Journal of Cardiology 1994; 24: 357–366

Different Clinical Implications for ST Depression and T Wave Inversion in Non-Q Wave Myocardial Infarction

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Abstract

The differences between ST depression and T wave inversion in non-Q wave myocardial infarction were investigated in 42 patients with initial non-Q wave infarction, 22 patients with ST depression (ST group), and 20 patients with T wave inversion (T group). The extent of ischemic area estimated by electrocardiography and two-dimensional echocardiography, characteristic features of electrocardiographic changes, and clinical findings on admission and outcome were estimated. ST elevation preceded T wave inversion in the same leads in 80% (16/20) of the T group, and transient Q waves developed in 55% (11/20). However, neither ST elevation nor transient Q waves were observed in the ST group. Two or three ischemic segments were present in 86% (19/22) of the ST group patients, but only one ischemic segment was present in 60% (12/20) of T group patients, predominantly the anterior segment. The short-axis view of the two-dimensional echocardiogram on the level of papillary muscle showed decreased contraction in two or three of the anterior, lateral, and inferior segments of the left ventricle in 78% (14/18) of ST group patients. Only one segment with decreased contraction was present in 100% (17/17) of T group patients. Cardiac status on admission was lower in the ST than the T group : Killip class II–IV, 59% (13/22) vs 20% (4/20), $p<0.05$; mortality rate after 1 month, 41% (9/22) vs 0% (0/20), $p<0.05$. Coronary angiograms, left ventriculograms, and autopsy findings also showed extensive myocardial lesions in accordance with multivessel disease in the ST group, but localized myocardial lesion suggesting one-vessel territory in the T group. T wave inversion in non-Q wave myocardial infarction indicates a recovery phase in transient transmural ischemia and localized subendocardial infarction within the presumed one-vessel territory, while ST depression suggests the presence of extensive ischemia in the subendocardium of multivessel territory, and infarction within that region.

Key Words

transmural ischemia, subendocardial infarction, prolongation of repolarization, Q wave infarction, non-transmural infarction

INTRODUCTION

The prognostic significance and severity of non-Q wave myocardial infarction as a coronary artery disease remain controversial, particularly in comparison with Q wave infarction. Most previous studies have considered non-Q wave myocardial infarction associated with ST segment depression or T wave inversion collectively as non-Q wave infarction, non-transmural infarction, or subendocardial infarction[1–3]. The separate clinical and prognostic implications of ST depression and T wave inversion in non-Q wave infarction have been little investigated[4,5]. Any significant differences between ST depression and T wave inversion in non-Q wave infarction would be a major cause of controversy.

This study investigated the differences between ST depression and T wave inversion in patients with non-Q wave myocardial infarction based on characteristic features of electrocardiographic changes from onset, severity of coronary artery lesions, clinical findings, and outcome.

MATERIALS AND METHODS

Patient selection

The study population was selected from 240 con-

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Received for publication January 28, 1994; accepted May 23, 1994 (Ref. No. 39–PS241)

Table 1 Clinical characteristics of 240 patients with acute myocardial infarction

	No. of patients (F/M)	Age (yrs)	In-hospital death
First AMI	181 (88/93)	76.8±8.4	54 (30%)
Q wave	130 (63/67)	76.6±8.4	40 (31%)
Negative T wave	20 (9/11)	76.0±10.0	0 (0)
ST depression	27 (16/11)	79.0±7.5	14 (52%)
LBBB	4 (0/4)	72.8±5.3	0 (0)
Recurrent AMI	59 (23/36)	77.0±8.5	18 (31%)
Q wave	23 (11/12)	77.7±9.1	7 (30%)
Negative T wave	4 (2/2)	67.5±5.3	0 (0)
ST depression	29 (9/20)	77.6±8.1	10 (34%)
LBBB	3 (1/2)	78.0±6.6	1 (33%)

AMI=acute myocardial infarction; LBBB=left bundle-branch block

secutive patients (129 men, 75.3±8.8 years; 111 women, 78.6±7.7 years) admitted to our coronary care unit within 24 hours of onset of acute myocardial infarction between July, 1986 and December, 1992. One hundred eighty-one cases were first myocardial infarction and 59 cases were recurrent infarction confirmed by increases in serum creatine kinase levels of twice or more beyond the upper normal limit associated with electrocardiographic changes. The patients were classified into four groups according to electrocardiographic changes : Q wave; T wave inversion without persistent Q wave development (usually accompanied by preceding ST elevation); persistent ST depression; and left bundle-branch block (**Table 1**).

The 27 patients with ST depression and 20 patients with T wave inversion, among the 181 patients with first myocardial infarction were assigned to the ST group and T group, respectively. Three of the 27 patients with ST depression died within 3 hours of admission and showed no evidence of increased cardiac enzyme levels. Another two patients were subsequently proved to have transmural lateral infarction by myocardial scintigrams and coronary angiograms. These five patients were excluded from the study. All patients in each group showed the following electrocardiographic patterns: ST group, horizontal or down-slope type ST depression of 0.1 mV or more at 80 msec from the J point; T group, isolated and symmetric T waves with negative amplitude of at least 0.5 mV without permanent Q waves (observed for less than 3 months). Representative electrocardiograms of cases of ST depression and T wave inversion are shown in **Fig. 1**.

Clinical characteristics

All 240 consecutive patients were classified by sex, age, and in-hospital mortality according to the type of electrocardiographic change. Clinical factors in the ST and T group patients were compared as follows : cardiac status on admission, serial electrocardiographic changes, extent of ischemic areas defined by electrocardiograms and two-dimensional echocardiograms, clinical outcome, and catheterization and autopsy findings.

Cardiac status

The cardiac status on admission was evaluated according to 1) Killip class, 2) use of catecholamines to treat heart failure or to sustain blood pressure, and 3) metabolic acidosis caused by circulatory failure meeting the following criteria : pH <7.3, $HCO_3^- < 15$ mEq/*l* and base excess < -5.0 mEq/*l*. Serum creatine kinase levels were measured on admission and at 4-hour intervals for the next 24 hours, then daily for a week. In-hospital mortality, long-term survival, recurrence of myocardial infarction, revascularization procedure, and causes of death were also compared.

Electrocardiographic and echocardiographic assessment

An electrocardiogram was taken on admission, every 6 hours for 2 days, every day for 1 week, and every month for 1 year from the time of admission. The electrocardiographic changes were assessed by defining the location of ischemia as follows : anterior, V_1–V_4 (inclusive of the extension to V_5, V_6); inferior, II, III and aV_F (inclusive of the extension to V_4–V_6); and lateral, I, aV_L and V_5, V_6.

A two-dimensional echocardiogram was per-

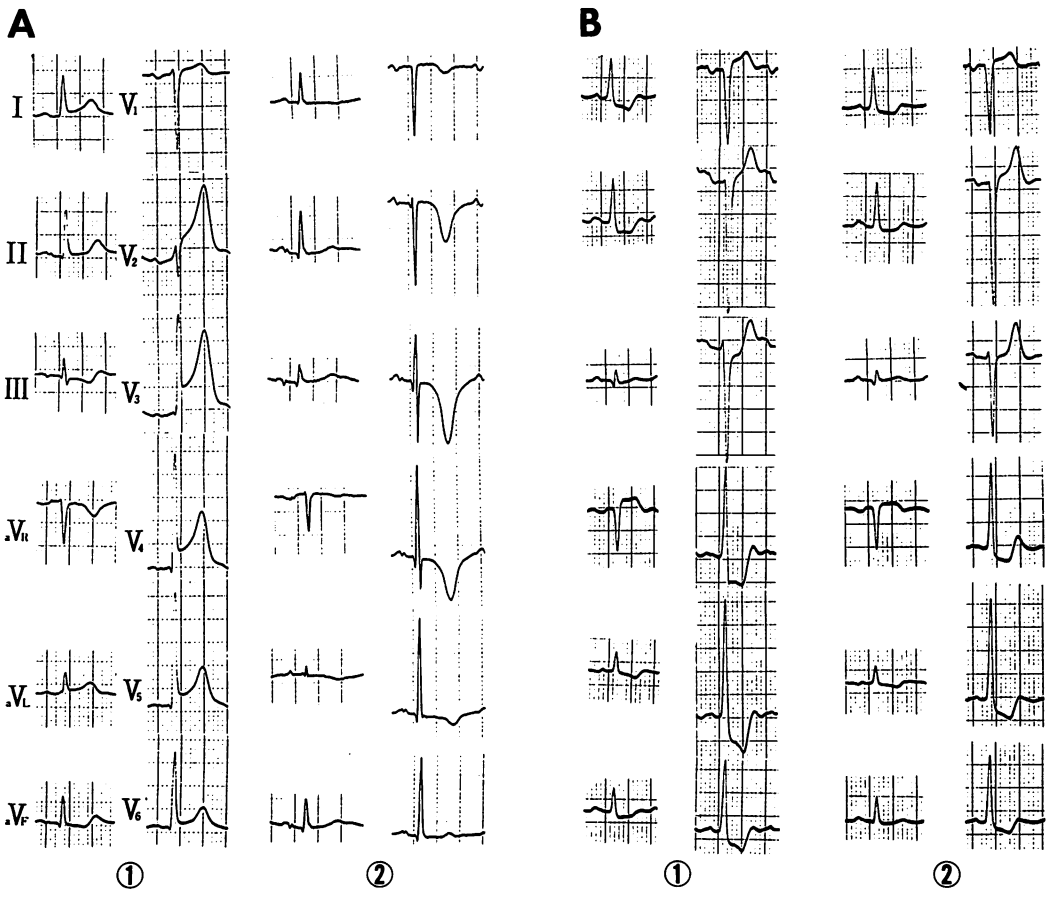

Fig. 1 Electrocardiograms on admission (①) and the second day (②) from a representative case of each group
A: A case of T group **B**: A case of ST group

formed on admission. Data from 35 patients (18 in the ST group and 17 in the T group) were available for analysis. To simplify image evaluation, the left ventricular wall was divided into three segments: anterior, inferior, and lateral wall on the short-axis view of the two-dimensional echocardiogram at the level of the papillary muscle. This level contains all territories of the three major coronary arteries. Left ventricular wall with hypokinesia, akinesia, and dyskinesia was defined as a lesion with contraction abnormalities.

Angiographic and autopsy findings

Eleven patients were considered candidates for cardiac catheterization. Cardiac catheterization was not performed in the other remaining 31 patients because: 22 were aged 80 years or older; 4 died soon after admission; and 5 had other complications (2 renal failure, 2 cerebral infarction, and 1 depression).

Twenty patients (15 in the ST group and 5 in the T group) died after admission or during the follow-up period. Autopsy was performed on 8 patients (5 in the ST group and 3 in the T group) to evaluate the extent of infarction and degree of coronary artery stenosis. Coronary artery stenosis was defined as significant if the coronary angiogram demonstrated more than 75% diameter stenosis or autopsy found more than 90% cross-sectional area stenosis.

Statistical analysis

Values are expressed as mean ± standard deviation. Statistical comparisons between the two groups were performed using the Student's *t*-test and chi-square test. The cumulative survival rate was calculated by the Kaplan-Meier method. A p value < 0.05 was considered significant.

RESULTS

Clinical characteristics

Table 1 shows the clinical characteristics of the whole consecutive patient population. There was no

Table 2 Clinical characteristics of the two groups

	ST group (22)	T group (20)	*p* value
Age (yrs)	80±7	76±10	NS
Sex (F/M)	14/8	9/11	NS
Killip class II-IV	13 (59%)	4 (20%)	<0.05
Use of catecholamines	9 (41%)	3 (15%)	NS
Metabolic acidosis	4 (18%)	1 (5%)	NS
Peak CPK (IU/*l*)	1,041±769	661±534*	NS
Preceding ST elevation	0	16 (80%)	<0.05
Transient Q waves	0	11 (55%)	<0.05
Mortality at 1 month (%)	41	0	<0.05

*n=14
CPK=creatine phosphokinase; NS=not significant

difference in age, sex, or in-hospital mortality between the patients with first and recurrent myocardial infarction. However, there was a significant difference in in-hospital mortality in the subgroups of patients with ST depression and with T wave inversion. There were no in-hospital deaths in the subgroup of patients with first or recurrent infarction associated with T wave inversion. In contrast, in-hospital mortality of the subgroup of patients with ST depression was 52% (14/27) in cases of first infarction, and 34% (10/29) in cases of recurrent infarction.

Table 2 shows the comparison of clinical factors between the ST and T groups. There was no difference in age and sex between the two groups. Fifty-nine percent (13/22) of the ST group and 20% (4/20) of T group were in Killip class II–IV on admission. Catecholamines to maintain blood pressure or treat heart failure were used in 41% (9/22) of the ST group and 15% (3/20) of the T group. Analysis of arterial blood gas showed metabolic acidosis in 18% (4/22) of the ST group and in 5% (1/20) of the T group. These indices indicated that the cardiac status on admission was worse in the ST group than in the T group. The peak value of serum creatine phosphokinase was detected in all patients in the ST group and in 14 patients (70%) in the T group. The other six patients of the T group showed decreasing levels of serum creatine phosphokinase on admission. The peak serum creatine phosphokinase level was higher in the ST group than in the T group (1,041±769 vs 661±534 IU/*l*).

Electrocardiographic changes

Tables 3, 4 show the electrocardiographic changes in the ST and T groups. ST depression was frequently observed in leads I (81%), II (95%), aVF (77%), V4 (86%), V5 (100%) and V6 (86%). T wave inversion was observed in the precordial leads in 95% of patients (19/20), including those with concomitant T wave inversion in the limb leads. The other patient showed T wave inversion in leads II, III and aVF.

The electrocardiograms of the ST group demonstrated ischemic areas in two or three segments in 86% (19/22) of patients (1 segment in 3 patients, 2 in 13 patients, and 3 in 6 patients). Ischemic areas in the T group were located in one segment in 60% (12/20) of patients (anterior 11 patients, inferior 1 patient), and in two (5 patients) or three segments (3 patients) in the others.

Eighty percent (16/20) of T group patients showed ST elevation preceding T wave inversion in the same leads on admission, and 55% (11/20) showed transient Q wave development: 5 patients in leads V1 to V2, 4 in leads V1 to V3, 1 in leads V2 to V4, and 1 in leads III and aVF. The Q waves disappeared within 1 month in 9 patients and within 3 months in the remaining 2, and the R waves subsequently reappeared. The time for normalization of inverted T waves varied : within 1 month in 3 patients, over 1 year in 1 patient, and from 1 to 12 months in the others.

In contrast, no patient in the ST group showed either preceding ST elevation or transient Q wave development, only persistent ST depression from the time of admission.

Extent of ischemic areas and wall motion abnormalities

Echocardiography on admission were performed in 18 patients of the ST group and 17 of the T group. Fourteen patients of the ST group (78%) showed reduced contraction of the left ventricular wall in two or three of the anterior, lateral, and inferior segments : 1 segment in 4 patients, 2 in 5 patients, and 3 in 9 patients. All 17 T group patients showed one segment of contractile abnormality (anterior in 16 patients, inferior in 1 patient).

Clinical outcomes

Patients surviving hospitalization were followed up for 38±24 months (range 2 to 81 months). Fifteen patients (68%) of the ST group died after admission and during the follow-up period. Nine patients died within 1 month of onset of infarction,

Table 3 Clinical characteristics of the T group

Patient			Electrocardiographic findings						
No.	Age (yrs)	Sex (M/F)	Negative T waves	ST elevation	Transient Q waves	Lesion*	CAG	Death	Autopsy
1	61	F	V_1–V_5		—	Ant	+		
2	80	F	V_2–V_6, I, II, aV_L	V_2–V_6, I, aV_L	—	Ant			
3	75	M	V_1–V_4	V_2–V_6, I, aV_L	—	Ant	+		
4	59	F	V_3–V_5	V_1–V_4	V_1, V_2		+		
5	67	M	V_1–V_6	V_1–V_4	V_2–V_4	Ant	+		
6	86	F	V_2–V_6, I, II, aV_L	V_2–V_6, I, aV_L	V_1–V_3	Ant		+	+
7	64	F	V_1–V_5		V_1, V_2	Ant	+		
8	82	F	V_1–V_6, II, III, aV_F	V_1–V_5	V_1, V_2			+	
9	82	F	V_2–V_6	V_3–V_6, I	—			+	+
10	82	F	V_1–V_4	V_2–V_5, I, aV_L	V_1–V_3	Ant		+	
11	56	M	V_1–V_5, aV_L	V_1–V_4	V_1, V_2	Ant	+		
12	78	F	V_1–V_4	V_1–V_5, I, aV_L	V_1–V_3	Ant			
13	84	M	V_1–V_5	V_1–V_6, I, aV_L	V_1–V_3	Ant			
14	82	M	V_1–V_5	V_1–V_4	V_1, V_2	Ant			
15	75	M	V_1–V_4	V_1–V_5	—	Ant	+		
16	85	M	V_3, V_4, II, III, aV_F		—	Ant			
17	67	M	II, III, aV_F		III, aV_F	Inf			
18	89	F	V_3–V_6, I, II, III, aV_F	V_2–V_4	—	Ant		+	+
19	84	M	V_3–V_6, I, II, III, aV_F	V_2–V_6	—	Ant			
20	83	F	V_1–V_5, I, aV_L	V_1–V_6, I, aV_L	—	Ant			

*Myocardial lesion by two-dimensional echocardiogram
CAG=coronary angiography; Ant=anterior wall of the left ventricle; Inf=inferior wall of the left ventricle

eight due to pump failure and one from rupture of the pulmonary artery caused by balloon inflation of a Swan-Ganz catheter. Six patients died more than 1 month after onset, two from pump failure, two from renal failure, one from pneumonia, and one from senile decay. Five of the 10 deaths from pump failure were due to recurrence of infarction. Five patients in the T group died during the follow-up, none within 1 month of onset. Only one patient died from pump failure caused by recurrent myocardial infarction, 2 years after the first infarction. Three patients died of pneumonia, and one of intestinal bleeding (**Fig. 2**).

Eleven patients (4 patients of the ST group and 7 of the T group) underwent coronary and left ventricular angiography during hospitalization. Three patients of the ST group had three-vessel disease and the other had two-vessel disease; left ventriculogram showed regional wall motion abnormalities in the corresponding territory. One patient underwent successful percutaneous balloon angioplasty. Coronary angiograms of the T group showed two-vessel disease in four patients, one-vessel disease in 2, and no significant coronary artery stenosis in 1; left ventriculogram revealed that six patients had contraction abnormalities in one-vessel territory (anterior), which corresponded to the region of T wave inversion in the electrocardiograms. The other patient had normal left ventricular wall motion. Three patients underwent successful percutaneous balloon angioplasty, and one patient

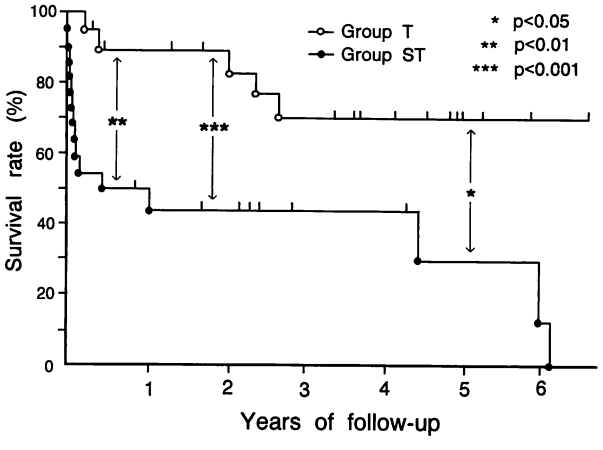

Fig. 2 Cumulative survival curves from the onset of myocardial infarction

Table 4 Clinical characteristics of the ST group

Patient			Electrocardiographic findings						
No.	Age (yrs)	Sex (M/F)	ST depression	ST elevation	Transient Q waves	Lesion	CAG	Death	Autopsy
1	68	M	V_5, V_6, I, II, III, aV_L, aV_F	aV_R	—	Ant, lat, inf		+*	
2	89	F	V_2–V_6, I, II, aV_F	aV_R	—	Ant, inf		+*	
3	73	F	V_1–V_5		—	Ant, lat, inf		+*	
4	75	F	V_4–V_6, I, II, aV_L ,aV_F	aV_R	—	Ant, inf		+*	+
5	86	F	V_4–V_6, I, II, aV_F	aV_R	—	Ant, lat, inf		+*	
6	76	F	V_4–V_6, I, II, III, aV_F	aV_R	—	Ant, lat, inf	+		
7	82	M	V_3–V_6, I, II, aV_L	aV_R	—			+*	+
8	74	M	V_4–V_6, I, II, III, aV_F	aV_R	—	Ant, lat, inf		+	
9	74	M	V_3–V_6, I, II, aV_F	aV_R	—	Ant, lat		+*	
10	74	F	V_5, V_6, I, II, aV_L	aV_R	—	Ant, lat, inf		+*	+
11	67	F	V_3–V_6, II, III, aV_F	aV_R	—	Ant, lat	+	+	
12	94	F	V_3–V_6, I, II, aV_L	aV_R	—	Ant		+	
13	85	F	V_2–V_6, I, II, III, aV_L, aV_F	aV_R	—	Ant, lat, inf		+*	+
14	88	M	V_5, V_6, II, III, aV_F	aV_R	—				
15	82	F	V_4–V_6, I, II, III, aV_F	aV_R	—	Ant, lat, inf		+*	
16	87	F	V_2–V_4, I, II, III, aV_F	aV_R	—				
17	79	F	V_4–V_6, I, II, aV_L	aV_R	—	Ant	+		
18	86	F	V_2–V_5, II, III, aV_F	aV_R	—	Ant			
19	86	M	V_4–V_6, I, II, III, aV_L, aV_F	aV_R	—	Ant, lat, inf		+*	+
20	75	M	V_4–V_6, I, II, aV_F	aV_R	—				
21	89	F	V_4–V_6, II, III, aV_F	aV_R	—	Ant		+*	
22	78	M	V_2–V_6, I, II, aV_L, aV_F	aV_R	—	Ant, inf	+		

*In-hospital death
lat=lateral wall of the left ventricle. Other abbreviations as in Table 3.

underwent bypass surgery.

Autopsy was performed in eight patients (5 patients of the ST group and 3 of the T group). Autopsy found circumferential subendocardial myocardial necrosis in four patients in the ST group, and necrosis of the subendocardium in the anterior to posterior through the septal wall in the other patient. All ST group patients had 90–100% stenosis in all three-vessels. Two patients of the T group had 90% or 75% stenosis of the right coronary artery. The other patient had no significant coronary artery stenosis. None showed macroscopic findings of myocardial infarction.

DISCUSSION

Clinical characteristics of non-Q wave myocardial infarction

The prognostic significance of non-Q wave myocardial infarction remains controversial. Previous studies have reported a lower in-hospital mortality in patients with non-Q wave infarction than in patients with Q wave infarction[6,7], the same mortality in both forms of myocardial infarction[8,9], and a comparable or greater long-term mortality in patients with non-Q wave infarction than in patients with Q wave infarction[10–12].

Our study found that the patients with non-Q wave infarction had a similar in-hospital mortality to those with Q wave myocardial infarction : 31 (40/130) vs 30% (14/47). However, when the 47 patients were divided into subgroups with ST depression and T wave inversion, there was a significant difference in the in-hospital mortality. The differences in clinical characteristics and outcome between ST depression and T wave inversion have not been widely studied. Lown *et al.*[13] studied the prognosis for subendocardial myocardial infarction according to the type of electrocardiographic findings, and reported that an extremely favorable in-hospital course is only associated with T wave inversion, and not persistent ST segment changes (mortality : 0 (0/50) vs 28% (8/29), $p=0.0003$). Abbott *et al.*[4] and Zema[5] reported similar results (mortality : 9 (3/34) vs 37% (14/38), $p<0.005$, 0 (0/22) vs

33% (1/3), $p=0.04$, respectively). These results are consistent with the present study, which showed significant differences in clinical and anatomical characteristics between patients with non-Q wave infarction associated with ST depression or T wave inversion.

This study compared other factors in the subgroups of patients with first non-Q wave myocardial infarction associated with ST depression or T wave inversion.

T wave inversion

Sixty percent (12/20) of T group patients had one ischemic segment defined by electrocardiography, and 100% (17/17) had only one segment with contraction abnormalities confirmed by echocardiography. These results suggest that the ischemic areas of T group patients were localized in one-vessel territory. The characteristic electrocardiographic features of preceding ST elevation and transient Q wave development indicate the presence of transmural ischemia in a one-vessel territory in the early phase of this type of non-Q wave infarction. The presence or reappearance of R waves after transient Q wave development suggests that the coronary flow of the distal portion of the occluded vessel was restored with early reperfusion either by recanalization of the occluded vessel or by collaterals.

Previous reports[14–17] of transient occlusion of the canine coronary artery have shown that when occlusion is relieved 15–20 minutes later, the membrane potential shows a prolongation of the repolarization period even after the recovery of resting and action potential (**Fig. 3–A,B**). In the normal heart, repolarization usually progresses from the subepicardial layer to the subendocardial layer, and the direction of the T wave vector is from the subendocardial toward the subepicardial layer, reflecting the positive T waves in the surface electrocardiogram. When repolarization of injured myocardial cells is delayed compared to normal ones, the direction of the T wave vector changes toward the injured region and causes QT prolongation as well. Prolongation of the repolarization of myocardial cells within the subendocardial layer is apparently not sufficient to cause a T wave vector reversal in the surface electrocardiogram, but must occur in the transmural or near transmural layer (**Fig. 3–D**). Furthermore, many authors have recently reported that persistent

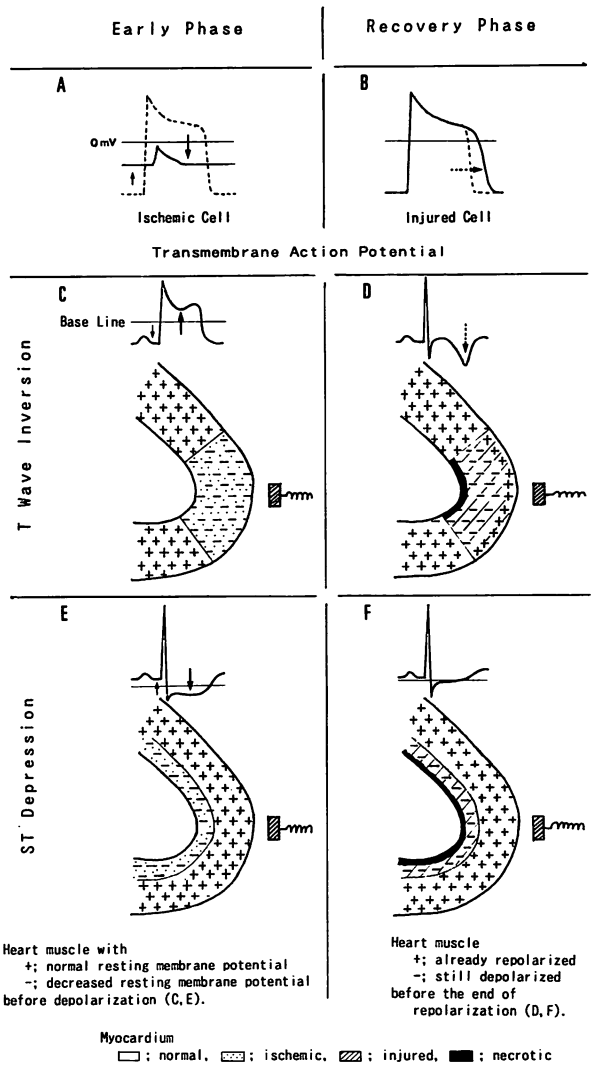

Fig. 3 Schematic presentation of possible electrophysiologic mechanism underlying electrocardiographic changes during early phase and recovery phase of non-Q wave myocardial infarction

Left: Early phase of myocardial infarction
Right: Recovery phase of myocardial infarction
Top: Membrane potential of (*A*) ischemic myocardial cell showing reduced resting membrane potential (*small arrow*) and decreased action potential (*large arrow*), (*B*) injured cell showing prolongation of repolarization period (*dot arrow*).
Middle: Surface electrocardiogram and myocardium with T wave inversion, (*C*) electrocardiogram showing difference of both resting and action membrane potential between normal and ischemic myocardium, causing PQ depression (*small arrow*) and ST elevation (*large arrow*) from the base line, and resulting in ST elevation and myocardium at rest, (*D*) electrocardiogram showing myocardium during repolarization and prolonged repolarization period of myocardial cell of transmural or near transmural layer causing T wave vector inversion (*dot arrow*).
Bottom: Surface electrocardiogram and myocardium with ST depression, indicating limitation of ischemic or injured cell to the subendocardium, causing only ST depression but no changes of the QRS or T vector.

T wave inversion, found in unstable angina without evidence of recent or ongoing myocardial infarction, also represents stunned myocardium after reperfusion of the culprit coronary artery lesion[18,19].

These findings indicate that T wave inversion in non-Q wave myocardial infarction does not reflect the presence of injured or necrotic myocardial cells within the subendocardium, but suggests that injured myocardial cells, which are in the recovery phase from ischemia and associated with the prolongation of the repolarization period, are present in enough layers (transmural or near transmural layers) in a one-vessel territory of the ventricular wall to reverse the direction of the T wave vector in the body surface electrocardiogram. Myocardial necrosis developing under these conditions would presumably be localized in the one-vessel territory subendocardium most susceptible to ischemia[20]. Cook *et al.*[21] reported 24 autopsy cases of small subendocardial myocardial infarction (15 anteroseptal, 8 posterior, 1 lateral). Ten of the 15 cases with anteroseptal subendocardial infarction were characterized by electrocardiographic findings of deeply inverted T waves in the precordial leads, also supporting the findings of the present study.

ST depression

Eighty-six percent (19/22) of ST group patients showed two or three ischemic segments defined by electrocardiography, and 78% (14/18) had two or three segments with wall motion abnormalities confirmed by echocardiography. Electrocardiograms of this group showed neither preceding ST elevation nor transient Q wave development, only ST depression from the onset. This suggests non-Q wave myocardial infarction with ST depression results in subendocardial ischemia, mainly in multivessel territory, from the beginning of infarction (**Fig. 3–E**), unlike T wave inversion, which appeared to start with transmural ischemia in one-vessel territory. Myocardial infarction would then develop in any part of the extensive subendocardial region (**Fig. 3–F**).

Previous authors have also reported a relationship between ST depression and extensive subendocardial infarction and/or multivessel coronary artery disease. Cook *et al.*[22] reported the relationship between acute circumferential or near circumferential subendocardial myocardial infarction and electrocardiographic findings in five cases. The most remarkable electrocardiographic findings were widespread ST depression and segmental ST elevation in lead aVR present in all cases. Sugiura *et al.*[23] reported eight cases with circumferential subendocardial myocardial infarction, which showed widespread ST depression at the time of infarction with severe stenosis of both right and left coronary arteries.

These findings indicate that ischemia begins within the subendocardium of the ventricular wall of multivessel territory, and injury or necrosis of the myocardial cells is confined to that region in non-Q wave myocardial infarction with ST depression.

Limitations of this study

The relatively small number of patients, absence of quantitative analysis of both wall motion abnormalities and coronary angiograms in all patients, and the high age of the study population are limitations of this study. However, more consideration of the clinical and anatomical implications and the significance of these two types of electrocardiographic changes in myocardial infarction will clarify some of the controversy concerning non-Q wave myocardial infarction.

CONCLUSION

Both the in-hospital and long-term survival rates showed significantly better clinical outcomes in patients with T wave inversion in comparison to those with ST depression. The difference in the clinical course between the two groups was supported by the difference in the number of coronary arteries responsible for the ischemic episode and the extent of ischemic, injured, or necrotic myocardium.

The present study suggests that T wave inversion in non-Q wave infarction indicates a recovery phase in transient transmural ischemia in a single-vessel territory, so patients with non-Q wave infarction of this type have favorable residual cardiac function, and are appropriate candidates for coronary angioplasty. In contrast, ST depression in non-Q wave infarction reflects extensive subendocardial ischemia of multivessel territory, so non-Q wave infarction of this type is associated with less favorable outcome. Intensive diagnostic and therapeutic management are required for these patients.

Acknowledgment

I would like to thank Prof. Masahiro Murayama of the Second Department of Internal Medicine, St. Marianna University School of Medicine, for reviewing the manuscript and his helpful comments. I would also like to express my gratitude to Dr. Shin-ichiro Ohkawa and the rest of the cardiology staff at our hospital responsible for the care of the patients in the coronary care unit, and to the staff of Mitsui Memorial Hospital and Toranomon Hospital who performed angioplasty or bypass surgery.

要　約

非Q波心筋梗塞におけるST低下と陰性T波の差異に関する検討

前田　茂

急性心筋梗塞の非Q波梗塞に関する報告は多く，その多くはST低下型と陰性T波型の両方を意味しているが，これら2型の非Q波梗塞が有する臨床的差異に関する報告は極めて少ない．これら2型の非Q波梗塞の違いを明らかにするため，初回発症非Q波急性心筋梗塞の42例をST低下型(ST群22例)と陰性T波型(T群20例)の2群に分類し，心電図変化の特徴とその範囲，心エコー図による心筋障害の範囲，発症時の臨床所見，予後に関して検討した．

T群の80%(16例)で陰性T波出現前にST上昇をみ，55%(11例)では一過性Q波が確認されたが，ST群ではそのような心電図変化はみられず，全例，入院時よりST低下を示した．心筋の前壁，側壁，下壁の3領域中，ST群の86%(19例)では2–3領域で心電図変化を示したのに対し，T群では60%(12例)が1領域の変化であった．入院時の心エコー図検査(乳頭筋レベルの短軸断層像)では，ST群の78%(14/18)が2–3領域の壁運動低下を示したのに対し，T群では全例(17/17)が1領域のみの壁運動低下を示し，2–3領域に及ぶ壁運動低下を示した例はなかった．入院時にKillip分類II–IVの心不全を示したのはST群では59%(13例)であり，発症1ヵ月以内の死亡が9例(8例はポンプ失調)にみられたのに対し，T群では心不全は20%(4例)のみにみられ，院内死亡例はなかった．冠動脈，左室造影所見や剖検所見も，ST群は冠動脈の多枝病変とそれに応じた広範囲の心筋病変を，T群は1枝領域の心筋病変の存在を示した．

非Q波梗塞においては，多くの場合，陰性T波は冠動脈の1枝と思われる領域における一過性貫壁性虚血の回復過程とその領域における心内膜下梗塞を示唆し，ST低下は多枝領域の心内膜下虚血で始まり，その領域内に生ずる心内膜下梗塞を示唆した．

J Cardiol 1994; 24: 357–366

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