A Case of Acute Myocardial Infarction with Reentrant Sustained Ventricular Tachycardia Developing in the Prehospital Phase

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SUMMARY

A case of development of monomorphic sustained ventricular tachycardia in the prehospital phase of acute myocardial infarction is reported. By performing pacing from the right ventricular outflow tract during ventricular tachycardia, constant fusion and progressive fusion were documented without constant and progressive fusion from the right ventricular apex pacing, and it was terminated by pacing from the right ventricular outflow tract. Thus, reentry was considered to be the mechanism of this ventricular tachycardia occurring in the prehospital phase. Direct angioplasty successfully recanalized the totally occluded coronary artery, but late potential was present probably because of late reperfusion. In an electrophysiologic study in the chronic phase, slow conduction areas were found at the interventricular septum and the exit of this ventricular tachycardia was at the mid-septum of the right ventricle. A review of the literature failed to reveal any report of a similar case. (Jpn Heart J 1997; 38: 117–125)

Key words: Sustained monomorphic ventricular tachycardia, Reentry, Late potential, Signal-averaged electrocardiogram, Time-domain analysis, Frequency-domain analysis.

In the acute phase, especially the prehospital phase, of acute myocardial infarction (AMI), various ventricular tachyarrhythmias occur. The arrhythmias in the prehospital phase are ventricular fibrillation and ventricular tachycardia (VT). The precise mechanism of VT is difficult to determine because it often culminates in ventricular fibrillation, and precise and time-consuming electrophysiologic study (EPS) cannot be performed because early recanalization of the occluded coronary arteries is necessary. Using experimental data, Janse clearly demonstrated that the mechanism of VT that occurs soon after coronary ligation is reentry as documented by the epicardial electrogram mapping tech-
nique. In this paper, we report a case in which sustained monomorphic VT developed in the prehospital phase of AMI in which the mechanism was considered to be reentry based on the evidence of an entrainment phenomenon.2)

Case Report

A 48-year-old man was admitted to our hospital because of chest pain and palpitation. He had first developed chest pain and palpitations of sudden onset while climbing a mountain at 10:00 a.m. on March 9, 1993. He had descended the mountain and visited a local physician 7 hours after the beginning of the episode. Twelve-lead electrocardiography (ECG) (Figure 1-A) revealed monomorphic sustained VT, the morphology of which was left bundle branch block

![Figure 1](image-url)
Figure 2. Recordings of pacing from the right ventricular apex with pacing cycle lengths of 340, 310, 290 and 250 msec. No progressive fusion was seen. VT = ventricular tachycardia; RV = right ventricle; CL = cycle length.
Figure 3. Recordings of pacing from the right ventricular outflow tract with pacing cycle lengths of 340, 330, 310, 290 and 270 msec. Constant and progressive fusion were seen. This ventricular tachycardia was terminated by pacing at a cycle length of 270 msec. VT = ventricular tachycardia; RV = right ventricle; CL = cycle length.

and superior axis pattern with a cycle length of 350 msec. Creatine phosphokinase (CK) was 476 mIU, and CK-MB (an isoenzyme of CK) was 100 mIU. The physician did not think that the patient had VT, and thought that this patient had AMI based on the results of ECG and elevated cardiac enzymes. He was transferred to our hospital's cardiac catheterization room by ambulance to un-
dergo recanalization of the occluded coronary artery 9.5 hours after the onset of symptoms.

On physical examination, the patient was alert and his blood pressure was 97/37 mmHg without any laterality between the upper and lower extremities; his

\[ \text{A} \]

\[ \text{fQRS 186msec, RMS40 6} \mu \text{V} \]

\[ \text{B} \]

(1)

\[ \text{Power from 10 to 50 Hz = 320.9} \]

(2)

\[ \text{Power from 20 to 50 Hz = 142.2} \]

Area ratio = \( \frac{\text{Power of 20 to 50 Hz}}{\text{Power of 10 to 50 Hz}} \times 1000 = 444 \)

**Figure 4.** A: Signal-averaged electrocardiogram time-domain analysis. The filtered QRS duration (fQRS) was 186 msec, and the root mean square voltage in the last 40 msec of the QRS complex (RMS) was 6\( \mu \)V. B: Signal-averaged electrocardiogram frequency-domain analysis. (1) The power from 10 to 50 Hz was 320.9. (2) The power from 20 to 50 Hz was 142.2. The area ratio was calculated as follows: The power from 20 to 50 Hz was divided by that from 10 to 50 Hz and the quotient was multiplied by \( 1 \times 10^3 \). In this case, the area ratio was 444.
pulse was 170 beats/min. There were no other abnormal physical findings for any structure, including the heart. Since we thought that this patient had monomorphic sustained VT given the ECG findings, we attempted to terminate the VT by right ventricular pacing. During right ventricular apical pacing, the ECG revealed no progressive fusion (Figure 2). However, it demonstrated constant fusion and progressive fusion, and the VT was terminated by right ventricular outflow pacing with a pacing cycle length of 270 msec (Figure 3). After the return to sinus rhythm, ST elevation in the left precordial leads with right bundle branch block (RBBB) was found (Figure 1-B). Coronary arteriography (CAG) disclosed that segment #7 of the left anterior descending artery was totally occluded. Direct percutaneous transluminal coronary angioplasty (PTCA) successfully recanalized the occluded segment 10 hours after the onset of symptoms.

After PTCA his clinical course was uneventful and his rehabilitation pro-
grams were carried out without any arrhythmic or ischemic event. CAG performed on the 22nd hospital day revealed no restenosis, but left ventriculography disclosed akinesis in segments #2, 3, and 4 with 40% ejection fraction. Signal-averaged electrocardiogram was performed using the VCM-3000 apparatus (Fukuda Denshi Company, Tokyo) on the 26th hospital day. The time-domain analysis of the signal-averaged electrocardiogram revealed that the filtered QRS duration was 186 msec, and the root mean square voltage in the last 40 msec of the QRS complex was 6 μV (Figure 4-A). Because of the presence of RBBB, frequency-domain analysis of the signal-averaged electrocardiogram was performed by the method of Lindsay et al. The offset was determined as the point 40 msec earlier than the end of the QRS complex, and the onset was 120 msec earlier than the offset. The area ratio was calculated as follows: The power from 20 to 50 Hz was divided by that from 10 to 50 Hz and the quotient was multiplied by $1 \times 10^3$. The value of the area ratio was 444, which is positive according to the Lindsay criteria (> 107) (Figure 4-B). Frequency-domain analysis mapping was performed in the same manner as the time-domain analysis mapping which we had already performed (Figure 5-A). The location of the maximum area ratio value was F-4 (Figure 5-B).

EPS was performed on the 29th hospital day. During sinus rhythm, fragmented electrographic patterns according to Simson’s criteria were detected at the anteroseptal and anterosuperior sides of the left ventricle (Figure 6). Nonclinical VT was induced by the single premature stimulation of the right ventricular outflow. Because of hemodynamic collapse, VT had to be terminated.
by DC shock. After the infusion of xylocaine (100 mg), the same nonclinical VT was induced and DC shock was again needed for termination of VT because of hemodynamic collapse. Therefore, precise mapping during VT was not carried out. Based on the pace-mapping findings during sinus rhythm, the exit of clinical VT was thought to be at the mid-septal wall of the right ventricle, and the exit of the nonclinical VT was at the septal wall of the right ventricular outflow tract. In accordance with the expressed desire of the patient, no further electropharmacological study was performed.

**DISCUSSION**

VT occurs in every phase of myocardial infarction. It commonly occurs after admission to the coronary care unit and is usually monomorphic. Sustained VT originates from the border of a ventricular aneurysm. The mechanism of this VT is considered to be reentry based on the observation of an entrainment phenomenon. In contrast, the mechanism of monomorphic sustained VT that occurs in the acute prehospital phase is considered to be reentry based on experimental data. However, in reviewing the literature we found no report that clarified the mechanism of monomorphic sustained VT occurring in the prehospital phase in humans.

Waldo et al. noted that the criteria of entrainment were constant fusion without fusion of the last pacing beat with return cycle of the last pacing beat equal to the pacing cycle length, progressive fusion and local block that terminated tachyarrhythmia. In this case, as the intracardiac electrograms were not recorded, it was not documented whether the return cycle of the last pacing beat equaled the pacing cycle length. However because constant fusion and progressive fusion were documented by right ventricular outflow pacing, the mechanism of this VT was highly suspected to be reentry. In EPS performed in the chronic phase, nonclinical VT was induced by premature stimulation, and a cycle length of this VT was very short even after administration of the class IA drug. This difference between the VT cycle lengths in the acute and chronic phases might be caused by the change of conduction velocity through the slow conduction area. Because of the acute ischemia in the prehospital phase, the conduction velocity through slow conduction was slower than that in the chronic phase. Based on the pace-mapping data for chronic phase EPS, the VT occurring in the prehospital phase had a slow conduction area at the interventricular septum and the exit was the mid-septal wall of the right ventricle.

In most studies, early recanalization abolished the substrate of slow conduction, i.e. the formation of late potential (LP). In this case, in spite of successful direct PTCA, LP was positive in the left ventricle, because the time of
recanalization was late. Because of the presence of RBBB, frequency-domain analysis of signal-averaged electrocardiogram was performed. We have previously performed body surface mapping of signal-averaged electrocardiogram by the time-domain method and compared these data with intracardiac electrocardiographic data.4) The intracardiac fragmented electrogram is distributed to a certain body surface area without overlapping the site of infarction significantly. Body surface mapping is useful in predicting the site of the intracardiac fragmented electrogram before performing EPS. We performed body surface mapping of the signal-averaged electrocardiogram by the frequency-domain method as well as by the time-domain method. The maximum area ratio value was at F-4, which according to the results of the time-domain analysis corresponds to the anterosuperior site of the left ventricle.

In conclusion, a case of sustained monomorphic ventricular tachycardia developing in the prehospital phase of AMI was encountered. Its mechanism was considered to be reentry given the documented entrainment phenomenon.

REFERENCES


