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Predisposing Factors and Prognostic Value of Sustained Monomorphic Ventricular Tachycardia in the Early Phase of Acute Myocardial Infarction

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Objectives. **The purpose of the study was to analyze the factors that favor the occurrence of sustained monomorphic ventricular tachycardia in the early phase (<48 h) of acute myocardial infarction and to establish its prognostic implications.**

Background. **Sustained monomorphic ventricular tachycardia early in the course of an acute myocardial infarction is an uncommon arrhythmia, and its significance has not been specifically studied.**

Methods. **The clinical characteristics and prognosis of sustained monomorphic ventricular tachycardia were studied in 21 (1.9%) of 1,120 consecutive patients admitted to the coronary care unit with a diagnosis of myocardial infarction.**

Results. **Patients with sustained monomorphic ventricular tachycardia had a larger infarct on the basis of peak creatine kinase, MB fraction (CK-MB) isoenzyme activity (435 ± 253 IU/liter vs. 168 ± 145 IU/liter, $p < 0.001$) and higher mortality rate (43% vs. 11%, $p < 0.001$). By logistic regression analysis, independent predictors of sustained monomorphic ventricular tachycardia were CK-MB (odds ratio [OR] 11.8), Killip class (OR 4.0) and bifascicular bundle branch block (OR 3.1). Moreover, sustained monomorphic ventricular tachycardia was itself an independent predictor of mortality (OR 5.0). Compared with patients with ventricular fibrillation, those with sustained monomorphic ventricular tachycardia had a worse Killip class (Killip class >I: 63% vs. 30%, $p < 0.05$), higher CK-MB activity (430 ± 260 IU/liter vs. 242 ± 176 IU/liter, $p < 0.01$) and higher arrhythmia recurrence rate (31% vs. 4%, $p < 0.01$). During the follow-up period, 5 (42%) of 12 survivors in the sustained monomorphic ventricular tachycardia group died of cardiac-related causes. Recurrence of ventricular tachycardia was seen in two patients (17%).**

Conclusions. **Sustained monomorphic ventricular tachycardia during the first 48 h of myocardial infarction is a sign of extensive myocardial damage and an independent predictor of in-hospital mortality.**

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The prognosis and the electrophysiologic mechanisms of sustained monomorphic ventricular tachycardia occurring in the subacute and chronic phases of myocardial infarction have been extensively investigated in patients and in experimental models (1,2). However, knowledge of the predisposing factors and the prognosis of sustained monomorphic ventricular tachycardia occurring during the first 48 h of myocardial infarction is very limited. Previous experimental data from our laboratory support the concept that spontaneous, sustained monomorphic ventricular tachycardia occurs in large myocardial infarctions (3). In contrast, the majority of clinical studies dealing with ventricular arrhythmias during the early phase of myocardial infarction are focused on ventricular fibrillation (4,5) or have considered ventricular tachycardia and ventricular fibrillation together (6–8). To our knowledge, only one study has analyzed the prognostic value of sustained ventricular tachycardia in the acute phase of myocardial infarction (9), but patients with hemodynamic compromise were excluded. Furthermore, no studies comparing patients suffering from sustained monomorphic ventricular tachycardia with those presenting with ventricular fibrillation have been reported. Such a comparison may be relevant because several investigators have demonstrated that the characteristics and the arrhythmogenic substrate of patients with sustained monomorphic ventricular tachycardia in the chronic phase of infarction are different from those with ventricular fibrillation (10,11).

The aim of the study was to analyze the clinical characteristics and the prognosis of patients with sustained monomorphic ventricular tachycardia during the early phase of myocardial infarction. In addition, we compared the clinical variables

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Abbreviations and Acronyms

CCC = coronary care unit
CK = creatine kinase
CK-MB = creatine kinase, MB fraction
ECG = electrocardiogram, electrocardiographic

of these patients with those from patients with ventricular fibrillation.

Methods

Patients. From January 1991 to January 1995, 1,124 patients with acute myocardial infarction were admitted to the coronary care unit (CCU) of our hospital. Readmissions to the CCU because of a reinfarction episode during follow-up occurred in 36 patients, but these episodes of reinfarction were not included in the analysis.

Data collection. Data on demographic and clinical characteristics, laboratory results and complications during the hospital stay were collected prospectively in a specially designed form. The diagnosis of myocardial infarction was based on clinical symptoms, evolving electrocardiographic (ECG) changes and serial elevation of total creatine kinase (CK) and CK-MB fraction isoenzyme (MB-CK) activity. Continuous ECG monitoring was started in the emergency room and was continued throughout the patients' stay in the CCU. Lidocaine was not administered prophylactically. Patients with sustained monomorphic ventricular tachycardia during the acute phase of infarction were followed up at the outpatient clinic. None of them was discharged on antiarrhythmic drugs, and they were not submitted to an electrophysiologic study.

To provide a comparative estimation of follow-up mortality among patients without sustained monomorphic ventricular tachycardia, we analyzed data from an institutional registry that began in April 1994 and included the last 167 patients of this nontachycardia group.

Definitions. *Sustained monomorphic ventricular tachycardia* was defined as a wide QRS (>120 ms) tachycardia with a uniform QRS configuration in at least one ECG monitor lead, and a constant cycle length that lasted longer than 30 s or provoked hemodynamic collapse.

For the purpose of the study, episodes of *ventricular fibrillation* that took place as a degeneration of a fast monomorphic ventricular tachycardia were not included in the ventricular fibrillation group. Also, patients who recovered from ventricular fibrillation outside the hospital were excluded because the initiating arrhythmia might have been a sustained monomorphic ventricular tachycardia. We did not consider an episode of ventricular fibrillation if it occurred as the final event of a cardiogenic shock.

Statistical analysis. Data are expressed as the mean value ± SD. Differences among groups were assessed using the chi-square test for qualitative variables and the Student *t* test for continuous variables. Logistic regression analysis was performed using forward stepwise selection and the method of the likelihood ratio test to obtain the model that better explained the occurrence of sustained monomorphic ventricular tachycardia and the model that better predicted in-hospital mortality—the dependent variables in both cases. CK-MB activity was transformed into a dichotomic variable according to the best sensitivity and specificity to predict the event using the receiver operating characteristic curves (12). Two different cutoff points were chosen: one for the prediction of sustained monomorphic ventricular tachycardia with a CK-MB activity of 350 IU/liter and another for the prediction of mortality with a CK-MB activity of 100 IU/liter. The level of statistical significance was $p < 0.05$. Statistical analysis was performed using the SPSS package (13).

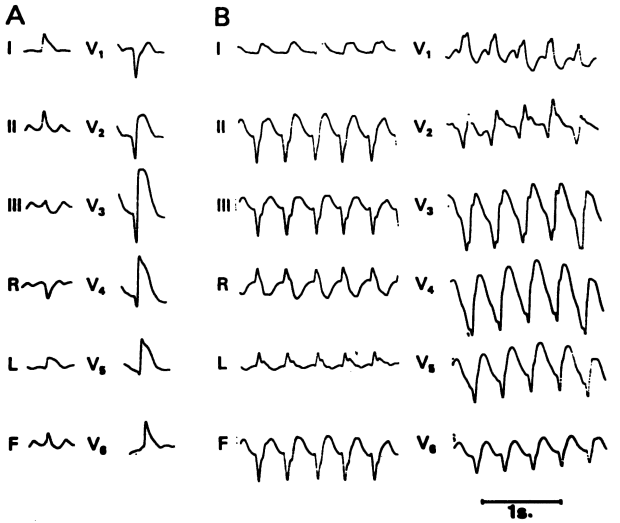

Figure 1. Twelve-lead ECG from Patient 5, who had an acute anterior myocardial infarction **(A)** complicated by an episode of sustained monomorphic ventricular tachycardia occurring 2 h after the onset of infarction **(B).**

Results

Among the 1,124 consecutive patients admitted with the diagnosis of myocardial infarction, 25 presented a sustained ventricular tachycardia during the first 48 h of infarction. Four of them were excluded from the analysis (one patient because the tachycardia was polymorphic and three because of incomplete evidence of the monomorphism of the QRS complex during tachycardia). Therefore, the frequency of sustained monomorphic ventricular tachycardia in the very early phase of myocardial infarction was 1.9% (21 of 1,120 patients). A 12-lead ECG during tachycardia could be obtained in 7 of the 21 patients.

Figure 1 depicts an example of a sustained monomorphic ventricular tachycardia in a patient with an extensive anterior myocardial infarction. Table 1 shows the clinical data from the 21 patients with sustained monomorphic ventricular tachycardia. Patients with this arrhythmia had a high peak CK and

Table 1. Clinical Characteristics of Patients With Sustained Monomorphic Ventricular Tachycardia

Pt. No./Gender	Age (yr)	Loc	CK (IU/liter)	CK-MB (IU/liter)	Thromb	Delay (h)*	Killip Class†	Time (h)‡	EF (%)	Death§	Follow-Up Event
1/M	69	Ant	2,119	177	SK	2	I	2.30	33	—	AMI, CABG, SMVT, dead
2/M	65	Ant	2,229	442	None	—	III	< 1	—	Shock	—
3/F	79	Ant	3,122	378	None	—	II	6	—	Arrhyth	—
4/M	52	Ant	3,822	301	t-PA	2	II	1	30	—	No events
5/M	57	Ant	5,652	754	t-PA	2	III	2	20	Rupture	—
6/M	58	Ant	3,948	416	SK	2	I	24	35	—	Angina
7/M	45	Inf	3,696	449	SK	5	I	44	35	—	No events
8/F	40	Inf	—	—	None	—	IV	9	—	Arrhyth	—
9/F	67	Ant	6,018	783	t-PA	4	IV	1	—	Shock	—
10/M	53	Ant	3,822	301	None	—	II	1	30	—	AMI, HF, dead
11/M	57	Inf	4,974	824	None	—	I	2	35	—	HF, SMVT, dead
12/M	76	Ant	1,506	409	t-PA	2	III	1	24	—	AMI, HF, dead
13/M	74	Ant	5,000	1,521	t-PA	2	II	12	—	Arrhyth	—
14/M	49	Inf	2,448	240	SK	3	II	3	46	—	HF
15/M	57	Ant	1,984	232	SK	3	I	10	—	—	No events
16/M	57	Inf	—	—	None	—	I	30	—	Shock	—
17/F	73	Ant	2,592	444	None	—	III	7	33	—	HF
18/M	65	Ant	4,845	605	SK	3	IV	38	—	Shock	—
19/M	50	Ant	4,662	448	SK	3	I	42	46	—	No events
20/M	75	Ant	611	104	None	—	II	40	30	—	AMI, dead
21/M	69	Ant	5,310	528	None	—	IV	6	—	Shock	—

*Time from onset of symptoms to onset of thrombolysis. †Killip class at admission. ‡Time of occurrence of ventricular tachycardia after onset of infarction. §Cause of in-hospital death. AMI = acute myocardial infarction; Ant = anterior wall myocardial infarction; Arrhyth = death due to ventricular arrhythmia; CABG = coronary artery bypass graft surgery; CK = creatine kinase; CK-MB = creatine kinase, MB fraction; EF = ejection fraction; HF = readmission due to heart failure; Inf = inferior wall myocardial infarction; Loc = localization of infarction; SK = streptokinase; SMVT = episode of sustained monomorphic ventricular tachycardia; Thromb = thrombolysis; t-PA = tissue-type plasminogen activator; — = data not available or applicable.

CK-MB isoenzyme activity and a low ejection fraction, although some data were not available in patients who died early in the course of myocardial infarction. Nine (42%) of the sustained monomorphic ventricular tachycardia episodes took place during the first 4 h of the infarction. Three of these nine episodes occurred during or immediately after thrombolysis. Of the 21 index episodes, 15 (71%) occurred during the first 12 h. Eight patients (38%) were in Killip class III or IV when the index episode of sustained monomorphic ventricular tachycardia took place. The tachycardia rate was 173 ± 38 beats/min (range 120 to 250). Circulatory arrest was seen in eight patients (38%) during the index episode of tachycardia and cardioversion was required in 16 patients (76%), whereas in five patients the arrhythmia was controlled by intravenous antiarrhythmic drugs. One patient died during the index episode, two patients died because of recurrent episodes of ventricular tachycardia in the same day, five patients died because of cardiogenic shock and one patient died from a late cardiac rupture.

Univariate analysis for sustained monomorphic ventricular tachycardia. Table 2 shows the results of the univariate analysis comparing the characteristics of patients with and without sustained monomorphic ventricular tachycardia. No differences in age, gender distribution or risk factors were observed. Patients with sustained monomorphic ventricular tachycardia had larger infarctions than patients without sustained monomorphic ventricular tachycardia, as judged by their higher peak CK-MB isoenzyme activity, worse Killip class at admission, higher frequency of anterior wall infarctions, higher frequency of bifascicular bundle branch block and lower ejection fraction. Patients with ventricular tachycardia more often received

Table 2. Clinical Characteristics, Complications and Mortality in Patients With and Without Sustained Monomorphic Ventricular Tachycardia

	No SMVT (n = 1,099)	SMVT (n = 21)	p Value
Male/female	846/253	17/4	0.66
Age (yr)	61 ± 10	61 ± 11	0.97
Hypertension	488 (44%)	8 (38%)	0.56
Smoker	700 (64%)	16 (76%)	0.23
Diabetes	274 (25%)	4 (19%)	0.54
Previous angina	427 (39%)	7 (33%)	0.61
Previous MI	210 (19%)	5 (23%)	0.59
Ant	409 (37%)	16 (76%)	< 0.001
Inf	433 (40%)	5 (23%)	< 0.02
Thromb	343 (31%)	11 (52%)	< 0.04
Killip class >I	244 (22%)	13 (62%)	< 0.001
CK (IU/liter)	1,573 ± 1,457	3,668 ± 1,559	< 0.001
CK-MB (IU/liter)	168 ± 145	435 ± 253	< 0.001
Bifascicular BBB	92 (8%)	8 (38%)	< 0.001
AV block	74 (7%)	4 (19%)	< 0.05
VF	33 (3%)	2 (10%)	0.08
EF	50 ± 14%	37 ± 12%	< 0.001
In-hospital death	126 (11%)	9 (43%)	< 0.001

Data presented are mean value ± SD, number of patients or number (%) of patients. AV = atrioventricular; BBB = bundle branch block; MI = myocardial infarction; VF = ventricular fibrillation; other abbreviations as in Table 1.

Table 3. Results of Logistic Regression Analysis: Independent Predictors of Sustained Monomorphic Ventricular Tachycardia Occurrence

Variable	Corr Coeff	OR (CI)	p Value
Constant	−5.53		
CK-MB	2.47	11.8 (4.47–31.43)	< 0.001
Killip class	1.39	4.04 (1.49–10.9)	< 0.004
Bifascicular BBB	1.14	3.12 (1.08–8.99)	0.04

CI = confidence interval; Corr Coeff = correlation coefficient; OR = odds ratio; other abbreviations as in Table 2.

thrombolytic therapy than their counterparts, and they had a higher mortality than those without sustained monomorphic ventricular tachycardia (43% vs. 11%). There were no differences in the characteristics of sustained monomorphic ventricular tachycardia (rate and time of presentation) between patients with and without thrombolysis.

Logistic regression analysis for prediction of sustained monomorphic ventricular tachycardia. As shown in Table 3, among a set of 10 variables included in the logistic regression analysis (age, previous angina, diabetes, gender, hypertension, prior myocardial infarction, anterior myocardial infarction, Killip class, bifascicular block and CK-MB activity), only the following three variables were chosen as independent predictors of the occurrence of sustained monomorphic ventricular tachycardia: 1) CK-MB activity >350 IU/liter; 2) Killip class >I; and 3) bifascicular bundle branch block. When these three conditions were present, the probability of developing sustained monomorphic ventricular tachycardia was 35.5%.

Univariate analysis for in-hospital mortality. Table 4 shows the comparison among survivors and patients who died during their hospital stay. All comparisons demonstrated statistical differences among groups with a borderline statistical significance for a previous infarction. The presence of sustained monomorphic ventricular tachycardia was less frequent in survivors (1%) than in those who died during their hospital stay (7%).

Table 5. Results of Logistic Regression Analysis: Independent Predictors of In-Hospital Death

Variable	Corr Coeff	OR (CI)	p Value
Constant	−6.19		
Killip class	1.77	5.89 (3.68–9.41)	0.001
SMVT	1.61	5.0 (1.63–15.3)	0.004
VF	1.11	3.06 (1.14–8.19)	0.02
Diabetes	0.99	2.69 (1.67–4.32)	< 0.001
Angina	0.72	2.06 (1.3–3.26)	0.019
CK-MB	0.67	1.95 (1.19–5.21)	0.008
Age	0.033	1.03 (1.00–1.06)	0.013

Abbreviations as in Tables 1 to 3.

Logistic regression analysis for prediction of mortality. As depicted in Table 5, 12 variables obtained during the first hours of hospitalization (age, gender, diabetes, hypertension, previous angina, previous infarction, Killip class, CK-MB activity, anterior myocardial infarction, bifascicular bundle branch block, ventricular fibrillation, as well as the presence of sustained monomorphic ventricular tachycardia) were included in the model of logistic regression analysis. Among these variables, age, Killip class, presence of sustained monomorphic ventricular tachycardia, occurrence of ventricular fibrillation, diabetes, previous angina, and CK-MB activity were selected as independent predictors of in-hospital mortality. The presence of sustained monomorphic ventricular tachycardia was an independent prognostic factor associated with a fivefold increase in mortality.

Comparison between patients with sustained monomorphic ventricular tachycardia and ventricular fibrillation. Table 6 shows the results of the univariate analysis comparing the

Table 4. Univariate Analysis of In-Hospital Death

	Alive (n = 985)	Dead (n = 135)	p Value
Male/female	781/204	82/53	< 0.001
Age (yr)	61 ± 10	66 ± 9	< 0.001
Hypertension	423 (43%)	73 (54%)	< 0.05
Smoker	659 (67%)	57 (42%)	< 0.001
Diabetes	227 (23%)	57 (42%)	< 0.001
Previous angina	366 (37%)	68 (50%)	< 0.001
Previous MI	181 (18%)	34 (25%)	0.06
Ant	353 (36%)	72 (53%)	< 0.001
Thromb	316 (32%)	38 (28%)	0.36
Killip class >I	169 (17%)	88 (65%)	< 0.001
CK (IU/liter)	1,561 ± 1,443	2,079 ± 1,777	< 0.001
CK-MB (IU/liter)	170 ± 149	206 ± 174	< 0.025
SMVT	12 (1%)	9 (7%)	< 0.001
VF	28 (3%)	7 (5%)	0.14
Bifascicular BBB	71 (7%)	29 (21%)	< 0.001
AV block	56 (6%)	22 (16%)	< 0.001

Data presented are mean value ± SD, number of patients or number (%) of patients. Abbreviations as in Tables 1 and 2.

Table 6. Comparison of Patients With Sustained Monomorphic Ventricular Tachycardia and Those With Ventricular Fibrillation

	SMVT (n = 19)	VF (n = 27)	p Value
Male/female	15/4	22/5	0.83
Age (yr)	61 ± 11	60 ± 8	0.65
Hypertension	8 (42%)	13 (48%)	0.68
Smoker	15 (79%)	19 (71%)	0.51
Previous angina	7 (37%)	7 (26%)	0.43
Previous MI	5 (26%)	4 (15%)	0.33
Ant	14 (73%)	12 (44%)	< 0.05
Thromb	10 (53%)	11 (41%)	0.42
Killip class >I	12 (63%)	8 (30%)	< 0.05
CK (IU/liter)	3,481 ± 1,545	2,863 ± 2,069	0.29
CK-MB (IU/liter)	430 ± 260	242 ± 176	< 0.01
Bifascicular BBB	7 (37%)	5 (19%)	0.16
AV block	3 (16%)	4 (15%)	0.92
EF	36 ± 12 (%)	38 ± 12%	0.72
In-hospital recurrence	6 (31%)	1 (4%)	< 0.01
In-hospital death	8 (42%)	4 (15%)	< 0.05

Data presented are mean value ± SD, number of patients or number (%) of patients. Abbreviations as in Tables 1 and 2.

clinical characteristics of patients with sustained monomorphic ventricular tachycardia with those with ventricular fibrillation. Two patients with both arrhythmias were excluded from the analysis. Furthermore, six patients who had recovered from ventricular fibrillation before hospital admission were also excluded because the initiating arrhythmia could have been a sustained monomorphic ventricular tachycardia. Patients with sustained monomorphic ventricular tachycardia had larger infarctions (inferred by their higher CK-MB activity, greater incidence of anterior wall infarction and worse Killip class), more arrhythmia recurrences and higher mortality during the acute phase of infarction than patients with ventricular fibrillation. In five of the six patients with recurrences of sustained monomorphic ventricular tachycardia, the arrhythmia developed within 3 h of the index episode, whereas in the remaining patient the episode occurred 24 h later.

Follow-up of patients with sustained monomorphic ventricular tachycardia. During a mean follow-up of 29 months (range 2 to 51), 5 (42%) of 12 patients discharged from the hospital had a cardiac death. Two patients died in cardiogenic shock after a reinfarction; one had a suspected reinfarction and died at home; one was admitted with advanced heart failure and died in cardiogenic shock; and one died perioperatively during coronary artery bypass graft surgery. Before the operation, the last patient had a reinfarction complicated by three episodes of sustained monomorphic ventricular tachycardia. Another patient had a recurrence of sustained monomorphic ventricular tachycardia of a different configuration 15 months after the myocardial infarction without evidence of reinfarction. The follow-up of the remaining seven surviving patients is summarized in Table 1. Cardiac mortality in the subset of 167 patients comprising the control group was 12% during a mean follow-up of 13 months (range 7 to 17).

Discussion

Main findings. This study shows that the occurrence of sustained monomorphic ventricular tachycardia during the early phase of myocardial infarction is a sign of extensive myocardial damage and an independent predictor of in-hospital mortality. Furthermore, when compared with patients with ventricular fibrillation, patients with sustained monomorphic ventricular tachycardia have larger infarctions, a higher in-hospital recurrence rate of the arrhythmia and a higher in-hospital mortality. In addition, during the follow-up, patients with sustained monomorphic ventricular tachycardia had a high risk of cardiac events but a low probability of arrhythmia recurrence or sudden death.

Role of infarct size. The influence of the infarct size on the development of sustained monomorphic ventricular tachycardia during the early phase of myocardial infarction is not well known because this arrhythmia has not been analyzed separately from ventricular fibrillation (6–8). Only indirect evidence obtained from a study performed by Eldar et al. (9) suggests that there may be a relation between infarct extension and occurrence of sustained ventricular tachycardia. In a multicenter trial involving 2,776 patients, these investigators separately analyzed patients with sustained ventricular tachycardia in the early phase of myocardial infarction from those with ventricular fibrillation. In the latter study, only patients without heart failure were included; thus, the role of infarct size on the genesis of sustained monomorphic ventricular tachycardia might have been underestimated. Despite this, the work by Eldar et al. (9) still suggests a relation between sustained ventricular tachycardia and infarct size, which may explain the higher in-hospital mortality in the arrhythmia group.

Our data indicate that sustained monomorphic ventricular tachycardia occurred in patients with extensive infarctions because it was associated with a high peak of CK-MB activity, heart failure and bifascicular bundle branch block. Furthermore, the ejection fraction of survivors was severely depressed.

Animal models analyzing the role of infarct extension in the genesis of spontaneous sustained ventricular tachycardia in the early phase of myocardial infarction are scarce, and there are no reports in which the polymorphic and monomorphic forms of tachycardia were studied separately. The work by Bolli et al. (14) in a dog model of acute ischemia demonstrated that the presence of prolonged ventricular tachycardia (>10 beats/min) was related to the amount of ischemic myocardium, to the severity of myocardial flow reduction and to the increase in heart rate. Our previous observations in a pig model of acute transmural infarction without reperfusion (3) showed that spontaneous, sustained monomorphic ventricular tachycardia occurred in large infarctions induced by the proximal ligature of the left anterior descending coronary artery, whereas ventricular fibrillation occurred in either the proximal or distal coronary ligature.

Possible mechanism of sustained monomorphic ventricular tachycardia. In the subacute and chronic phases of myocardial infarction, it has been well recognized that reentry through a stable circuit involving the infarct scar tissue is the most likely mechanism of sustained monomorphic ventricular tachycardia (15–17). In contrast, in the very acute phase of a first myocardial infarction, when no previous scar tissue is present, the mechanism of the sustained monomorphic ventricular tachycardia is not known. During acute ischemia, it has been shown (18,19) that there are zones of slow conduction and block that may create the conditions for reentry. Our observation that patients with sustained monomorphic ventricular tachycardia have large infarcts supports the concept that, even in the absence of a healed myocardial infarction, a very large acute ischemic zone may create the conditions for a transiently stable reentry circuit capable of sustaining a monomorphic reentrant tachycardia. Alternatively, a focal mechanism linked to a high sympathetic neural drive or to mechanical stretching (20) elicited by left ventricular dysfunction could be also considered.

Prognostic implications. Our data show that patients with sustained monomorphic ventricular tachycardia had a poor prognosis and a very high mortality (43%). Moreover, sustained monomorphic ventricular tachycardia during the very

early phase of myocardial infarction was an independent predictor of in-hospital mortality, even when other significant prognostic variables were included in the model of regression. In view of the very high mortality observed in these patients and also considering that many episodes of sustained monomorphic ventricular tachycardia occurred during the first 4 h of infarction, it would probably be advisable to adopt a more aggressive therapeutic approach in these patients, even considering the need for emergency catheterization and revascularization through coronary angioplasty.

Differences between sustained monomorphic ventricular tachycardia and ventricular fibrillation. We have observed that sustained monomorphic ventricular tachycardia occurred in a subset of patients different from that of ventricular fibrillation. Although this concept has been well established in chronic infarction arrhythmias (10,11), no comparisons have been made in the acute phase of myocardial infarction. Our data show that patients with sustained monomorphic ventricular tachycardia had larger infarctions than patients with ventricular fibrillation. Furthermore, during the hospital stay, the former group had more recurrences of the arrhythmia and had a higher mortality rate (42% vs. 15%).

Role of thrombolysis. Although reperfusion arrhythmias are very prominent in experimental studies (21), they do not seem to be clinically relevant in patients treated with thrombolytic agents (22). The most frequent reperfusion arrhythmias are ventricular premature beats, accelerated idioventricular rhythms and nonsustained ventricular tachycardia, rather than ventricular fibrillation or sustained ventricular tachycardia (23–27). A meta-analysis of ventricular arrhythmias in thrombolytic trials (7) showed that the risk for ventricular fibrillation is greater in patients receiving placebo than in those treated with thrombolytic agents, whereas the risk for ventricular tachycardia was higher in thrombolyzed patients. Nevertheless, because sustained and nonsustained tachycardias were considered together, no definitive conclusions could be drawn. In patients treated with tissue plasminogen activator, Berger et al. (6) found a lower patency rate of the infarct-related artery in cases with ventricular tachycardia or ventricular fibrillation, suggesting that these arrhythmias are more often associated with occlusion of the infarct-related artery. Although in our series there are no data on early patency of the artery, it is very unlikely that sustained monomorphic ventricular tachycardia was related with reperfusion. The fact that thrombolytic therapy was more frequently used in the ventricular tachycardia group was probably due to the higher percentage of transmural anterior infarctions associated with prominent ST segment changes. These ECG features promote a higher use of thrombolytic agents, as recently demonstrated by Sharkey et al. (28).

Follow-up. Although patients with sustained monomorphic ventricular tachycardia had a high incidence of cardiac events and death during follow-up, the risk for arrhythmia recurrence was low. Moreover, recurrences were much lower than those observed when sustained monomorphic ventricular tachycardia occurs in patients with chronic infarctions (1).

The poor prognosis of these patients was mainly related to the impaired ventricular function and to the increased risk for a new infarction rather than to arrhythmic events. Thus, an electrophysiologic study to guide further therapeutic interventions may not be necessary.

Study limitations. One of the variables that led us to conclude that patients with sustained monomorphic ventricular tachycardia had large infarctions is the observed peak CK-MB activity; however, because many patients with a sustained monomorphic ventricular tachycardia received cardioversion this could have increased the CK-MB activity. Nevertheless, it is unlikely that this factor distorted the results, because CK and CK-MB activity was comparable in patients with sustained monomorphic ventricular tachycardia treated or not treated with cardioversion. Furthermore, patients with sustained monomorphic ventricular tachycardia had a higher CK-MB activity as compared with patients with ventricular fibrillation, even though all the patients in the ventricular fibrillation group were defibrillated. In fact, O'Neill et al. (29), showed that cardioversion only induces a very slight increase in CK-MB activity, unless a high number of discharges are applied. A second limitation of the study is the lack of a 12-lead ECG during the episode of tachycardia in the 14 patients who required immediate cardioversion. This fact may theoretically result in a misinterpretation of a ventricular tachycardia as a supraventricular tachycardia with aberrant conduction. However, our assumption that a regular, fast and poorly tolerated wide QRS tachycardia occurring in the early phase of myocardial infarction is a ventricular tachycardia seems reasonable. A 2:1 atrial flutter with aberrant conduction could mimic a sustained monomorphic ventricular tachycardia, but this arrhythmia would have had a frequency of 150 beats/min and probably would have been well tolerated. The presence of atrial tachycardia with aberrant conduction and poor hemodynamic tolerance in the acute phase of myocardial infarction is very uncommon, and it is highly unlikely that any of these patients suffered from this arrhythmia.

Conclusions. Sustained monomorphic ventricular tachycardia occurring in the acute phase of myocardial infarction is a marker of extensive myocardial infarction associated with a high in-hospital mortality. This fact would probably warrant more aggressive diagnostic and therapeutic interventions and a longer stay in the CCU. Mortality and morbidity in patients with sustained monomorphic ventricular tachycardia during follow-up were high but generally not related to arrhythmic events.

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References

1. Josephson ME. Recurrent ventricular tachycardia. In: Josephson ME, editor. Clinical Cardiac Electrophysiology: Techniques and Interpretations. Philadelphia, Lea & Febiger, 1993:417–615.
2. Wit AL, Janse MJ. Ventricular arrhythmias in the acute phase of myocardial ischemia and infarction. In: Wit AL, Janse MJ, editors. The Ventricular

Arrhythmias of Ischemia and Infarction. Mount Kisco, NY: Futura, 1993: 161–260.
3. Cinca J, Bardají A, Carreño A, et al. Low incidence of ventricular arrhythmias during acute ischaemia at the borders of a chronic infarction in pigs. Cardiovasc Res 1994;28:1018–24.
4. Volpi A, Maggioni A, Franzosi MG, Pampallona S, Mauri F, Tognoni G. In-hospital myocardial infarction: prognosis of patients with acute myocardial infarction complicated by primary ventricular fibrillation. N Engl J Med 1987;317:257–61.
5. Behar S, Goldbourt U, Reicher-Reiss H, Elieser K, and the Principal Investigators of the SPRINT study. Prognosis of acute myocardial infarction complicated by primary ventricular fibrillation. Am J Cardiol 1990;66:1208–11.
6. Berger PB, Ruocco NA, Ryan TJ, Frederick MM, Podrid PJ. Incidence and significance of ventricular tachycardia and fibrillation in the absence of hypotension or heart failure in acute myocardial infarction treated with recombinant tissue-type plasminogen activator: results from the Thrombolysis in Myocardial Infarction (TIMI) phase II trial. J Am Coll Cardiol 1993;22:1773–9.
7. Solomon SD, Ridker PM, Antman EM. Ventricular arrhythmias in trials of thrombolytic therapy for acute myocardial infarction: a meta-analysis. Circulation 1993;88:2575–81.
8. Tofler GH, Stone PH, Muller JE, et al., and the MILIS study group. Prognosis after cardiac arrest due to ventricular tachycardia or ventricular fibrillation associated with acute myocardial infarction (the MILIS study). Am J Cardiol 1987;60:755–61.
9. Eldar M, Sievner Z, Goldbourt U, Reicher-Reiss H, Kaplinsky E, Behar S, for the SPRINT Study Group. Primary ventricular tachycardia in acute myocardial infarction: clinical characteristics and mortality. Ann Intern Med 1992;117:31–6.
10. Brugada P, Waldecker B, Kersschot Y, Zehender M, Wellens HJJ. Ventricular arrhythmias initiated by programmed stimulation in four groups of patients with healed myocardial infarction. J Am Coll Cardiol 1986;8:1035–40.
11. Martínez-Rubio A, Shenasa M, Borggrefe M, Chen X, Benning F, Breithardt G. Electrophysiologic variables characterizing the induction of ventricular tachycardia versus ventricular fibrillation after myocardial infarction: relation between ventricular late potentials and coupling intervals for the induction of sustained ventricular tachyarrhythmias. J Am Coll Cardiol 1993;21:1624–31.
12. Feinstein AR. Diagnostic and spectral markers. In: Feinstein AR, editor. Clinical Epidemiology: The Architecture of Clinical Research. Philadelphia: WB Saunders, 1985:597–631.
13. Norusis MJ. SPSS for Windows Base System User's Guide, Release 6.0. Chicago: SPSS Inc., 1993.
14. Bolli R, Fisher DJ, Entman ML. Factors that determine the occurrence of arrhythmias during acute myocardial ischemia. Am Heart J 1986;111:261–70.
15. Marchlinsky FE, Waxman HL, Buxton AE, Josephson ME. Sustained ventricular tachyarrhythmias during the early post-infarction period: electrophysiologic findings and prognostic for survival. J Am Coll Cardiol 1983;2: 240–50.
16. De Bakker JMT, van Capelle FJL, Janse M, et al. Reentry as a cause of ventricular tachycardia in patients with ischemic heart disease: electrophysiologic and anatomic correlation. Circulation 1988;77:589–606.
17. Littman L, Svenson RH, Gallagher JJ, et al. Functional role of the epicardium in postinfarction ventricular tachycardia: observations derived from computerized epicardial activation mapping, entrainment, and epicardial laser photoablation. Circulation 1991;83:1577–91.
18. Janse MJ, Van Capelle FJL, Morsink H, et al. Flow of "injury" current and patterns of excitation during early ventricular arrhythmias in acute regional myocardial ischemia in isolated porcine and canine hearts: evidence for two different arrhythmogenic mechanisms. Circ Res 1980;47:151–65.
19. Podgwizd SM, Corr PB. Reentrant and nonreentrant mechanisms contribute to arrhythmogenesis during early myocardial ischemia: results using three-dimensional mapping. Circ Res 1987;61:352–71.
20. Wit AL, Rosen MR. After depolarizations and triggered activity: distinction from automaticity as an arrhythmogenic mechanism. In: Fozzard HA, Haber E, Jennings RF, Katz AM, Mogan HE, editors. The Heart and Cardiovascular System. New York: Raven, 1991:2113–63.
21. Cinca J, Worner F, Carreño A, Bardají A, Salas-Caudevilla A, Soler-Soler J. Reperfusion arrhythmias after chronic regional denervation of the ischaemic myocardium in pigs. Cardiovasc Res 1992;26:962–7.
22. Cinca J. Arritmias de reperfusión durante la trombolisis coronaria: ¿realidad clínica o curiosidad experimental? Rev Esp Cardiol 1992;45 Suppl 2:60–5.
23. Goldberg S, Greenspon AJ, Urban PL, et al. Reperfusion arrhythmias: a marker of restoration of antegrade flow during intracoronary thrombolysis for acute myocardial infarction. Am Heart J 1983;105:26–32.
24. Wei JY, Markis JE, Malagold M, Braunwald E. Cardiovascular reflexes stimulated by reperfusion of ischemic myocardium in acute myocardial infarction. Circulation 1983;67:796–801.
25. Kuck KH, Schoffer J, Schlüter J, Bleifeld W, Mathey D. Reperfusion arrhythmias in man: influence of intravenous lidocaine. Eur Heart J 1985; 6:163–7.
26. Buckingham TA, Devine JE, Redd RM, Kennedy HL. Reperfusion arrhythmias during coronary reperfusion therapy in man. Chest 1986;90:346–51.
27. Gore JM, Ball SP, Corrao JM, Goldberg RJ. Arrhythmias in the assessment of coronary artery reperfusion following thrombolytic therapy. Chest 1988; 94:727–30.
28. Sharkey SW, Berger CR, Brunette DD, Henry TD. Impact of the electrocardiogram on the delivery of thrombolytic therapy for acute myocardial infarction. Am J Cardiol 1994;73:550–3.
29. O'Neill PG, Faitelson L, Taylor A, Puleo P, Roberts R, Pacifico A. Time course of creatine kinase release after termination of sustained ventricular dysrhythmias. Am Heart J 1991;122:709–14.