ELSEVIER

International Journal of Cardiology 46 (1994) 37–47

International Journal of CARDIOLOGY

Isolated mid-anterior myocardial infarction: a special electrocardiographic sub-type of acute myocardial infarction consisting of ST-elevation in non-consecutive leads and two different morphologic types of ST-depression

Samuel Sclarovsky*[a], Yochai Birnbaum[a], Alejandro Solodky[a], Nili Zafrir[b], Mordechai Wurzel[a], Eldad Rechavia[a]

[a]*Department of Cardiology, Beilinson Medical Center, Petah Tiqva, Israel 49 100*
[b]*Nuclear Cardiology Unit, Sackler School of Medicine, Tel Aviv University, Tel Aviv, Israel 49 100*

Received 31 January 1994; revision accepted 12 April 1994

Abstract

We describe eight patients with a distinct electrocardiographic pattern of anterior wall myocardial infarction characterized by three main features: (1) a pattern of 'transmural ischemia' (ST-elevation with positive T-wave) in non-consecutive leads: aVL and V_2, and two different types of ST-depression; (2) a pattern of 'true reciprocal changes' (ST-depression and negative T-wave) in III and aVF; (3) a pattern of 'sub-endocardial ischemia' (ST-depression with positive T-wave) in V_{4-5}, while ST in V_3 was either isoelectric or depressed. We characterize the electrocardiographic features and correlate them with the echocardiographic, radionuclide, and angiographic data. All patients admitted to the coronary care unit from January 1990 to April 1992 with evolving acute myocardial infarction were evaluated prospectively. Patients whose admission electrocardiogram met the description above were included. The electrocardiographic evolution, echocardiographic, Technetium MIBI tomography, and coronary angiography are described. Of 471 patients with acute anterior wall myocardial infarction, admitted to the coronary care unit during the study period, eight patients met the inclusion criteria (1.7% of acute anterior wall myocardial infarction). Echocardiographic studies revealed mid-anterior hypokinesis in two patients, anterior and apical hypokinesis in one, and no wall motion abnormality in four patients. Technetium MIBI tomography, done in five patients, was consistent with mid-anterior or mid-anterolateral infarction without involvement of the septum or apex. Coronary angiography, performed in seven patients, demonstrated significant obstruction of the first diagonal branch in all of the patients. In four patients, the diagonal occlusion was the only significant coronary lesion in the left coronary artery. *Conclusion:* Most of the anterior myocardial infarctions also involve the septal and apical regions. Anterior wall myocardial infarctions limited to the mid-anterior or mid-anterolateral wall, without apical or septal wall involvement are relatively rare. This study describes a special electrocardiographic form of anterior wall acute myocardial infarction. This distinct electrocardiographic pattern represents true mid-anterior wall myocardial infarction, caused by occlusion of a first diagonal branch of the left anterior descending coronary artery. The septal and apical regions are not involved because the blood supply via the left anterior descending artery is not interrupted.

* Corresponding author.

0167-5273/94/$07.00 © 1994 Elsevier Science Ireland Ltd. All rights reserved
SSDI 0167-5273(94)02073-R

Keywords: Anterior wall acute myocardial infarction; Coronary angiography; Echocardiography; Electrocardiography; First diagonal branch; Left anterior descending coronary artery; Technetium MIBI tomography

1. Introduction

The electrocardiogram (ECG) is quite reliable in detecting anterior wall myocardial infarction [1–6] or infarction caused by left anterior descending coronary artery obstruction [7,8]. Based on the ECG, several sub-types of anterior wall myocardial infarction are recognized (anteroseptal, anterolateral, apical, etc.) [9]; however, correlation of the various ECG sub-types with the exact anatomical location of the infarct as determined by autopsy is poor [4,5,9]. The left anterior descending artery supplies the anterior and apical regions of the left ventricle, portions of the interventricular septum, anterolateral wall, papillary muscle and, in some patients, the inferoapical region [10]. Occlusion of the left anterior descending artery commonly results in infarcts involving the apical and/or septal region [4]. Infarcts limited to the basilar area of the anterior free wall of the left ventricle, without involvement of the apical region, are less frequent.

Despite widespread clinical concern, most published studies relating ECG data to autopsy or coronary angiographic findings provide no information regarding infarcts in which the culprit vessels are branches of the major coronary arteries [1–8].

In this study we describe eight patients with a distinct ECG pattern representing a special subtype of anterior wall myocardial infarction, which has not been well characterized before now. We describe the ECG findings during the acute phase and their evolution, and compare them to the echocardiographic, radionuclide imaging and coronary angiographic features of these patients.

2. Methods

2.1. Patients

All patients admitted to the coronary care unit from January 1990 to April 1992 with evolving acute myocardial infarction were evaluated prospectively. Patients who met the following criteria were included in the study: (1) severe chest pain of more than 30 min duration; (2) transient elevation of serum creatine kinase; (3) ECG during the acute stages showing ST-segment elevation with positive peaked T-wave in aVL and V_2, ST-depression with negative T-wave in the inferior leads, and ST-depression with positive T-wave in the precordial V_{3-5} leads; (4) no history or ECG evidence of previous myocardial infarction.

2.2. Electrocardiographic evaluation

All patients underwent continuous three-lead ECG recording for 2 to 3 h after admission, with repeated standard 12-lead ECG every 5 to 10 min. Thereafter, standard 12-lead ECG was recorded every 24 h and during every episode of chest pain. ST-segment deviations from the isoelectric line, as was determined by a line that was drawn between subsequent TP-segments, were measured at 0.06 s after the J-point. The T-wave was measured at the maximal deflection (positive or negative) more than 120 ms after the J-point. The ST-segment and the T-wave were considered positive or negative if they were ≥ 0.1 mV above or ≤ -0.1 mV below the isoelectric line, respectively. ST-segments or T-waves > -0.1 mV and < 0.1 mV were considered isoelectric.

2.3. Echocardiographic evaluation

Technically satisfactory 2-dimensional echocardiography recordings were obtained within the first 24 h of admission and on the third to seventh day in seven patients. A Diasonics CV100 echocardiograph equipped with a 2.5 MHz transducer was used. Patients were examined in the supine and left lateral decubitus position, and the heart was imaged through several acoustic windows (longitudinal and cross-sectional parasternal, apical four- and two-chamber, and subxiphoid four-chamber). All examinations were recorded on videotape and

analyzed by two independent observers. Wall motion score was determined using a 14-segment model as described by Oh et al. [11].

2.4. Radionuclide evaluation

Five patients underwent technetium MIBI radionuclide imaging within 24 h of admission. Each patient was injected with 25 mCi technetium MIBI intravenously. Ninety minutes later myocardial perfusion tomography imaging was performed by a gamma camera (SP-6 Elscint Haifa Ltd.). Data acquisition was carried out over an 180° angle starting from the right anterior oblique 45° position and ending at the left posterior oblique, with a zoom of 1.33. Thirty views were recorded into a 64 × 64 matrix with an acquisition time of 25 s per view. The data were filtered back projection with a Butterworth filter. Slices (thickness, 1 cm) were reconstructed. Three sets of slices were obtained in short, long and horizontal axes. In each patient four segments of the left ventricle were examined in short axis view (septal, anterior, inferior, posterolateral) and two segments in long axis view (apex and anterolateral). The segments were visualized subjectively for filling defects.

2.5. Coronary angiographic evaluation

Selective coronary cineangiography was performed within 10 days of admission in seven patients by the Judkins technique. Cine recordings were made of each vessel in multiple projections (right and left anterior oblique, cranial and caudal projections) on 16 mm film. Each cineangiography was analyzed by two independent observers.

2.6. Left cineventriculographic evaluation

Cineventriculographies in the right anterior oblique and left lateral projections were performed in seven patients. Each cineventriculography was analyzed by two independent observers. The anterolateral, apical, diaphragmatic, septal, posterobasal and posterolateral segments were examined for evidence of regional wall motion abnormality.

3. Results

Of 471 patients with acute anterior wall myocardial infarction admitted to the coronary care unit during the study period, eight patients met the inclusion criteria (1.7% of acute anterior wall myocardial infarction) (Table 1). These included five males and three females, aged 58 ± 11 years (38–71 years). Two patients, Nos. 1 and 8 received intravenous thrombolytic therapy with rtPA. Peak creatine kinase levels were 806 ± 361 U/l (455–1464 U/l) (normal range, 24–203 U/l). All patients were in Killip class I. None of the patients died or developed heart failure, shock, arrhythmia or re-ischemia during the index hospitalization.

3.1. Electrocardiographic findings

Fig. 1, and Tables 2 and 3 show all patients were in sinus rhythm on admission and on the following

Table 1
Clinical, echocardiographic, and radionuclide perfusion scan of the patients

Patient No.	Age (years)	Sex	Peak CK (U/l)	Echocardiogram	MIBI
1	60	F	1023	—	—
2	47	F	1464	normal	A + AL
3	59	M	1111	A + apical hypokinesis	A + AL
4	60	M	455	normal	A + AL
5	71	F	624	normal	—
6	67	M	759	A hypokinesis	A
7	59	M	515	A hypokinesis	—
8	38	M	494	normal	A + small septal

A, anterior; AL, anterolateral; CK, creatine kinase; F, female; M, male.

ECG recordings. No-one displayed ECG evidence of bundle branch block, intraventricular conduction defect, or left ventricular hypertrophy.

The admission ECG of all the patients demonstrated ST-segment elevation (≥ 0.1 mV) with positive T-wave in aVL and V_2, ST-segment depression with negative T-wave in III and aVF, and ST-segment depression with positive T-wave in V_4 and V_5 (Fig. 1, Table 2). In six patients there was ST-segment elevation with positive T-wave in I, and in four patients ST-segment depression with positive T-wave in V_3. In the other four patients the ST-segment was isoelectric in V_3. ST-segment depression in II was found in all of the patients, two with negative and six with positive T-wave.

The ECG recording on the following days revealed resolution of the ST-segment elevation in leads I, aVL and V_2 in 3/6, 2/8, and 3/8 patients, respectively, and inversion of the T-wave in five, eight, and three patients respectively. New pathological Q-wave appeared in five patients (in aVL in four patients, and in V_2 in three). Shortening of the R-wave amplitude in V_{2-4} was demonstrated in four patients. However, in two patients no pathological Q-wave or alteration in the R-wave amplitude was noted. The ECG evolution in III

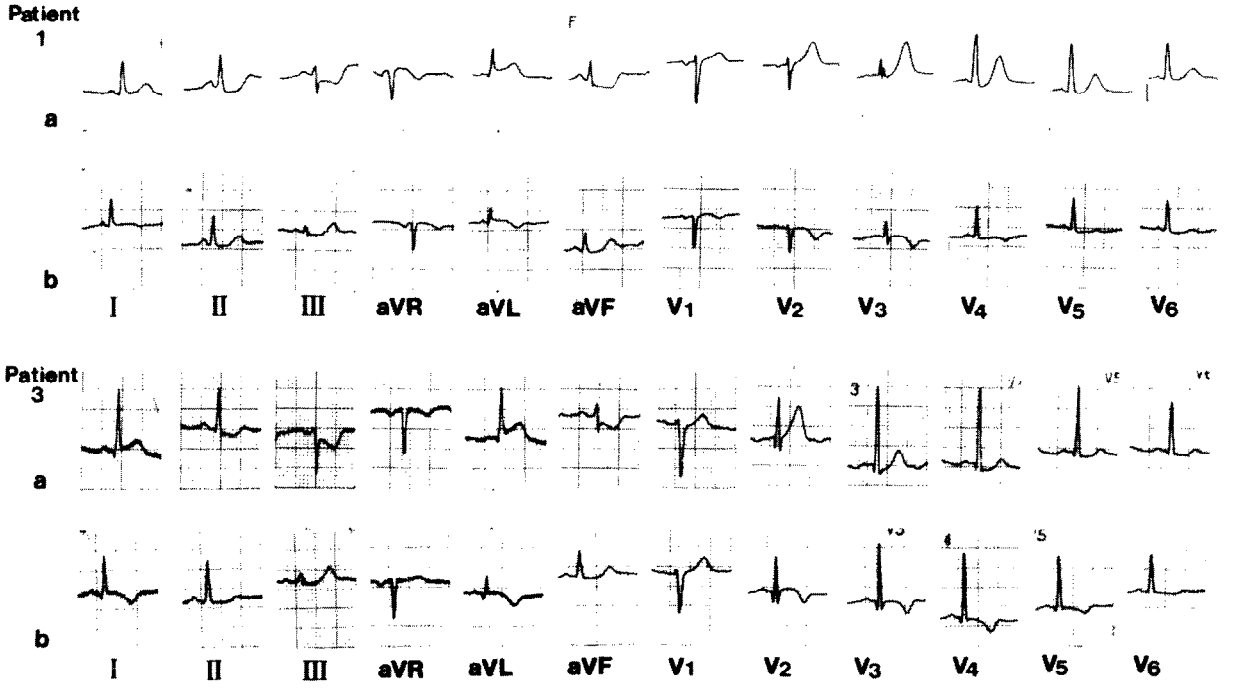

Fig. 1. Electrocardiograms of the acute and fully evolved stages of the myocardial infarction. Patient 1 (a) electrocardiogram on admission showing ST-elevation in aVL and V_2, ST-depression with negative T-wave in II, III and aVF, and ST-depression with positive T-wave in V_4 and V_5. (b) electrocardiogram of the fully evolved stage (third day) showing inversion of the T-wave in I and aVL, while the ST in the inferior leads become positive. There is resolution of the ST-depression in II, III and aVF, and inversion of the T-wave in V_{1-4}. There is deviation of the QRS axis to the right. Patient 3 (a) electrocardiogram on admission showing ST-elevation in I, aVL and V_2, ST-depression with negative T-wave in III and aVF, and ST-depression with positive T-wave in V_3 and V_4. (b) electrocardiogram of the fully evolved stage (third day) showing inversion of the T-wave in I and aVL, resolution of the ST-depression, while the T-wave become positive in III and aVF, and inversion of the T-wave in V_{2-5}. There is deviation of the QRS axis to the right.

Table 2
ECG data of the acute stage

Lead	I		II		III		aVR		aVL		aVF		V_1		V_2		V_3		V_4		V_5		V_6	
	ST	T	ST	T	ST	T	ST	T	ST	T	ST	T	ST	T	ST	T	ST	T	ST	T	ST	T	ST	T
Positive	6	8	0	6	0	0	4	0	8	8	0	0	2	4	8	8	0	8	0	8	0	8	0	8
Isoelectric	2	—	0	—	0	—	4	—	0	—	0	—	6	—	0	—	4	—	0	—	0	—	5	—
Negative	0	0	8	2	8	8	0	8	0	0	8	8	0	4	0	0	4	0	8	0	8	0	3	0
Total	8	8	8	8	8	8	8	8	8	8	8	8	8	8	8	8	8	8	8	8	8	8	8	8

The table presents number of patients in each category.

and aVF consisted of resolution of the ST-depression in seven and two patients respectively, and of positivation of the T-wave in all eight patients. These changes occurred simultaneously with the resolution of the changes in leads I, aVL, and V_2. ST-segment depression in V_4 and V_5 resolved in all patients. The T-wave became inverted in V_3 and V_4 in two and three patients respectively, while in the others the T-wave amplitude was shortened (Fig. 1, Table 3).

3.2. Echocardiographic findings

Seven patients underwent echocardiographic examination (Table 1). In four patients no wall motion abnormality was detected, while in two patients there was hypokinesis of the mid-anterior area (segment 7 according to Oh et al. [11]), and in one anterior and apical hypokinesis (segments 7 and 12).

3.3. Radionuclide findings

Five patients underwent technetium MIBI radionuclide study (Fig. 2). A perfusion defect in the anterior segment was found in all five, in three it extended into the anterolateral segment. In one patient, a small septal perfusion defect was observed. No apical, inferior or posterior involvement was noted.

3.4. Coronary angiographic findings

Seven patients underwent coronary angiography (Table 4, Fig. 3). Significant luminal narrowing ($\geq 75\%$) in the left anterior descending artery, circumflex, obtuse marginal, and the right coronary artery was found in two, one, one, and two patients. respectively. However, significant obstruction of the first diagonal branch was found in all patients. In four patients there was a com-

Table 3
ECG data of the fully evolved stage

Lead	I		II		III		aVR		aVL		aVF		V_1		V_2		V_3		V_4		V_5		V_6	
	ST	T	ST	T	ST	T	ST	T	ST	T	ST	T	ST	T	ST	T	ST	T	ST	T	ST	T	ST	T
Positive	2	3	0	8	0	8	6	1	6	0	0	8	0	6	5	5	2	6	0	5	0	6	0	6
Isoelectric	6	—	7	—	7	—	2	—	2	—	6	—	8	—	3	—	6	—	8	—	8	—	8	—
Negative	0	5	1	0	1	0	0	7	0	8	2	0	0	2	0	3	0	2	0	3	0	2	0	2
Total	8	8	8	8	8	8	8	8	8	8	8	8	8	8	8	8	8	8	8	8	8	8	8	8

The table presents number of patients in each category.

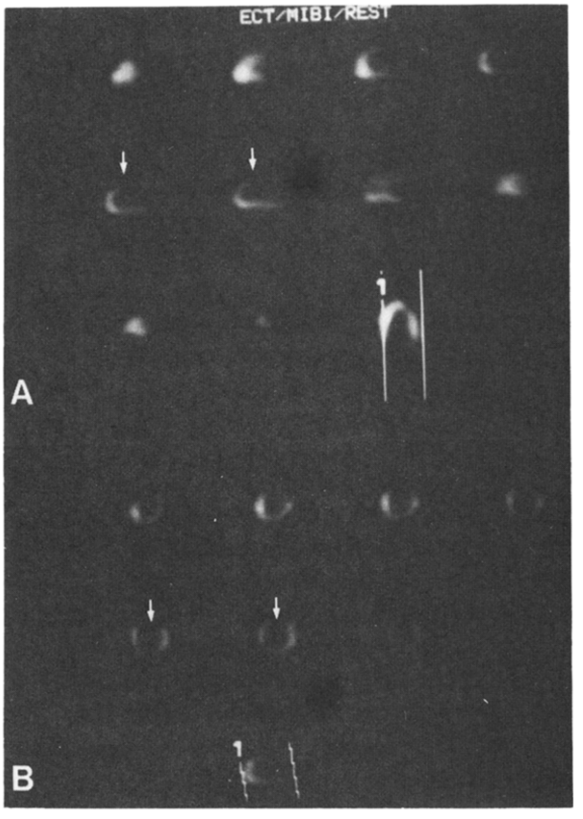

Fig. 2. Technetium MIBI tomography of patient No. 4 demonstrating a filling defect in the mid-anterior and anterolateral segments in long axis (A) and short axis (B). The septum and the apical region are normally perfused.

plete obstruction of the first diagonal branch, while in the other three, a severe complex lesion with evidence of residual thrombus was found. In four patients, the only significant lesion in the left coronary artery was in the first diagonal branch.

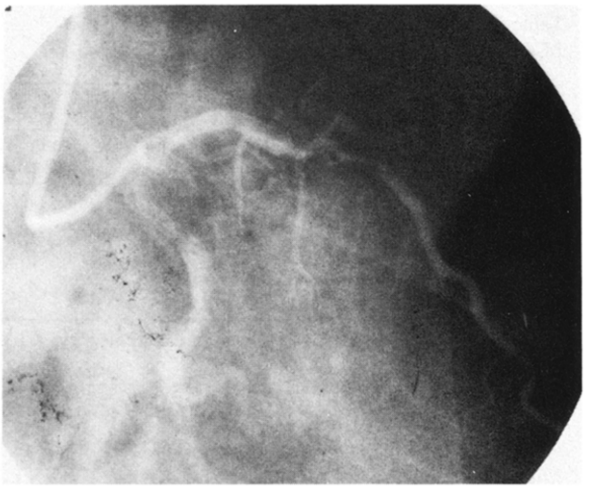

Fig. 3. Selective left coronary cineangiography of patient No. 3 in the right anterior oblique projection. There is complete obstruction of the first diagonal branch and an irregular lesion with 60% luminal narrowing of the left anterior descending coronary artery distal to the origin of the diagonal branch.

3.5. *Left ventricular cineangiography findings*

Seven patients underwent left ventriculography (Table 4). The left ventricular ejection fraction was 61 ± 12% (52–72%). Anterolateral wall motion abnormality was noted in five patients (four with hypokinesis and one with akinesis). Mild hypokinesis of the apex was found in one patient, while in two patients, left ventricular wall motion was entirely normal.

Table 4
Coronary and left ventricle cineangiography

Patient	LAD	First diagonal	Cx	Cx-M1	Cx-M2	RCA	EF	Wall motion
1	95	100	60	0	0	0	0.62	AL hypokinesis
2	50	100	0	0	0	75	0.70	normal
3	60	100	0	0	0	0	0.52	AL akinesis
5	45	90	40	50	0	50	0.72	normal
6	30	95	80	70	0	90	0.56	AL hypokinesis
7	55	99	60	0	0	0	0.41	AL + apical hypokinesis
8	75	100	0	99	60	40	0.71	AL hypokinesis

Numbers represent percentage of luminal narrowing. AL, anterolateral segment; EF, left ventricular ejection fraction.

4. Discussion

Eight patients with a distinct sub-type of anterior wall myocardial infarction are described.

4.1. Electrocardiographic examination

The ECG pattern in the acute stage of this subtype of anterior wall myocardial infarction consists of two main features: 1. A pattern of transmural ischemia in leads that are not considered consecutive; 2. Simultaneous appearance of two types of ST-segment depression in different leads, ST-depression with positive T-wave in the precordial leads, and ST-depression with negative T-wave in the inferior leads (III and aVF). This ECG pattern, although mentioned before in the literature [12], has not been fully characterized and correlated with anatomic or angiographic data. Moreover, in a recent work this pattern was considered non-diagnostic [13]. Schamroth [12] interpreted this pattern as an anteroseptal and high lateral myocardial infarction. This implies infarction of two separated segments supplied by two different branches of the left coronary system.

In all of our patients ST-elevation was found in V_2 and aVL, while in six of them ST-elevation was detected also in lead I. The ST-segments in the anterior and apical leads V_{3-6} were either isoelectric or negative. Lead I faces the lateral aspect of the left ventricle and thus records the transverse (lead X) component of the cardiac vector. Lead aVL faces the basal portion of the anterolateral free wall of the left ventricle [9,14]. Lead V_2 faces the anteroseptal area [9]. ST-elevation in I and aVL usually represents lateral or anterolateral wall transmural ischemia, while ST-elevation in V_2 is usually found in anteroseptal wall ischemia [9] or even in right ventricle infarction [15]. However, no evidence of septal or apical involvememt was found by the radionuclide, echocardiographic and left ventriculographic examinations. Thus, ST-elevation in V_2, without ST-elevation in other precordial leads, such as V_1 or V_3, probably represents anterior and not septal wall involvement.

Two distinct forms of ST-depression were present simultaneously during the acute stage [16,17]. While ST-depression with negative T-wave was found in the inferior leads III and aVF, ST-depression with positive T-wave appeared in the precordial leads, apart from V_2 (Fig. 1, Tables 2,3). These two types evolve by different electrophysiological mechanisms and have a different time course of evolution during the later stages of the infarction. ST-segment depression with negative T-waves in leads III and aVF was not accompanied by either a perfusion defect on the technetium MIBI scan or wall motion abnormality in the posterior or inferior regions. Therefore, these changes represent true reciprocal changes and may be considered a 'mirror image' of the transmural ischemia affecting the mid-anterior region (ST-elevation with positive T-wave in I, aVL, and V_2). The ECG evolution in the inferior leads III and aVF occurred simultaneously with the decline of the magnitude of ST-segment elevation and the inversion of the T-wave in aVL and V_2, thus, further supporting a reciprocal etiology.

The second type of ST-depression (with positive T-wave), found during the acute stage in V_4 and in V_5, while aVL and V_2 still manifest ST-elevation and positive T-wave, has a different electrophysiological explanation. In this type of ST-elevation, the vectors of the ST-segment and the T-wave are shifted in opposite directions [17]. This has been ascribed to a sub-endocardial ischemic and injury pattern [9,16]. Ekmekci et al. [18] reported that ligation of a branch of a left anterior descending artery in an open chest dog induces almost immediate ST-elevation in the central part of the ischemic area, however, ST-segment depression appears later in the border zones surrounding the infarction. In these zones only partial or sub-endocardial ischemia is present. Janse et al. [19] demonstrated that occlusion of the left anterior descending artery in open chest pigs produces ST-elevation with positive T-wave in epicardial leads orientated to the central ischemic area. However, ST-depression and positive T-wave were detected over the border zone. This border zone was characterized by intermediate levels of lactate, creatine phosphate and adenosine triphosphate (ATP). The ECG evolution of this pattern consisted of restoration of the ST-depression and inversion of the T-wave, which is compatible with a non-Q-wave infarction. This is probably the only clinical example of the simultaneous appearance of

the two types of ST-segment depression in different leads.

4.2. Echocardiographic examination

Two-dimensional echocardiography is considered to be more sensitive and accurate than ECG for the early detection of acute myocardial infarction, especially in patients with first transmural myocardial infarction [11,20–25]. It was stated that 'when asynergy was absent, it was unlikely that an acute myocardial infarction was missed' [11,24]. But others found that there is a myocardial infarct size threshold for 2-dimensional echocardiographic detection, even for transmural infarctions [26–28].

In 3/7 patients regional asynergy was detected. In two patients, Nos. 6 and 7, hypokinesis was limited to the mid-anterior segment, while in another, No. 3, the mid-anterior and the apical segments were hypokinetic. In the latter patient, no apical wall motion abnormality was detected by angiographic left ventriculography (Table 4), and the technetium MIBI perfusion defect was limited to the mid-anterior and anterolateral regions, without apical involvement (Table 1). It appears that the anteroapical hypokinesis in this patient did not represent myocardial ischemia, but a tethering phenomenon [29–32].

In four of our patients no wall motion abnormality was detected. In this specific type of anterior wall myocardial infarction, the sensitivity of 2-dimensional echocardiography seems to be low in comparison with an ECG. A sensitivity of only 43% is even lower than that reported for detection of non-Q-wave myocardial infarction (66%) [22]. The low ability to detect wall motion abnormality in this particular type of anterior wall myocardial infarction is explained by the relatively small size of the infarction. Another explanation is that the infarction is surrounded by normally contracted areas, the apex and the basal segments of the free wall of the left ventricle, and by the septal region. The normally functioning segments help 'pull' the relatively small infarcted area, thus obscuring the infarction.

4.3. Mid-anterior location

Infarcts that are limited to the basilar or middle-portion of the anterior or anterolateral free wall of the left ventricle, without apical or septal involvement, are quite rare [4,29]. None of the 50 patients described by Sullivan et al. [5], had isolated anterior wall infarction without either septal or apical wall involvement. All of our patients, except No. 5, had at least one positive study relating the infarction to the mid-anterior or mid-anterolateral region (echocardiography, MIBI scan, or angiographic left ventriculography). In patient No. 5 no motion abnormality was detected by echocardiography or angiographic ventriculography, and a technetium MIBI scan was not preformed. Only one patient, No. 8, had a small septal perfusion defect on the technetium MIBI scan (which may be related to a previous infarction), and in none of the five patients was apical involvement found. In only one patient, No. 7, apical hypokinesis was detected by angiographic ventriculography, but no apical asynergy was detected by echocardiographic examination.

The location of the anterior wall myocardial infarction in our patients is best explained by obstruction of a diagonal branch supplying the anterolateral aspect of the left ventricle, without reaching the apex. There is a wide variability in the number and size of the diagonal branches. More than 90% have one to three such branches. Less than 1% have no diagonal branches [33]. Total obstruction of a diagonal branch is frequently overlooked. Thus, if none are seen, the angiographer should consider the possibility that a diagonal branch might have originally been present but become totally occluded at its origin from the left anterior descending artery [10]. The clinical, ECG, angiographic and anatomic differences between infarcts caused by a diagonal branch occlusion and those related to left anterior descending artery obstruction have not been delineated in the literature.

The ECG manifestations of this sub-type of anterior wall myocardial infarction are best explained by a 'wedge-shape' infarction located in the mid-anterior or mid-anterolateral region. The central area of transmural ischemia (ST-segment elevation in leads I, aVL, and V_2) is relatively narrow, as reflected by the sharp difference between the ST-elevation in V_2 and the isoelectric or negative ST-segment in the adjacent precordial leads V_1, V_{3-5}. The central zone is surrounded by a

relatively wide 'border zone', as reflected by the ECG pattern of 'sub-endocardial ischemia' (ST-segment depression with positive T-wave) in V_{3-5}. However, in most of the clinical anterior wall myocardial infarctions in humans, the border zone is not expressed by the routine 12-lead ECG. Moreover, the area of this border zone in experimental animals with normal coronary arteries is narrow [34]. Perhaps occlusion of a secondary branch of a coronary artery, in contrast to obstruction of a major artery, may induce a relatively wider 'border zone', especially when the collateral flow is augmented by a pre-existing luminal narrowing of the culprit artery. Another explanation could be, that in a larger anterior wall myocardial infarction, the solid angle arising from the transmural infarction obscures the expression of the sub-endocardial ischemia in the precordial leads. However, in the mid-anterior infarction, there is a relatively small central transmural infarction surrounded by normal basal and apical segments. This factor, combined with the special geometrical orientation of the infarction relative to the precordial leads, may lead to a relatively small or even negative solid angle [35], enabling expression of the 'border zone' sub-endocardial ischemia in the precordial leads V_{3-5}.

The obtuse marginal branches of the circumflex artery supply the lateral zone of the left ventricle. There is overlap of the diagonal and obtuse marginal branches [10]. Frequently, acute infarctions that are caused by obstruction of the circumflex artery are not accompanied by typical ST-segment elevation [36]; however, in cases with ST-segment elevation, it is usually found in the inferior leads (II, III, and aVF) and in V_5, V_6. ST-elevation in V_2 and ST-depression in the inferior leads are uncommon [7,37].

Because of the high variability in the number and course of the diagonal branches [33], it is possible that obstruction of a diagonal branch may lead to several different electrocardiographic manifestations, according to the size and location of the vascular bed distal to the obstruction, and the collateral flow from adjacent left anterior descending, other diagonal branches, and the marginal branches of the circumflex artery. However, we describe a specific electrocardiographic sub-type of acute anterior wall myocardial infarction that is related to an obstruction of the first diagonal branch. A prospective trial of electrocardiographic recording during angioplasty is needed to clarify the various electrocardiographic manifestations of myocardial ischemia due to first diagonal branch obstruction.

5. Conclusions

We describe a special form of anterior wall myocardial infarction, characterized in the acute stage by a pattern of 'transmural ischemia' (ST-elevation with positive T-wave) in aVL and V_2, a pattern of 'sub-endocardial ischemia' (ST-depression and positive T-wave) in V_{4-5}, and a pattern of 'true reciprocal changes' (ST-depression and negative T-wave) in the inferior leads. This ECG pattern is correlated with 2-dimensional echocardiographic, radionuclide, and angiographic evidence of mid-anterior wall infarction without septal or apical involvement, best explained by obstruction of a diagonal branch of the left anterior descending coronary artery. The ability to recognize this pattern may enable the clinician to identify patients with relatively small anterior wall myocardial infarction and a relatively favorable hospital course, and to diagnose the culprit artery with a simple non-invasive tool, the ECG.

References

1 Myers GB, Klein HA, Stofer BE. I. Correlation of electrocardiographic and pathologic findings in anteroseptal infarction. Am Heart J 1948; 36: 535–575.

2 Myers GB, Klein HA, Hiratzka T. II. Correlation of electrocardiographic and pathologic findings in large anterolateral infarcts. Am Heart J 1948; 36: 838–881.

3 Myers GB, Klein HA, Hiratzka T. III. Correlation of electrocardiographic and pathologic findings in anteroposterior infarction. Am Heart J 1949; 37: 205–236.

4 Roberts WC, Gardin JM. Location of myocardial infarcts: a confusion of terms and definitions. Am J Cardiol 1978; 42: 868–872.

5 Sullivan W, Vlodover Z, Tuna N, Long L, Edwards JE. Correlation of electrocardiographic and pathologic findings in healed myocardial infarction. Am J Cardiol 1978; 42: 724–732.

6 Wilkinson RS, Schaeffer JA, Abildskov JA. Electrocardiographic and pathologic features of myocardial infarction in man: a correlative study. Am J Cardiol 1963; 11: 24–35.

7 Blanke H, Cohen M, Schlueter GU, Karsch KR, Rentrop KP. Electrocardiographic and coronary arteriographic correlations during acute myocardial infarction. Am J Cardiol 1984; 54: 249–255.

8 Williams RA, Cohen PF, Vokonas PS, Young E, Herman MV, Gorlin R. Electrocardiographic, arteriographic and ventriculographic correlations in transmural myocardial infarction. Am J Cardiol 1973; 31: 595–599.
9 Schamroth L. Anterior wall myocardial infarction. In: Schamroth L, editor. The electrocardiology of coronary artery disease, 2nd ed. London: Blackwell Scientific Publications, 1984; 61–70.
10 Levin DC, Gardiner GA. Coronary arteriography. In: Braunwald E, editor. Heart disease, 4th ed. Philadelphia: W B Saunders Company, 1992; 239–241.
11 Oh JK, Miller FA, Shub C, Reeder GS. Evaluation of acute chest pain syndromes by 2-dimensional echocardiography: its potential application in the selection of patients for acute reperfusion therapy. Mayo Clin Proc 1987; 62: 59–66.
12 Schamroth L. Case study 5: anteroseptal and high lateral myocardial infarction: septolateral infarction. In: Schamroth L, editor. The electrocardiology of coronary artery disease 2nd ed. London: Blackwell Scientific Publications, 1984; 224.
13 Eisenberg JD. Thrombolytic treatment of an acute anterior myocardial infarction based upon echocardiography and in the absence of electrocardiographic criteria. Echocardiography 1992; 9: 357–362.
14 Cooksey JD, Dunn M, Massie E. Anterior myocardial infarction. In: Cooksey JD, Dunn M, Massie E, editors. Clinical vectrocardiography and electrocardiography 2nd ed. Chicago: Year Book Publishers Inc., 1977; 378–399.
15 Geft IL, Shah PK, Rodriguez L, et al. ST-elevation in leads V_{1-5} may be caused by right coronary artery occlusion and acute right ventricular infarction. Am J Cardiol 1984; 53: 991–996.
16 Sclarovsky S, Rechavia E, Strasberg B, et al. Unstable angina: ST-segment depression with positive versus negative T-wave deflections—clinical course, ECG evolution, and angiographic correlation. Am Heart J 1988; 116: 933–941.
17 Sclarovsky S, Mager A, Kusniec J, et al. Electrocardiographic classification of acute myocardial ischemia. Isr J Med Sci 1990; 26: 525–533.
18 Ekmekci A, Toyoshima H, Kwoczynski JK, Nagaya T, Prinzmetal M. Angina pectoris. IV. Clinical and experimental differences between ischemia with ST-elevation and ischemia with ST-depression. Am J Cardiol 1961; 7: 412–426.
19 Janse MJ, Cinca J, Morena H, et al. The border zone in myocardial ischemia: an electrophysiological, metabolic, and histochemical correlation in the pig heart. Circ Res 1979; 44: 576–588.
20 Horowitz RS, Morganroth J, Parrotto C, Chen CC, Soffer J, Pauletto FJ. Immediate diagnosis of acute myocardial infarction by 2-dimensional echocardiography. Circulation 1982; 65: 323–329.
21 Oh JK, Shub C, Miller-Jr FA, Evans WE, Tajik AJ. Role of 2-dimensional echocardiography in the emergency room. Echocardiography 1985; 2: 217–226.
22 Arvan S, Varat MA. 2-dimensional echocardiography versus surface electrocardiography for the diagnosis of acute non-Q-wave myocardial infarction. Am Heart J 1985; 110: 44–49.
23 Loh IK, Charuzi Y, Beeder C, Marshall LA, Ginsburg JH. Early diagnosis of non-transmural myocardial infarction by 2-dimensional echocardiography. Am Heart J 1982; 104: 963–968.
24 Peels CH, Visser CA, Kupper AJF, Visser FC, Roos JP. Usefulness of 2-dimensional echocardiography for immediate detection of myocardial ischemia in the emergency room. Am J Cardiol 1990; 65: 687–691.
25 Otto CM, Stratton JR, Maynard C, Althouse R, Johannessen KA, Kennedy JW. Echocardiographic evaluation of segmental wall motion early and late after thrombolytic therapy in acute myocardial infarction: the Western Washington tissue plasminogen activator emergency room trial. Am J Cardiol 1990; 65: 132–138.
26 Lieberman AN, Weiss JL, Jugdutt BI, et al. Two-dimensional echocardiography and infarct size: relationship of regional wall motion and thickening to the extent of myocardial infarction in the dog. Circulation 1981; 63: 739–746.
27 O'Boyle JE, Parisi AF, Nieminen M, Kloner R, Khuri S. Quantitative detection of regional left ventricular contraction abnormalities by 2-dimensional echocardiography. Comparison of myocardial thickening and thinning and endocardial motion in a canine model. Am J Cardiol 1983; 51: 1732–1738.
28 Pandian NG, Skorton DJ, Collins SM, et al. Myocardial infarct size threshold for 2-dimensional echocardiographic detection: sensitivity of systolic wall thickening and endocardial motion abnormalities in small versus large infarcts. Am J Cardiol 1985; 55: 551–555.
29 Stamm RB, Gibson RS, Bishop HL, Carabello BA, Beller GA, Martin RP. Echocardiographic detection of infarct-localized asynergy and remote asynergy during acute myocardial infarction: correlation with the extent of angiographic coronary disease. Circulation 1983; 67: 233–243.
30 Heger J, Weyman AE, Wann LS, Dillon JC, Feigenbaum H. Cross-sectional echocardiography in acute myocardial infarction: detection and localization of regional left ventricular asynergy. Circulation 1979; 60: 531–538.
31 Weiss JL, Bulkley BH, Hutchins GM, Mason SJ. Two-dimensional echocardiographic recognition of myocardial injury in man: comparison with post-mortem studies. Circulation 1981; 63: 401–407.
32 Pandian NG, Koyanagi S, Skorton DJ, et al. Relations between 2-dimensional echocardiographic wall thickening abnormalities, myocardial infarct size and coronary risk area in normal and hypertrophied myocardium in dogs. Am J Cardiol 1983; 52: 1318–1325.
33 Levin DC, Harrington DP, Bettmann MA, Garnic JD, Davidoff A, Lois J. Anatomic variations of the coronary arteries supplying the anterolateral aspect of the left ventricle: possible explanation for the unexplained anterior aneurysm. Invest Radiol 1982; 17: 458–462.
34 Factor SM, Okun EM, Kirk ES. The histological lateral

border of acute canine myocardial infarction: a function of microcirculation. Circ Res 1981; 48: 640–649.

35 Holland RP, Arnsdorf MF. Solid angle theory and the electrocardiogram: physiologic and quantitative interpretations. Prog Cardiovasc Dis 1977; 19: 431–452.

36 Huey BL, Beller GA, Kaiser DL, Gibson RS. A comprehensive analysis of myocardial infarction due to left circumflex artery occlusion: comparison with infarction due to right coronary artery and left descending artery occlusion. J Am Coll Cardiol 1988; 12: 1156–1166.

37 Bairey CN, Shah PK, Lew AS, Hulse S. Electrocardiographic differentiation of occlusion of the left circumflex versus the right coronary artery as a cause of inferior acute myocardial infarction. Am J Cardiol 1987; 60: 456–459.