Clinical and Electrophysiological Characteristics of Sustained Ventricular Tachycardia Occurring 3 to 21 Days After Acute Myocardial Infarction

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Patients with sustained ventricular tachycardia (VT) in the post-infarction period, especially more than 48 h after acute myocardial infarction (AMI), have a high mortality. However, clinical characteristics of these patients are unknown, since previous studies have not clarified the relationship between sustained VT and acute myocardial damage.

To clarify the prognostic factors in patients with sustained VT in the early post-infarction period, we retrospectively surveyed 961 consecutive patients with AMI. Eleven patients (9 men and 2 women, aged 54 to 80 years) who had no previous myocardial infarction had at least one documented episode of sustained VT between 3 and 21 days after AMI.

Late potentials were detected in 7 of 7 patients who underwent signal-averaged electrocardiography within 2 days after the first occurrence of VT. Entrainment was seen in 2 patients. The 5 patients who died in hospital had the following clinical characteristics: 1) cardiogenic shock, 2) extensive infarction, 3) a short interval from AMI to the onset of VT (mean $\pm$ SD: 4 ± 2 days), and 4) recurrent and refractory VT. In contrast, the 6 patients who survived had the following clinical characteristics: 1) no cardiogenic shock, 2) a relatively late occurrence of VT (mean $\pm$ SD: 14 ± 7 days), 3) few episodes of VT, and 4) no recurrence of VT during the follow-up period of 12 to 58 months.

The occurrence of sustained VT within 3 weeks after AMI was influenced by the general condition of the patient, and the prognosis was mainly related to cardiac function.

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BETWEEN 5 and 10% of patients develop sustained ventricular arrhythmias after acute myocardial infarction (AMI)!^{–3} Most of these life-threatening tachyarrhythmias occur within the first 48 h after the onset of AMI, and the mechanism is regarded as non-reentrant^{4–7} After prompt management with conversion to normal sinus rhythm, the prognosis of these arrhythmias is

relatively good^{2,3,8} However, some patients develop sustained ventricular tachycardia (VT) more than 48 h after AMI. In contrast to ventricular tachyarrhythmias within the initial 48 h period, this later-onset VT is usually recurrent and refractory to therapy. Some previous clinical and experimental studies have suggested that VT occurring more than 48 h after AMI has a reentrant mechanism similar to that in the chronic state of infarction^{9–13} In general, sustained VT which develops 48 h to several months after AMI is associated with a high

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TABLE I CLINICAL CHARACTERISTICS OF THE 11 PATIENTS

Pt. no.	Age	Sex	VT onset (days)*	CHF Class**	Site of AMI	Peak CK	CAG	Treatment Dose of catecholamine (μ g/min/Kg)	Follow-up (months)
1	61	M	3	4	Ant (ext.)	10541	—	PCPS DOA 5, DOB 5	dead
2	80	M	3	4	Inf-Post	15120	—	IABP DOA 10, DOB 10	dead
3	69	M	3	4	Ant (ext.)	10223	TVD	IABP, LVAS DOA 10, DOB 10	dead
4	63	M	5	4	Inf-Post	6888	TVD	PTCA, PCPS DOA 5, DOB 8	dead
5	59	F	5	1	Post-Lat	5886	DVD	Conventional†	alive (43)
6	72	F	6	2	Inf-Post	6952	—	Conventional†	alive (17)
7	54	M	8	4	Ant (ext.)	6822	SVD	PTCA, PCPS, AN, CR, DOA 10, DOB 10	dead
8	59	M	14	1	Ant (ext.)	7600	DVD	AN	alive (53)
9	79	M	19	1	Ant (ext.)	5012	—	Conventional†	alive (12)
10	61	M	19	1	Ant (ext.)	5100	DVD	Conventional†	alive (58)
11	69	M	20	1	Ant (ext.)	5489	SVD	Conventional†	alive (12)

AN=aneurysmectomy; AMI=acute myocardial infarction; Ant(ext.)=extensive anterior; CAG=coronary angiography; CHF class**=grade of congestive heart failure by Killip's classification; CK=creatinine kinase (IU/L); CR=cryosurgery; DOA=dopamine; DOB=dobutamine; DVD=double-vessel disease; IABP=intra-aortic balloon pumping; Inf-Post=inferoposterior; LVAS=left ventricular assist system; PCPS=percutaneous cardiopulmonary support; Post-Lat=posterolateral; SVD=single-vessel disease; TVD=triple-vessel disease; VT=ventricular tachycardia; VT onset*=numbers of days from AMI to the occurrence of VT; Conventional†=medical treatment without surgery or PTCA

mortality^{1,10,14,15}

However, these previous studies included patients with previous myocardial infarction and did not clearly indicate whether VT was caused by AMI or originated from substrate related to previous myocardial infarction^{9-12,14,15}. Histological evaluation of ventricular myocardium after the onset of AMI suggests that the substrate for ventricular arrhythmias changes as the AMI heals.¹⁶ In the present study, we focused on the relationship between sustained VT and AMI in the early post-infarction period. To clarify the prognostic factors for VT in the early post-infarction period, we retrospectively evaluated the clinical and electrophysiological characteristics of 11 patients who had sustained VT between 3 and 21 days after AMI.

METHODS

Patients

We retrospectively surveyed 961 consecutive AMI patients who were admitted to our coronary care unit between May 1986 and March 1992. Eleven of these patients developed at least one documented episode of sustained VT between 3 and 21 days after AMI. Patients with previous myocardial infarction were excluded from the study.

Previous myocardial infarction was diagnosed if the patient had previously been hospitalized for a documented myocardial infarction, or if abnormal Q waves were found in a previous electrocardiogram before admission.

Clinical Evaluation

Clinical information was obtained from hospital records and the following factors were evaluated: 1) the site of infarction, 2) the number of coronary arteries with >75% stenosis, 3) the performance of percutaneous transluminal coronary angioplasty (PTCA) in the acute stage, 4) the severity of congestive heart failure on occurrence of sustained VT (graded according to the classification of Killip¹⁷), and 5) the peak creatine kinase level (IU/L).

Evaluation of VT

Sustained VT was defined as VT persisting for more than 30 sec or causing cardiovascular collapse that required cardioversion within 30 sec. The first drug administered for the termination of VT was generally lidocaine, but the second drug given, and other means used to terminate VT, varied from case to case. The following drugs were used to prevent VT: lidocaine, mexiletine, procainamide, and quinidine. Signal-averaged elec-

TABLE II CHARACTERISTICS OF VT

Pt. no.	No. of VT Episodes	Treatment Terminating VT	Drug Prophylaxis for VT	S-A ECG	EPS
1	>5	DC	ineffective	—	—
2	>5	DC	ineffective	—	—
3	>5	DC	ineffective	—	—
4	>5	DC	ineffective	LP (+)	—
5	>5	procainamide	quinidine	LP (+)	—
6	1	DC	—	LP (+)	—
7	>5	DC	ineffective	LP (+)	—
8	>5	lidocaine	procainamide	LP (+)	—
9	1	DC	—	—	—
10	2	spont. termination	—	LP (+)	1 month
11	1	DC	—	LP (+)	1 month

EPS=electrophysiological study; DC=direct current; LP=late potential; No.=number; pt.=patient; S-A ECG=signal-averaged electrocardiogram; VT=ventricular tachycardia; (—)=not evaluated

trocardiograms were recorded within 2 days after the first episode of VT. Late potentials were defined as positive when the following criteria were met: 1) the total filtered-QRS duration was more than 120 msec, 2) low-amplitude signals of less than 40 μ V persisted for more than 40 msec, and 3) the root-mean-square voltage of the terminal 40 msec of the QRS complex was less than 20 μ V. Two patients underwent electrophysiological studies 1 month after AMI.

Follow-up

The 6 patients who survived the acute stage were followed up for 12 to 58 months (mean $\pm$ SD: 32.5 $\pm$ 21.3 months).

Statistical Analysis

Data are expressed as the mean $\pm$ SD. Differences in clinical variables among patient subgroups were analyzed by Student's *t* test. A probability (*p*) value of less than 0.05 was considered significant.

RESULTS

Clinical Characteristics (Table I)

The subjects consisted of 9 men and 2 women aged from 54 to 80 years (66 $\pm$ 8.5 years).

Seven patients had extensive anterior infarction, 3 patients had inferoposterior infarction, and 1 patient had posterolateral infarction. All of the patients had peak creatine kinase levels above 5000 IU/L.

Six patients underwent coronary angiogra-

phy (CAG) in the acute stage (patients no. 4, 5, 7, 8, 10, and 11). Patients no. 7 and 11 had single-vessel disease, patients no. 5, 8, and 10 had double-vessel disease, and patient no. 4 had triple-vessel disease. Although CAG could not be performed on admission in patient no. 3, angiograms obtained 3 months before admission showed triple-vessel disease. Patients no. 1 and 2 were in very poor condition and could not undergo CAG.

PTCA of the infarct-related artery was performed in 2 patients (patients no. 4 and 7). In patient no. 4, the CAG on admission showed an occlusion at the proximal right coronary artery and 50% stenosis of the left main trunk. PTCA was performed in the right coronary artery under percutaneous cardiopulmonary support, and successful recanalization was obtained 6 h after the onset of AMI. Patient no. 7 developed frequent drug-refractory VT beginning the 8th hospital day. PTCA was attempted on the 10th hospital day to improve the blood supply to the area adjacent to the infarction. However, PTCA of the left anterior descending artery did not help to control VT. The other patients who underwent CAG (patients no. 5, 8, 10, and 11) did not receive PTCA or thrombolytic therapy because they were admitted more than 12 h after the onset of AMI.

Four patients were in cardiogenic shock on admission (patients no. 1–4) and their hemodynamic condition did not improve with the first occurrence of VT. Patient no.

Fig. 1. Duration from AMI to the first episode of VT. The black bars indicate patients with cardiogenic shock and the open bars indicate those without cardiogenic shock. Patients with cardiogenic shock developed VT earlier than those without shock (4 ± 2 days vs 14 ± 7 days, $p < 0.01$).

7 was not in cardiogenic shock on admission, but his condition deteriorated rapidly to shock with the occurrence of VT.

The major clinical differences between patients with cardiogenic shock (patients no. 1, 2, 3, 4, and 7) and those without shock (patients no. 5, 6, 8, 9, 10, and 11) were evaluated. There was no difference in age (68 ± 9 years vs 65 ± 9 years, NS). The severity of coronary artery disease could not be compared because CAG was not performed in all patients. Patients with cardiogenic shock showed higher peak creatine kinase levels than those without shock (10693 ± 3382 IU/L vs 6123 ± 1008 IU/L, $p < 0.01$).

In patients with cardiogenic shock, intra-aortic balloon pumping or percutaneous cardiopulmonary support, in combination with catecholamine infusion (the maximum dose is shown in Table I), was required to maintain the blood pressure, and patient no. 3 was treated with a left ventricular assist system. Five other patients (patients no. 5, 8, 9, 10, and 11) had no signs of congestive heart failure, while patient no. 6 developed mild congestive heart failure.

Characteristics of VT (Table II)

Patients with cardiogenic shock developed VT sooner after AMI than those without shock (mean number of days between AMI and onset of VT: 4 ± 2 days vs 14 ± 7 days, $p < 0.01$) (Fig. 1).

All of the patients with cardiogenic shock had more than 5 episodes of VT and cardioversion was always required for

termination, since antiarrhythmic drugs were ineffective. Antiarrhythmic therapy was also ineffective in preventing VT. These patients had 2 or 3 morphological types of VT and their heart rates ranged from 136 to 186 min, but VT did not degenerate to ventricular fibrillation. In patient no. 3, sustained monomorphic VT occurred incessantly from the third day after AMI. This VT was occasionally terminated by rapid pacing from the right ventricular apex and exhibited constant fusion with the pacing beats.

Two of the 6 patients without cardiogenic shock, (patients no. 5 and 8) had more than 5 episodes of VT, while the remaining patients (patients no. 6, 9, 10, and 11) had only 1 or 2 episodes. All 6 of these patients had only 1 morphological type of VT and their heart rates ranged from 144 to 160 min. Procainamide and lidocaine were effective for terminating VT in patients no. 5 and 8, with no recurrence after quinidine or procainamide was started. In 3 of the remaining patients (patients no. 6, 9, and 11), VT was terminated by cardioversion because of cardiovascular collapse, while it ceased spontaneously in patient no. 10.

Aneurysmectomy with cryosurgery was performed in patient no. 7 on the 14th day after AMI because recurrent VT was refractory to medical therapy. Aneurysmectomy alone was performed in patient no. 8 on the 30th day after AMI.

Signal-averaged electrocardiography was performed in 7 patients (patients no. 4, 5, 6, 7, 8, 10, and 11) within 2 days after the first occurrence of VT, and late potentials were detected in each case.

Electrophysiological studies were performed in 2 patients (patients no. 10 and 11) 1 month after AMI. In patient no. 10, the study showed abnormally low and fragmented potentials in the left anterior endocardial region and septum, which corresponded to the infarcted area. However, VT could not be induced even by triple paired extrastimuli or by burst pacing from the right ventricular apex. In patient no. 11, the electrophysiological study showed low and fragmented potentials in the left anterolateral region. Three morphological types of VT were induced by double paired extrastimuli to the right ventricular apex. Constant fusion and progressive fusion were obtained

by pacing from the right ventricular apex.

Follow-up

Five patients with cardiogenic shock (patients no. 1, 2, 3, 4, and 7) died in the hospital due to pump failure or complicating acute renal failure. The duration from AMI to death ranged from 3 to 73 days. The 6 patients without cardiogenic shock (patients no. 5, 6, 8, 9, 10, and 11) survived and had no recurrence of VT during the follow-up period of 12 to 58 months (32.5 ± 21.3 months). Patients no. 5 and 8 continued to receive quinidine and procainamide, while the other patients received no antiarrhythmic drugs during the follow-up period.

DISCUSSION

Sustained VT occurring more than 48 h after AMI are regarded as reentrant. However, previous reports have not clarified whether the substrate for reentrant VT was caused by acute myocardial damage or previous myocardial infarction⁹⁻¹². In the present study, to limit the investigation to patients with reentrant VT originating from substrate caused by AMI, patients with previous myocardial infarction were excluded from enrollment. Although the reentrant mechanism was not clarified in each episode of VT, signal-averaged electrocardiograms showed late potentials, suggesting an anatomic substrate for reentry, in 7 of these patients within 2 days after the first occurrence of VT. In addition, constant fusion or progressive fusion with pacing beats was seen during VT in patients no. 3 and 11. These results suggest that the VT was due to reentry.

Clinical Characteristics and Prognosis

Sustained ventricular arrhythmias occurring more than 48 h after AMI are associated with a very high mortality, ranging as high as 50 to 80% at 1 year^{1,10,14,15}. Kleiman et al reported that the following factors were independently associated with an increased risk of mortality: a shorter interval from AMI to the onset of VT (<1 month), more than 3 episodes of spontaneous VT, anterior AMI, and multivessel coronary artery disease¹⁵. Although there have been few reports of sustained VT associated with AMI

in Japan, Hayakawa et al emphasized that frequent and later-onset VT after the acute stage of AMI was associated with a poor prognosis¹⁸.

In this study, we evaluated 11 patients who developed sustained VT within 3 weeks after AMI. Five of these 11 patients had a poor prognosis. Although we can not propose a definitive conclusion, due to the small number of patients, there appeared to be several differences between the clinical characteristics of patients who died and those who survived. All 5 patients who died in the hospital had extensive myocardial damage and were in cardiogenic shock. The first episode of VT developed sooner after AMI than it did in the 6 surviving patients, and the former patients' VTs were recurrent and refractory to drugs. In contrast, the 6 patients who survived were not in cardiogenic shock and had a relatively late occurrence of VT. Their episodes of VT were generally infrequent, and VT could be controlled by antiarrhythmic drugs, even in the 2 patients with frequent episodes. However, there was no apparent difference in the infarction site between the 2 groups. The severity of coronary artery disease could not be compared because CAG was not performed in all patients.

Gomes et al reported that there was a correlation between late potentials and the occurrence of VT in the acute and post-coronary care period after AMI¹⁹. Seven patients had late potentials in our study. A pathological study¹⁶ has suggested that the anatomic substrate for reentry is a scattered band of surviving subendocardial myocytes combined with interstitial edema, which separates the myocytes and is associated with acute inflammatory infiltrates by the third day after AMI. A gradual change to chronic inflammatory infiltrates occurs by 6 weeks, and a firm collagenous scar with interspersed surviving muscle fibers eventually develops. This suggests that the anatomic substrate in patients who develop VT in the very early period after AMI might differ from that in patients with a later onset of VT.

Among the patients who died, the occurrence of VT may have been influenced by systemic factors associated with circulatory failure, such as increased sympathetic tone, catecholamine infusion or electrolyte imbal-

ances. The final hemodynamic deterioration was closely linked to refractory VT in these patients. Among the patients who survived, 4 had only 1 or 2 episodes of VT and the 2 patients with recurrent VT were well controlled by antiarrhythmic drugs. In these patients, systemic factors were less likely to have had an influence on the occurrence of VT.

One interesting finding is that the patients who survived had no recurrence of VT during the follow-up period of 12 to 58 months. In a report by Kleiman et al, among patients who developed 1 or 2 episodes of VT within 3 to 90 days after AMI the incidence of VT recurrence during the next 6 months was 14%, compared with 42% for those with 3 or more episodes of VT.¹⁵ It is unknown whether patients who survived in our series resembled those with VT in the chronic phase of infarction. However, patients with sustained VT in the chronic stage of infarction do not always develop VT in the early post-infarction period. Sustained VT can be induced by programmed ventricular stimulation in up to 70% of AMI patients who have not had spontaneous VT.^{20,21} Thus, further prospective studies will be required to clarify the factors which determine the onset of VT after the anatomic substrate is established.

Treatment

CAG was performed in 7 patients and PTCA was performed in patients no. 4 and 7 with cardiogenic shock. Although the infarct-related artery was recanalized 6 h after AMI, extensive infarction occurred in patient no. 4. There is little information available regarding the efficacy of PTCA in suppressing VT. Since VT was refractory to medical therapy in patient no. 7, PTCA was attempted to improve the blood supply to the area adjacent to the infarction. However, this attempt was not successful. One possible reason why PTCA was not effective in suppressing VT in either of our patients may be that recanalization of the infarct-related artery was performed too late to salvage the affected myocardium and to prevent the formation of an anatomic substrate for VT.

Two patients underwent aneurysmectomy. Aneurysmectomy with cryosurgery was performed on the 14th day after AMI in

patient no. 7. Although it was effective in suppressing VT, the patient died of acute renal failure and congestive heart failure on the 59th day after surgery. Patient no. 8 underwent aneurysmectomy without cryosurgery on the 30th day after AMI, when VT had already been suppressed by drug treatment. The efficacy of surgical therapy for VT in the early post-infarction period is controversial because of the high operative mortality.^{10,15} If surgical therapy is to be used in the suppression of refractory VT, patients should be carefully selected. Recently, the use of intracoronary ethanol ablation for recurrent sustained VT has been reported.²²

Major limitations of our study included its retrospective nature and the small number of patients. Although these limitations, particularly the latter, prevent us from drawing any definite conclusions, our findings highlight the heterogeneity of patients with sustained VT in the early post-infarction period.

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