed when the following criteria were met: prolonged chest pain of more than 30 min duration suggestive of AMI, diagnostic increase and decrease of serum creatine kinase and evolution of new Q waves in at least two of the inferior leads, II, III and aVF following the acute event. Patients with negative T wave or Q wave in II, III and aVF on admission, and patients with bundle branch block or ECG signs of left ventricular hypertrophy were excluded. Also, patients with a history of previous AMI or of a coronary artery bypass graft or patients with significant valvular, congenital or cardiomyopathic heart disease were not included.

ELECTROCARDIOGRAPHIC EVALUATION

Standard 12-lead ECG obtained on admission at a calibration of 10 mm = 1 mV were evaluated. ST segment displacements were measured 0.08 s after the J point, using the preceding TP segment as a baseline. Patients were allocated into two groups according to the presence or absence of ST elevation in II, III and aVF. Group A consisted of patients with ST segment elevation of at least 0.1 mV in one lead or more. Group B consisted of patients

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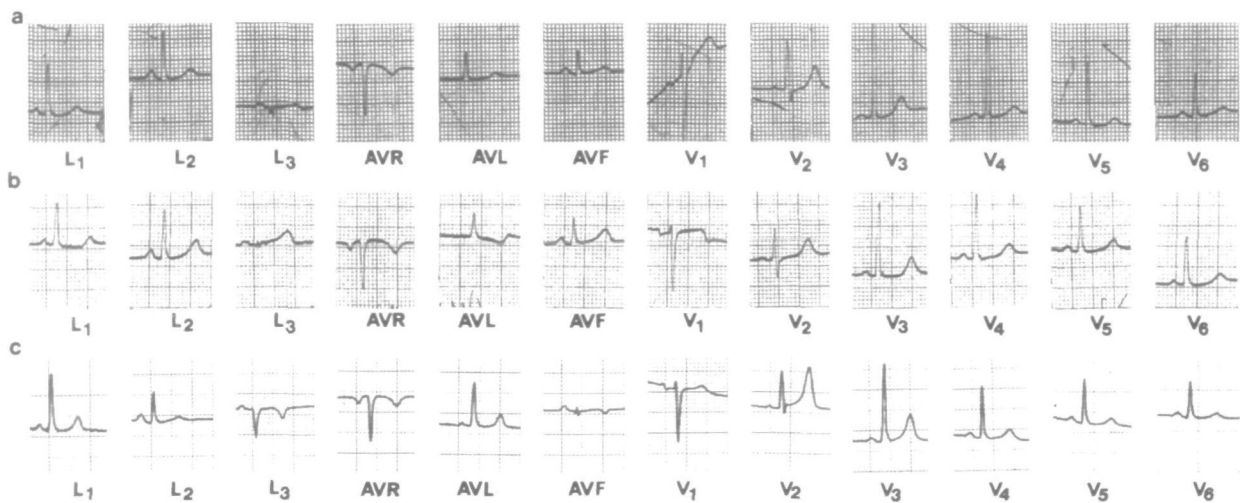


Figure 1 A 63-year-old woman admitted with acute inferior wall myocardial infarction. (a) routine electrocardiogram a month before admission. No ST deviation is found; (b) half an hour after beginning of chest pain. The ECG demonstrates minimal ST elevation of less than 0.1 mV in III and in aVF and ST depression of 0.05 mV in aVL. The T wave in aVL is biphasic. No reciprocal changes are found in the precordial leads or in I; (c) 48 h after the beginning of chest pain. There is a new Q wave in III, and the amplitude of the R wave in II and aVF is decreased. T wave inversion in aVF and III is seen and there is a tall T wave in V₂ and V₃. The ST depression in aVL subsequently resolved and the T wave became positive again.

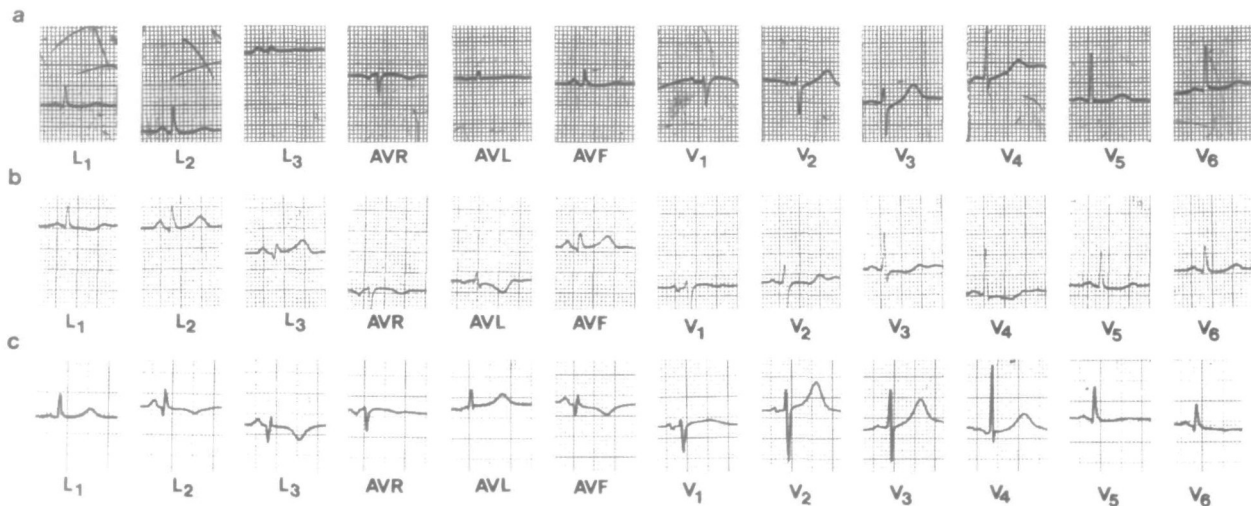


Figure 2 A 57-year-old man admitted with acute inferior wall myocardial infarction. (a) routine electrocardiogram 5 months before admission. No ST deviation is found; (b) one hour after the beginning of chest pain. The ECG demonstrates ST elevation of less than 0.05 mV in II, III and in aVF. There are minimal ST-T changes in V₂, V₄, but clear ST depression with negative T wave is seen in aVL; (c) ECG taken 5 days after admission. There is a new Q wave and a negative T wave in II, III and in aVF. The ST depression and T wave inversion in aVL resolved.

in whom ST segment displacement did not reach 0.1 mV in any of these leads (Figs 1 and 2).

Results

One hundred and seven patients were included in the study, 93 in group A and 14 in group B. Data are depicted in Table 1. Seven out of the 14 patients in group B subsequently developed ST elevation in the inferior leads on serial ECG. Significant (≥ 0.1 mV) reciprocal ST depression occurred more frequently in aVL than in any

other lead in both groups, in 78 of the patients in group A and in five of the patients in group B (Fig. 3). Another 21 patients had ST depression of 0.05 mV (15 in group A and 6 in group B). Only three patients had no ST depression in aVL. For comparison, ST depression in leads I, V₁, V₂, V₃, V₄, V₅, and V₆ was found in 65%, 38%, 78%, 65%, 57%, 41%, and 25% of the patients in group A, respectively, and in 50%, 14%, 29%, 43%, 36%, 50%, and 21% of the patients in group B, respectively (Fig. 3). Mean ST depression in aVL was greater than in I or the precordial leads (Table 2). In eight patients (7.5%) ST depression in

Table 1 Clinical characteristics

	Group A*	Group B*
No. of patients	93	14
Age (years)	53.1 ± 16.3	75.2 ± 20.3
Sex		
Male	72	11
Female	21	3

*Group A, patients with ST elevation ≥ 0.1 mV in at least one lead of II, III or aVF on the admission ECG. group B, patients without ST elevation ≥ 0.1 mV in II, III or aVF on the admission ECG

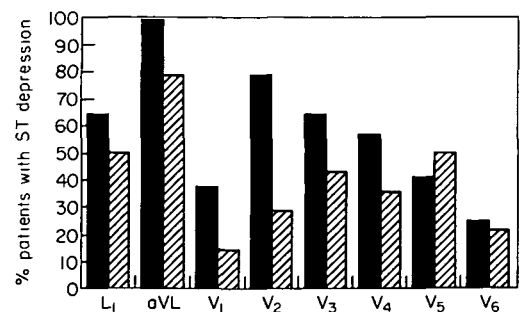


Figure 3 Prevalence of ST depression (0.05 mV or more) in I, aVL and the precordial leads in patients with acute inferior wall myocardial infarction. Group A (■) 93 patients with ST elevation of at least 0.1 mV in one of the inferior leads (II, III aVF). Group B (▨) 14 patients without ST elevation in any of the inferior leads.

Table 2 Magnitude of ST deviation in mV (mean ± standard deviation) in the standard 12-lead ECG

	I	aVL	V ₁	V ₂	V ₃	V ₄	V ₅	V ₆
Group A mean	-0.074	-0.163	-0.026	-0.148	-0.127	-0.090	-0.039	+0.007
SD	0.094	0.100	0.126	0.149	0.150	0.144	0.108	0.103
Group B mean	-0.039	-0.068	+0.032	-0.021	-0.025	-0.036	-0.036	-0.004
SD	0.047	0.049	0.096	0.105	0.118	0.087	0.044	0.035
Total mean	-0.070	-0.150	-0.018	-0.131	-0.113	-0.083	-0.038	+0.006
SD	0.090	0.100	0.124	0.151	0.150	0.139	0.102	0.097

aVL (0.05 mV or more) was the sole ECG marker of the inferior location of the AMI in the early stage (Figs 1 and 2). In five of them (4.7%) ST depression in aVL was ≥ 0.01 mV.

Discussion

Approximately 10% of patients admitted for AMI do not receive thrombolytic therapy because of equivocal admission ECG; this is particularly true for those with inferior wall infarction^[1]. Huey and colleagues^[9] found that 52% and 29% of patients with AMI resulting from circumflex or right coronary artery occlusion respectively do not have ST elevation. Of our 107 patients with a first AMI involving the inferior wall, 13% did not have ST elevation of 0.1 mV or more in one of the inferior leads on admission.

The significance of ST depression in the precordial leads in inferior AMI has been extensively studied, but with conflicting results^[10,11]; however, the importance of the reciprocal changes, particularly in aVL, for the early diagnosis of inferior AMI, especially in those without diagnostic ST elevation in the inferior leads has not been stressed.

'Reciprocal changes' in the precordial leads are influenced by many factors including the size^[12-17], the

exact location of the infarct and the presence of left anterior descending coronary artery disease^[14,18,19] or three vessel disease^[18,20-22].

The precordial leads are facing the anterior and apical regions of the left ventricle and thus detect reciprocal changes mainly from the posterior wall. This explains why reciprocal changes in the precordial leads are more dependent on the exact location of the inferior AMI. It was suggested that lateral left ventricular involvement causes more pronounced ST depression in the precordial leads^[8,23,24], while right ventricle involvement causes attenuation of the precordial ST depression, especially in V₁-V₂^[24,25], or even precordial ST elevation^[26]. By contrast, lead aVL faces the superior or high lateral part of the left ventricle^[27] and thus is the only lead that is truly reciprocal to the inferior wall. aVL is the only lead that faces the infarct vector which is directed in a superior direction perpendicular to the horizontal plane^[27] and therefore it should be less influenced by involvement of the right ventricle or by infarct extension to the posterior segment.

Most of our patients with inferior AMI had transient ST segment depression in aVL on the ECG recorded during chest pain (Fig. 3). Only three patients showed no ST depression in aVL on the admission ECG. In eight patients (7.5%) ST depression in aVL was the sole ECG marker of the inferior location of the AMI in the early

stage (Figs 1 and 2). In five of them (4.7%) ST depression in aVL was ≥ 0.1 mV.

When using the criterion of ST elevation ≥ 0.1 mV in II, III and aVF^[3,4,6,7], the sensitivity of the admission ECG for the diagnosis of inferior AMI was 60.7%. When using the criterion of ST elevation ≥ 0.3 mV in the sum of II, III and aVF^[5] the sensitivity was 71.0%. Use of significant (≥ 0.1 mV) ST depression in aVL, in addition to the established ECG signs of inferior AMI, increased the sensitivity of ECG detection of inferior AMI to 77.6%. The use of mild (≥ 0.05 mV) ST depression in aVL increased it further to 97.2%. The specificity of ST depression in aVL was beyond the prospects of our study and merits further investigation.

We conclude that ST depression in aVL is a sensitive ECG sign of evolving inferior AMI and it sometimes precedes other electrocardiographic manifestations compatible with inferior AMI such as ST elevation, T wave inversion and new Q wave appearance in the corresponding inferior leads.

Conclusions

In many cases the reciprocal changes are the only electrocardiographic clue for early diagnosis and localization of evolving myocardial infarction^[8]. We have demonstrated that in almost all cases of inferior AMI there is transient ST depression in aVL during the early stage. In 7.5% of the patients, ST depression in aVL is the only early ECG clue to the diagnosis of inferior AMI.

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