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Characteristics of electrocardiographic repolarization in acute myocardial infarction complicated by ventricular fibrillation☆,☆☆

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Abstract

Background and Purpose: Some de- and re-polarization patterns can reflect an increased risk of ventricular tachyarrhythmias. We studied whether some electrocardiographic (ECG) patterns are able to predict the development of ventricular fibrillation (VF) during acute myocardial infarction (MI).
Methods: We compared the patterns of ST-T segment of 78 patients who developed VF during acute MI (patient with VF) vs 170 comparable patients with acute MI but with no VF complications.
Results: Of the VF group, 47 developed out-of-hospital VF and 31 developed VF after their admission to the hospital. A steep downsloping ST segment toward a negative T wave with or without a short, flat, or rising portion at the initial portion was observed in 69.2% of the 78 patients: 61.3% in patients with pre-VF and 74.5% in patients with post-VF, vs 9.4% of patients who did not develop VF ($P < .0001$). In 90.6% of the latter, a typical upward-concave or convex "ischemic" pattern of the ST segment was observed. Thus, the characteristic ST-T patterns were highly associated with VF with a specificity greater than 90%.
Conclusions: A steep downsloping ST segment may characterize the ECGs of patients who develop VF during acute MI.

Keywords: Acute myocardial infarction; Ventricular fibrillation; ST-T pattern

Introduction

Up to 20% of out-of-hospital cardiac arrest from malignant ventricular tachyarrhythmias, including ventricular fibrillation (VF), develops in the acute phase of myocardial infarction (MI).[1-4] Despite the lowering of in-hospital mortality using therapeutic interventions, most notably, early reperfusion, the prognosis after out-of-hospital cardiac arrest due to acute MI, remains poor.[5]

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A systematic review and meta-analysis demonstrated some demographic, clinical, hemodynamic, and electrocardiographic (ECG) parameters as risk factors for primary VF in the acute phase of MI.[6] ST elevation and/or distortion of the QRS complexes will reflect the severity of ischemia, and some ECG patterns in acute MI were found to be associated with VF or the type of VF.[7,8] However, among ECG findings, multivariate analysis demonstrated that only the ST deviation score and RR irregularity that was calculated as the mean square of differences of successive RR intervals measured in 12-lead Holter were demonstrated to be associated with primary VF that developed in 41 of 1473 patients with ST elevation MI.[9,10]

Meanwhile, marked alteration of the ST segment was noted in association with VF early in acute MI: a steep-declining ST segment forming a "lambda" or "coved" ST-T pattern.[11,12] This pattern can be seen during spontaneous or induced coronary spasms before the onset of VF.[13,14]

On the basis of our preliminary observations,[11,12,14] we compared the ECGs of patients who did with those who did not develop VF during acute MI, with particular focus on the pattern of ST-segment elevation (ST-T). We also compared the ECGs before and after the VF episodes to examine the effects of direct current (DC) shocks.

Methods

We retrospectively collected ECGs recorded in 94 patients from April 2006 to March 2011 who had developed VF during the acute phase of MI (VF group) at 7 Japanese and 3 Polish medical centers. The criteria for inclusion in the VF group were (1) VF developed early (within 6 hours) after the onset of MI and before reperfusion therapy, (2) availability of a 12-lead ECG recorded before or after the episode of VF, and (3) confirmation of acute MI by ECG, cardiac enzymes, and coronary angiography.[15] Ventricular fibrillation was defined as disorganized electrical activity of varying amplitude and morphology, without distinct QRS complexes or T waves. The ST segments were measured in the lead with the maximal amplitude 60 milliseconds after the J point and defined as elevated if at least 0.2 mV in leads V_2 through V_3 in men and at least 0.15 mV in women higher than the isoelectric line, as long as the patient did not have left bundle branch block or left ventricular hypertrophy.[15-17]

We identified 120 consecutive patients treated at one Japanese and 50 consecutive patients treated at one Polish enrolling medical center from April 2010 to March 2011 who had a confirmed acute MI uncomplicated by VF (non-VF group), who served as the controls. On admission, all patients underwent a physical examination, recordings of ECGs, and screening blood tests. Unless unconscious, they underwent cardiac catheterization and reperfusion therapy. This study was approved by the Institutional Review Board of Niigata University School of Medicine.

Electrocardiogram analysis

When patients were defibrillated out of the hospital, the 12-lead ECG recorded upon their admission to the hospital was used for this analysis. When VF occurred after their admission to the hospital, the ECG recorded closest to the onset of VF was used. In the non-VF group, the ECG recorded upon admission to the hospital and with the greatest ST-segment deviation before coronary intervention was retained for this analysis. The analysis was based on earlier observations, which particularly focused on the morphology of the ST segment (ST-T).[11,12] The ECGs were classified into 3 main types:

(1) Type I revealed a typical downsloping J-ST segment toward T waves immediately after R without a flat or rising portion. The initial part of the ST segment began at the halfway point to the descending limb of R or close to the top resulting in a "lambda" pattern or "monophasic" pattern, respectively (Fig. 1A);
(2) Type II showed an intermediate pattern of acute ischemia, with a short, flat, or convex upward initial ST segment, then a downsloping segment toward the baseline (Fig. 1B);
(3) Type III showed a concave or convex upward elevation of the ST segment merging into the T wave (Pardee sign), typically observed in acute MI (Fig. 1C).[16-20] Other patterns were classified as "others."

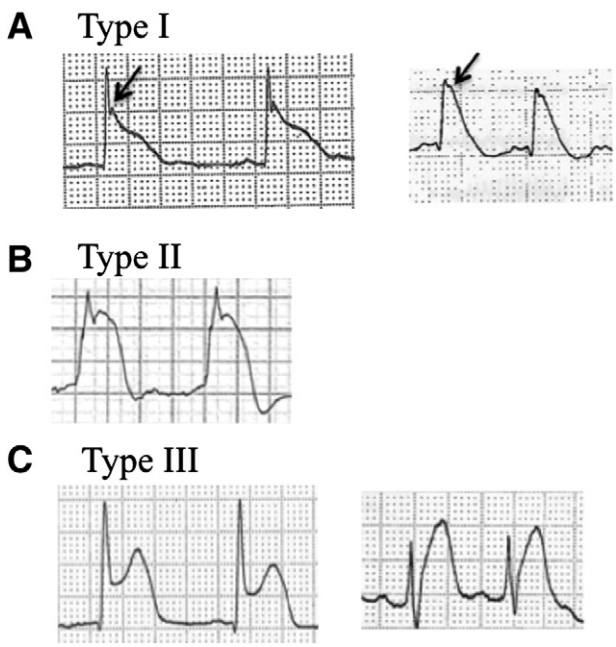

Fig. 1. Classification of typical ST-segment patterns. A, Type I is characterized by a steeply downsloping ST segment toward the T wave that is often inverted. The ST segment may separate midway of the descending limb of QRS or close to the peak of QRS resulting in a "lambda" (left) or "monophasic" pattern (right), respectively. Significance of notches or deflection is to be determined. B, Type II showed an intermediate pattern of acute ischemia, with a short, flat, or convex upward initial ST segment, then a downsloping ST segment toward the baseline. C, Type III is the most common pattern observed during acute MI (Pardee sign) as described in the literature.

The ECGs were classified as type I if a type I pattern was present in any lead, type II if a type II pattern was present in any lead and in absence of a type I pattern, and type III if a type III pattern was present in any lead in the absence of a type I or II pattern (Fig. 2).

The ECGs were assessed by 3 cardiologists unaware of the patient's clinical status whose classifications of the patterns were highly concordant (data not shown). At 5-fold magnification, the amplitude of the ST-segment deviation was measured to a precision of 0.02 mV. The ECG leads with ST elevation were used to assign the MI location as follows: leads V1 through V6 = anteroseptal and anterior MI; lead I or aVL = high lateral MI; and leads II, III, and aVF = inferior MI and their combinations.

Depending on the culprit lesions, patients were divided into 2 groups: left anterior descending artery (LAD) MI with the culprit lesion in the LAD or non-LAD MI with the culprit lesion in the right coronary artery or left circumflex artery.

Data analysis

The clinical characteristics were compared between the patients with and without VF. The relationship between the ECG patterns, and the culprit lesion was determined. Then, the eligible patients were divided into 2 groups: those in whom VF developed before (post-VF) and those in whom it developed after their admission to the hospital (pre-VF). The ST-T pattern and magnitude of ST-segment deviation as well as the clinical characteristics were compared between tracings of the pre-VF group vs the control. The magnitude of ST-segment deviation and the location of MI were compared between LAD and non-LAD MI. When available, the pre-VF ECGs were compared with those recorded after DC shocks that were used to terminate VF. Then, we compared the pre-VF and post-VF tracings for the prevalence of ST-T patterns and ECG parameters. Other clinical characteristics were obtained from the patients' medical records and used for comparisons.

Statistical analysis

Continuous variables are presented as means ± standard deviation, and categorical variables are presented as counts and percentages. Continuous variables were compared using the Student *t* test, and categorical variables were compared using the Pearson χ^2 test. Correction for multiple comparison was

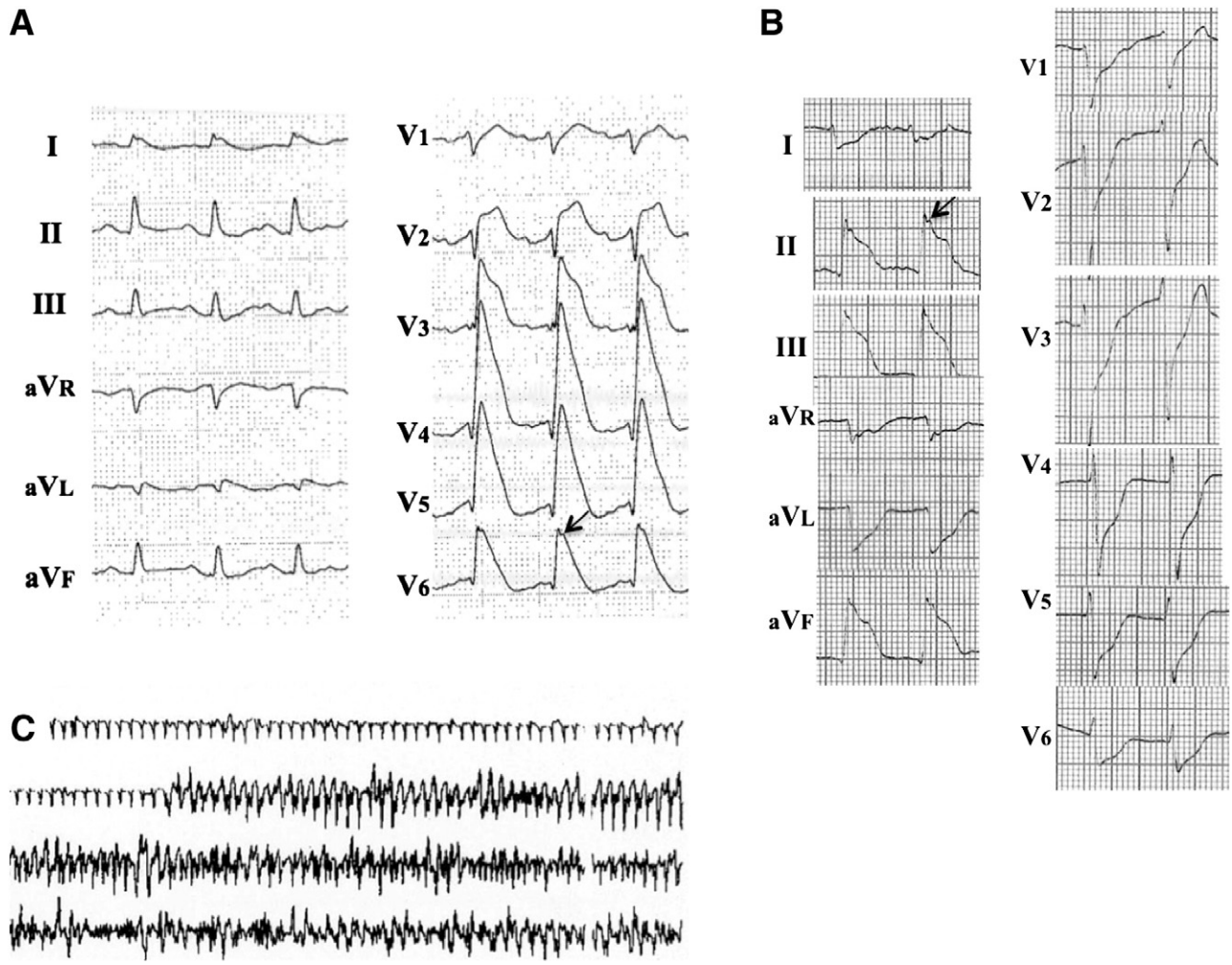

Fig. 2. Electrocardiogram on admission and monitoring. A, Electrocardiogram from a 57-year-old man. On admission, the ECG showed a type I pattern in leads V_3 through V_6. A type I pattern is also present in leads I and aVL. B, The first VF developed during ECG monitoring after recording of 12-lead ECG as shown in A. C, Another example of type I in leads II, III, and aVF. Reciprocal depression of the ST segment was evident in the precordial and high lateral leads.

undertaken for location of MI and culprit vessels. A P value of less than .05 was considered statistically significant. The sensitivity, specificity, and positive and negative predictive values of type I and type I plus II for VF were determined. Odds ratio was calculated to compare mortality in the VF and non-VF group. The analyses were performed, using SPSS statistical software, version 12.0 (SPSS Inc, Chicago, Ill).

Results

Clinical and ECG characteristics

We identified 94 patients who were admitted to the hospital within 6 hours after the onset of chest pain, of whom 14 were excluded from the analysis because they developed complete heart block (n = 6), right bundle branch block (n = 6), or left bundle branch block (n = 2). Another 2 patients who showed no elevation of ST segment were also excluded though acute MI was diagnosed from other data. Among the 78 patients, 5 had ischemic brain injury or shock and did not receive catheterization, 71 patients underwent percutaneous coronary intervention, and 2 underwent coronary artery bypass graft surgery; 3 received no reperfusion therapy because of hemodynamic deterioration. Of these, 47 developed out-of-hospital VF and 31 developed VF after their admission to the hospital, before receiving coronary reperfusion therapy.

The location of MI and culprit vessels showed similar distribution in the VF and non-VF groups (Table 1). Multivessel disease was more frequent in the VF group compared with that in the control: 19.2% vs 7.6%, respectively (P = .0156), and a lower left ventricular ejection fraction was found in the VF group. However, other clinical and ECG characteristics were similar in the VF and non-VF groups (Table 1).

Table 1
Clinical characteristics of MI survivors who did (VF group) vs did not (control group) develop VF

	VF group (n = 78)	Control group (n = 170)	P
Men	63 (80.7)	129 (75.9)	.3927
Age, y	65.7 ± 12.9	66.2 ± 12.3	.7612
Onset to admission, min	110 ± 152	121 ± 87	.6015
Peak serum creatine kinase,[a] IU/L	3740 ± 6392	3630 ± 4115	.4723
Left ventricular ejection fraction, %	49.1 ± 15.4	54.3 ± 13.5	.0097
Location of MI			
LAD area (V_1 through V_6)	23 (29.5)	53 (31.2)	.3412
High lateral (I, aVL)	1 (1.3)	4 (2.4)	
Inferior (II, III, aVF)	24 (30.8)	65 (38.2)	
Inferior + LAD area	16 (20.5)	19 (11.2)	
High lateral + LAD area	14 ((17.9)	29 (17.1)	
Culprit vessel,[b] %			
Left anterior descending	39 (50.0)	80 (47.1)	.3389
Left circumflex	25 (32.1)	71 (41.8)	
Right	12 (15.4)	17 (10.0)	
Left main	2 (2.6)	2 (1.2)	
Medical history			
Hypertension	40 (48.8)	101 (59.4)	.2300
Dyslipidemia	34 (41.5)	92 (54.1)	.1236
Diabetes mellitus	19 (23.2)	42 (24.7)	.9530
Healed MI	12 (14.6)	20 (11.8)	.4298
Stroke	3 (3.6)	3 (1.8)	.3219
Current or past smoking	40 (4.1)	86 (50.7)	.9192

Values are presented as number (percentage) of observations or mean ± SD.
[a] Median was 2360 and 2410, respectively.
[b] All of the control and 73 patients with VF underwent catheterization.

Table 2
Clinical characteristics of the pre-VF and control groups

	VF after admission (n = 31)	Control group (n = 170)	P
Men	25 (80.6)	129 (75.9)	.7692
Age, y	66.5 ± 14.9	66.2 ± 12.4	.6185
ST-segment elevation, mV	0.642 ± 0.425	0.367 ± 0.246	.0001
LAD MI	0.790 ± 0.410	0.489 ± 0.270	<.0001
Non-LAD MI	0.488 ± 0.359*	0.259 ± 0.146**	<.0001
Onset to admission, min	110 ± 152	121 ± 87	.4310
Peak serum creatine kinase, IU/L	4797 ± 5412	3641 ± 4239	.2904
Left ventricular ejection fraction, %	54.6 ± 14.0	54.3 ± 13.5	.9330
Location of MI			
LAD area (V_1 through V_6)	11 (35.5)	53 (31.2)	.1431
High lateral (I, aVL)	0 (0)	4 (2.4)	
Inferior (II, III, aVF)	7 (22.6)	65 (38.2)	
Inferior + LAD area	8 (25.8)	19 (11.2)	
High lateral + LAD area	5 (0)	29 (17.1)	
Culprit vessel			
Left anterior descending	15 (48.4)	80 (47.1)	.2525
Left circumflex	12 (38.7)	71 (41.8)	
Right	2 (6.5)	17 (10.0)	
Left main	2 (6.5)	2 (1.2)	
Medical history			
Hypertension	15 (48.4)	101 (59.4)	.2532
Dyslipidemia	11 (35.5)	92 (54.1)	.0563
Diabetes mellitus	6 (19.4)	42 (24.7)	.5205
Healed MI	3 (9.6)	20 (11.8)	.7371
Previous stroke	2 (6.5)	3 (1.8)	.1233
Current and past smoking	11 (35.5)	86 (50.7)	.1217

Values are presented as number (percentage) of observations or means ± SD.
Other abbreviations are same as those in Fig. 1.
* P = .0011, LAD vs non-LAD MI.
** P < .0001, LAD vs non LAD MI.

ST-T patterns in the pre-VF and non-VF groups

The clinical and ECG characteristics of the 31 patients in the pre-VF group and of 170 controls are shown in Table 2. ST-segment elevation was significantly greater in the pre-VF than in the non-VF group (P = .0001). The ECGs recorded in the pre-VF group revealed the presence of a type I pattern in 16 patients (51.6%), type II in 3 (9.7%), and type III in 12 (38.7%) (Fig. 3). ST-segment elevation was higher in type I (0.721 ± 0.425 mV) than in type III (0.489 ± 0.369 mV) and in the presence of LAD (0.790 ± 0.410 mV) than non-LAD (0.488 ± 0.359 mV) wall MI (P < .0001 and P = .0011, respectively).

Of the 170 controls, types I and II ECG patterns were observed in 7 (4.1%) and 9 (5.3%) patients, respectively, whereas the remaining 154 patients (90.6%) demonstrated typical ST-segment elevation (Figs. 1C and Fig. 3), and the distribution of ST-T patterns in patients with vs those without VF was significantly different (P = .0001).

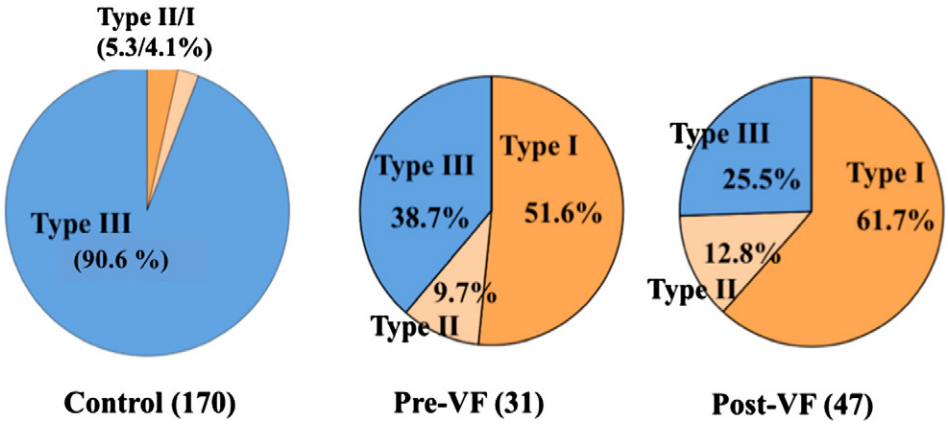

Fig. 3. Distributions of ST-T patterns in acute MI with and without VF. Left, Classical type III pattern was present in nearly all patients who did not develop VF (controls). Center, ST-T patterns of patients who developed VF after admission to the hospital (pre-VF). The ECGs were recorded before defibrillation. Right, ST-T patterns in patients who developed out-of-hospital VF (post-VF). The ECGs were recorded immediately after defibrillation. Type I was more prevalent in post-VF ECG than that in pre-VF ECG, but the difference was statistically nonsignificant. The distribution of ST-T patterns between the VF groups and non-VF group was significantly different ($P = .0001$).

The magnitude of ST elevation was significantly smaller in the control than in the VF group ($P < .0001$) and was greater in the presence of LAD (0.489 ± 0.270 mV) than non-LAD (0.259 ± 0.146 mV) MI ($P < .0001$). The sensitivity, specificity, and positive and negative predictive values for VF were 48.4%, 95.9%, 68.2%, and 92.1%, respectively, for type I and 54.8%, 90.6%, 51.5%, and 91.7%, respectively, for types I and II.

Effect of DC shock on ECG patterns

In 13 patients of the pre-VF group, pre- and post-shock ECGs were available. Of these, types I, II, and III were found in 1, 3, and 9 patients, respectively, in the pre-shock tracings. The post-shock tracings were obtained within 30 seconds after the DC shock. Two were complicated by transient complete right bundle branch block after DC shocks and excluded from the analysis. The DC shocks caused changes in the ST segment: further elevation in 6 and depression in 5 patients. In one patient with a type I pattern, a shift from type II to type I was observed in lead V_4, and including this case, the patterns belonged to the original categories. None with type III showed a progression to type I or II.

Pre- and post-VF electrocardiograms

Ventricular fibrillation occurred out of the hospital in 47 patients who fulfilled the criteria for inclusion in this analysis. They were rescued by emergency medical services equipped with automatic external defibrillators. The time between the onset of VF and recording of the ECG was less than 30 minutes and the onset to time of admission was shorter in patients with post-VF than in those with pre-VF but nonsignificant (Table 3.) Left ventricular ejection fraction and the prevalence of smokers differed ($P = .0077$ and $P = .0373$, respectively).

Type I pattern was more prevalent on the post- (61.7%) than on the pre-VF ECG (51.6%), though the difference was not significant (Fig. 3). The magnitude of ST-segment deviation was not different between the post- (0.694 ± 0.455 mV) compared with the pre-VF (0.642 ± 0.425 mV) ECG, and this was significantly greater in the presence of LAD MI in the post-VF group ($P = .0370$). In the post-VF group, the level of ST-segment deviation was greater in LAD than in non-LAD MI ($P = .0466$). The sensitivity, specificity, and positive and negative predictive values for VF were 57.1%, 95.9%, 86.3%, and 83.6%, respectively, for type I and

Table 3
Clinical characteristics of the post- and pre-VF group

	Post VF (n = 47)	Pre-VF (n = 31)	*P*
Men, n (%)	37 (78.7)	25 (80.6)	.8366
Age, y	64.2 ± 12.3	67.5 ± 14.9	.2872
ST-segment elevation, mV	0.694 ± 0.455	0.642 ± 0.425	.4294
LAD MI	0.856 ± 0.539	0.790 ± 0.410	.0370
Non-LAD MI	0.574 ± 0.336*	0.488 ± 0.359**	.4036
Onset to admission, min	63 ± 76	110 ± 152	.2684
Peak serum creatine kinase, IU/L	3440 ± 7232	4797 ± 5412	.4995
Left ventricular ejection fraction, %	40.4 ± 16.0	54.6 ± 14.0	.0077
ST-segment deviation, mV	0.694 ± 0.455	0.642 ± 0.425	.4293
Location of MI			
LAD area (V_1 through V_6)	12 (29.8)	11 (35.5)	.5240
High lateral (I, aVL)	1 (14.9)	0 (0)	
Inferior (II, III, aVF)	17 (17.0)	7 (22.6)	
Inferior + LAD area	8 (25.5)	8 (25.8)	
High lateral + LAD area	9 (51.1)	5 (0)	
Culprit vessel			
Left anterior descending	24 (51.1)	15 (48.4)	.1766
Right	15 (31.9)	12 (38.7)	
Left circumflex	8 (17.0)	2 (6.5)	
Left main	0 (0)	2 (6.5)	
Concomitant disorders, n (%)			
Hypertension	25 (53.2)	15 (48.4)	.6778
Dyslipidemia	23 (48.9)	11 (35.5)	.2410
Diabetes mellitus	13 (27.7)	6 (19.4)	.4031
Healed MI	9 (19.1)	3 (9.6)	.2566
Previous stroke	1 (2.1)	2 (6.5)	.3312
Smoking[a]	28 (59.6)	11 (35.5)	.0373

[a] Former smokers were included.
* $P = .0466$, LAD vs non-LAD MI.
** $P = .0011$, LAD vs non-LAD MI.

57.9%, 90.6%, 86.3%, and 83.6%, respectively, for types I and II.

Clinical outcomes

In-hospital death occurred in 12 of 47 patients of the post- and in 2 of 31 patients of the pre-VF group from heart failure, irreversible ischemic brain injury, or sepsis. Coronary intervention for revascularization was successful in each patient that improved type I or II toward normal, and VF did not recur in any patient. The patients were discharged from the hospital, except three who were transferred to another medical facility after having ischemic cerebral injury. For a mean follow-up of 855 ± 546 days, only a single patient died of a noncardiac cause, 5.5 months after discharge from the hospital.

In the control group, 168 patients underwent percutaneous coronary intervention, 2 underwent coronary artery bypass graft surgery, and 6 (3.5%) died in the hospital (5 from cardiogenic shock and 1 from mediastinitis). For a mean follow-up of 907 ± 596 days, 1 patient died of congestive heart failure, 1 died suddenly, and 4 patients died of noncardiac causes. The overall odds ratio for all-cause mortality in the VF (19.2%) vs non-VF group (7.06%) was 3.13 (95% confidence intervals = 1.39-7.03).

Discussion

In this study, a steep descent of the ST-T segment (type I or type II pattern) was found in most patients who developed VF during the acute phase of MI. The patterns were predictors of VF with high specificity: greater than 95% for type I and greater than 90% for types I and II. In a small number of patients, the characteristic patterns were not induced by DC shocks. After successful intervention therapy of coronary revascularization, the patterns were resolved and VF no longer recurred. The present study confirmed our preliminary observations that some ECG patterns were a risk factor for VF during the acute phase of MI,[11,12] and prompt coronary revascularization is warranted to resolve ECG patterns and to avoid VF recurrence.

In the modern era, patients with acute MI would be treated using percutaneous coronary intervention for revascularization as soon as acute MI is diagnosed and the door-to-balloon time would be less than 60 minutes in most hospitals. Under such speedy management of acute MI, an ECG can be the simplest and yet most useful tool for the diagnose of MI and clinical status and has been analyzed qualitatively and quantitatively.

In the present study, ECG patterns were classified into 3 types: types I, II, and III. Type I covered these of a "lambda"[11,12] or "monophasic pattern"[21,22] and was highly associated with VF with a high specificity, but the significance of this pattern has been rarely stressed. So far, Sclarovsky and Birnbaum classified the ECG findings of acute MI into 3 patterns, and Sclarovsky and Birnbaum pattern C represents a major distortion of the terminal portion of the QRS complexes leading to disappearance of S waves or emergence of the J point at a level higher than one half of the R-wave amplitude.[7,8] Pattern C has been shown to be associated with higher mortality, larger final infarct size, less myocardial salvage by thrombolytic therapy, and a more rapid progression of necrosis, but its relation to VF has not been fully understood. However, in extreme cases, the Sclarovsky-Birnbaum pattern C may show a "monophasic" pattern[8,9,22,23] meaning some cases with this pattern share common findings as type I in the present study.

From these findings of the present study, it would be safe to say that types I and II are predictors for impending VF during early acute MI. The best therapy for the patients with these ECG patterns would be prompt revascularization that will normalize the findings of the ECG and prevent further attack of VF. The ECG patterns can be very easily recognized in the emergency room.

During the acute phase of MI, the pattern of ST-T would be affected by many factors: rapidity and extent of myocardial ischemia, intramyocardial conduction delay, and/or block in the Purkinje system[21-23], but the pathogenesis of the ECG patterns: the steep-declining ST segment seems to be not yet fully elucidated.

Because of deformation of the QRS complexes and elevated ST segment, it might be difficult to point out the J point exactly in type I but would be usually near the beginning of the declining ST segment, and marked J point elevation was expected. An elevated J point would be mainly the result of focal myocardial block that results in widening of the QRS complex with notching or slurring,[21-24] and the surface leads overlying the ischemic myocardium may show prominent J waves.[21] Such delayed activation might be expected to be easier to detect in right coronary artery and left circumflex artery MI because the conduction delay could be hidden within the QRS complex in LAD MI.[24] However, type I was observed similarly in LAD-and non-LAD MI but only in leads reflecting areas being activated late. Left anterior descending artery MI revealed higher ST elevation and more extensive elevation (Table 1). This would suggest that LAD MI is related to severer myocardial ischemia.

Acute myocardial ischemia has been shown to affect many cardiac ionic channels such as the K_{ATP}, sodium, and calcium channels and shorten the duration of the action potential.[25,26] These changes of the ionic channels would create voltage gradient during the systolic phase and result in additional ST elevation on the surface ECG caused by diastolic injury currents.

A J point followed by a declining ST segment in type I or a "lambda" pattern resembles the "coved" type ECG pattern of Brugada syndrome.[27,28] Although ischemia-induced J waves can be produced and considered to be due to local conduction delay,[23-25] it is to not yet known whether ischemia-induced augmented transient outward currents (Ito) play a role in J-point elevation during acute MI. Prominent J waves unrelated to myocardial ischemia may be seen in patients with idiopathic VF[29,30] or even apparently healthy subjects[31,32] and have been revealed to be a risk factor for sudden cardiac death due to VF. Both prominent J waves due to ischemia-induced delayed activation[33,34] and Ito-induced prominent J waves have been observed to be arrhythmogenic during ischemia.[23,25,26]

Whether the characteristic ST-T pattern found in patients who develop VF during acute MI is genetically determined or not is an important question because a similar ECG pattern is genetically determined in some cases of Brugada syndrome.[35-40] A higher prevalence of familial sudden death has been reported in patients who developed primary VF during acute MI compared with patients who did not.[41] Furthermore, the observation of (a) a higher likelihood of developing VF during acute right ventricular MI compared with other sites[42] and (b) sex-related differences in mortality in acute MI[43] may be explained using Ito that is predominantly expressed in the right ventricular outflow tract and is more prevalent in men than in women.[44] An isolated missense mutation in an SCN5A polymorphism has been reported in 1 of 19 patients who developed VF during acute MI.[45]

Limitations of our study

We acknowledge limitations of this study. First, our classification of the ST-T pattern is arbitrary, though it was based on our preliminary studies as well as on previous reports.[11,12,14] Including historical reports, appropriate naming may be desired to characterize the ECG patterns that represent risk of developing VF. Second, because the ECG could only be recorded from patients who were successfully defibrillated, the ECG patterns in nonsurvivors in the very early phase of acute MI could not be analyzed. Third, a greater prevalence of type I or II and greater ST deviation in the post- than in the pre-VF group might be partly due to DC shocks as well as reanimation procedures,[46] but a comparison of pre- and post-shock ECGs showed that progression from type III to I or II was infrequent though a study of a larger number of cases is warranted.

Finally, the significance of the immediate outcome of the VF group was limited because the proportion of patients who died out of the hospital was unknown; however, the successful treatment of VF appears to be associated with a favorable long-term outcome. To this end, prompt intervention therapy of coronary revascularization is essential. It might be suggested that the prevalence of the ECG pattern is affected by race, but this was not evaluated in our study.

Conclusions

We observed an association between (a) a steep downsloping of the ST segment on the ECGs and (b) VF developing during the early phase of acute MI. This ECG pattern might be an expression of an arrhythmogenic substrate in acute MI.

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