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Rare Survival in a Patient With Severe Complications of Acute Myocardial Infarction: A Case Report

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Abstract

A 50-year-old man presented with acute myocardial infarction. Coronary angiography showed the left anterior descending artery (segment 6) was totally occluded. Direct percutaneous transluminal coronary angioplasty was performed, but a distal embolism occurred by the procedure. Consequently, he suffered cardiogenic shock and sustained ventricular tachycardia/fibrillation which exacerbated his condition. Finally, the patient was successfully treated with catheter ablation and cryosurgery to control the incessant ventricular arrhythmias, and partial left ventricular volume reduction and coronary artery bypass grafting to improve contractile performance. His left ventricular contractility did not improve, but the incessant ventricular arrhythmias could be controlled. His condition remarkably improved and he was discharged on foot. This patient with severe complications of acute myocardial infarction showed unusually good response and recovery.

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Key Words

■**Myocardial infarction, treatment (acute)** ■**Arrhythmias (fatal, cryosurgery)**
■**Shock (cardiogenic)** ■**Ventricular function (left ventricular volume reduction)**

INTRODUCTION

Generally, both intraaortic balloon pumping (IABP) and percutaneous cardiopulmonary support (PCPS) are effective for myocardial infarction with cardiogenic shock.[1–4] However, these treatments are not effective in patients with severe complications causing exacerbation of acute myocardial infarction. We describe a remarkable patient who survived after treatment by cryosurgery, left ventricular volume reduction, and coronary artery bypass grafting (CABG).

CASE REPORT

A 50-year-old man had a 28-year history of untreated hypertension. The patient developed anterior chest pain and electrocardiography showed ST elevations in leads Ⅰ, aV_L, V_2–V_6. Two hours later, he was transferred to our hospital with continuing chest pain. Chest radiography revealed enlargement of the cardiac silhouette and pulmonary edema (**Fig. 1**). The ST segments remained

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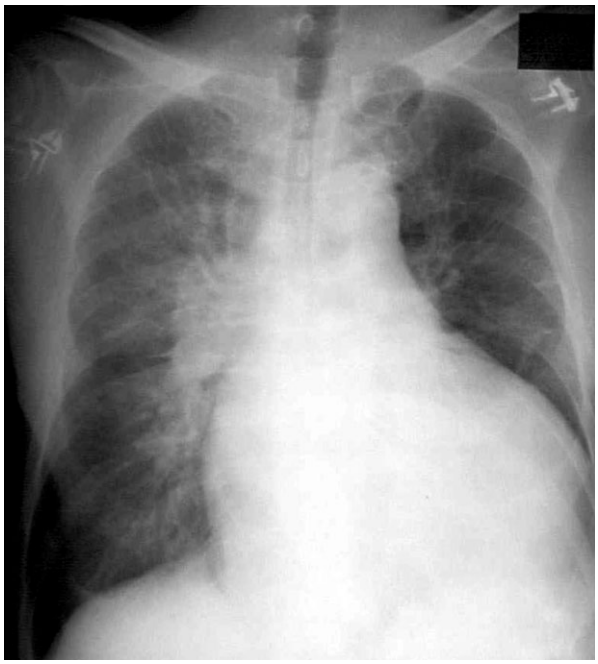

Fig. 1 Chest radiograph showing cardiomegaly (cardiothoracic ratio of 67%) and significant congestion in both lungs

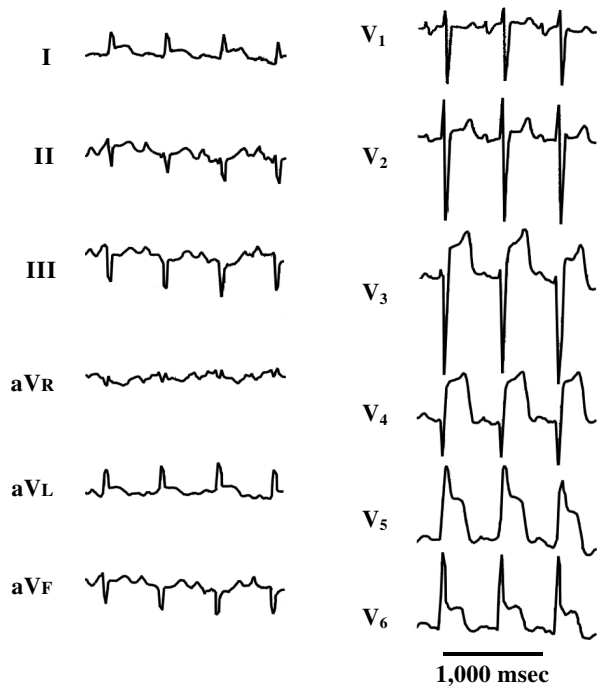

Fig. 2 Electrocardiogram showing ST segment elevation in leads I, aVL, V2–V6 with sinus tachycardia (102 beats/min)

elevated in leads I, aVL, V2–V6 (**Fig. 2**). Transthoracic echocardiography revealed akinesis at anteroseptal wall area and diffuse hypokinesis with left ventricular end-diastolic dimension (LVDd) of 87 mm, and left ventricular end-systolic dimension (LVDs) of 80 mm. The ejection fraction was 33%. Laboratory examinations showed elevation in the serum creatine kinase (CK) activity (CK: 617 IU/*l*, CK-MB: 62 IU/*l*). Based on the results of these examinations, acute myocardial infarction was suspected.

Emergent coronary angiography showed the left anterior descending artery (LAD; segment 6) was totally occluded without collateral circulation. The right coronary artery and left circumflex coronary artery were normal. During percutaneous transluminal coronary angioplasty (PTCA), a guide wire was easily passed through the culprit lesion and a PTCA balloon (3.0 mm) was inflated. However, a distal embolism was produced by the procedure. The LAD (segment 7), first diagonal branch (D1) and septal branch were almost totally occluded. Unfortunately, aspiration therapy was impossible to perform at that time. IABP was placed to treat cardiogenic shock and to increase coronary flow. The patient was treated with aspirin (81 mg/day), ticlopidine (200 mg/day), and heparin sodium (10,000 U/day). The maximum CK release was 4,196 IU/*l*.

The patient developed sustained ventricular tachycardia (VT) resulting in near-syncope on the day after PTCA (**Fig. 3**). Anti-arrhythmic agents (lidocaine: 120 mg/hr, amiodarone: 800 mg/day) were administered to treat, and the defibrillator was used, but were not effective and his vital signs were exacerbated. A PCPS was initiated with IABP (PCPS at 4 *l*/min). The blood pressure was maintained at 80 mmHg, with a cardiac output of 1.9 *l*/min and a mean pulmonary capillary wedge pressure of 19 mmHg. However, the sustained VT persisted and the patient developed sustained ventricular fibrillation (VF). The VT and VF continued for 2 days. The sustained VF resulted in low cardiac output and multiple organ failure. Blood examination found hemoglobin 10.3 g/d*l*, platelet count was $6.0 \times 10^4/\mu l$, blood urea nitrogen was 65 mg/d*l*, and serum creatinine concentration was 2.5 mg/d*l*.

Four days after admission, an electrophysiological study to determine the focus of the VT showed the earliest electrical potential originated from the region from the septum to the middle inferior wall, and almost perfect mapping was shown (**Fig. 4**).

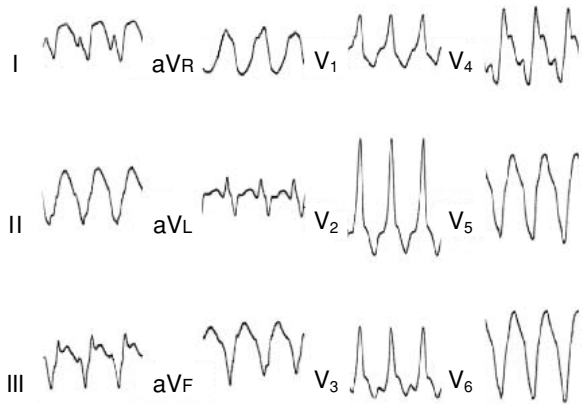

Fig. 3 Electrocardiogram showing sustained ventricular tachycardia with complete right bundle branch block and left axis deviation pattern
Cycle length is 280 msec.

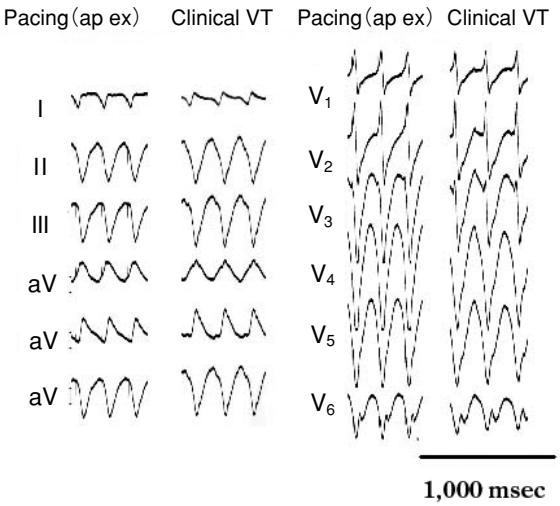

Fig. 4 Left ventricular pace mapping showing almost perfect mapping
In this position, entrainment phenomenon is observed (not shown).
VT = ventricular tachycardia.

Then catheter ablation was performed. Unfortunately, it was impossible to completely inhibit the incessant potentially fatal arrhythmias.

Finally, the following strategy was decided. We decided to perform cryosurgery to treat the incessant arrhythmias, then to perform left ventricular volume reduction for wall motion improvement, and finally to perform CABG. Accordingly, left ventricular mapping was performed again, and cryosurgery was performed to treat the sustained VT for 180 sec from the left ventricular septum to

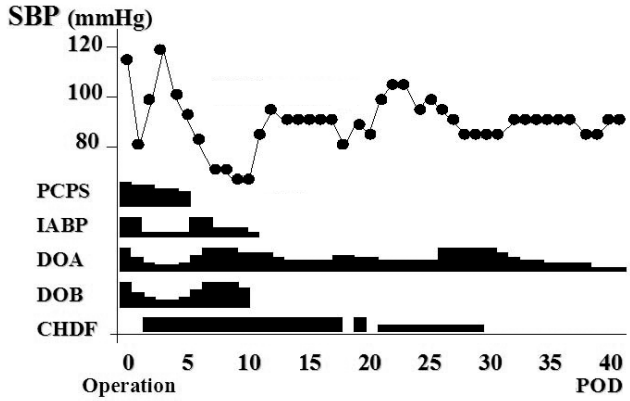

Fig. 5 Postoperative clinical course
After cryosurgery, partial left ventricular volume reduction, and coronary artery bypass grafting operation, PCPS could be stopped on the fourth postoperative day. Inotropic agents could be discontinued on the 40th postoperative day.
SBP = systolic blood pressure; PCPS = percutaneous cardiopulmonary support; IABP = intraaortic balloon pumping; DOA = dopamine; DOB = dobutamine; CHDF = continuous hemodiafiltration; POD = postoperative day.

the inferior wall. Moreover, antiarrhythmic agents (amiodarone: 50 mg/day and d,l-sotalol: 80 mg/day) were also administered. The cryosurgery focus of the sustained VT was marked in catheter ablation, and was used as a medical guideline. The sustained VT and VF were completely ameliorated. Partial left ventricular volume reduction surgery was successfully performed. We appropriately excised the myocardium detected to be the infarct area. However, the border of the acutely infarcted myocardium was impossible to differentiate accurately, because it also contained an area of very recent infarction. Postoperative measurement of transthoracic echocardiography revealed LVDd of 70 mm, LVDs of 59 mm, and ejection fraction of 33%, and left ventricular contraction was impossible to improve. The patient suffered from hypertension, but there was no evidence to show hypertensive heart disease. Endomyocadial biopsy found only accumulation of neutrophile and fibrosis. CABG was also performed using a saphenous vein grafted to the LAD.

The postoperative clinical course for this patient is summarized in **Fig. 5**. Seven months after admission, repeat coronary angiography was performed. The LAD (segment 7), D1 and septal branch were totally occluded, as before. However, the bypass graft was patent. Left ventriculography revealed

severe diffuse hypokinesis with apical aneurysm. The ejection fraction was 22%. Electrophysiological study was also performed, but no potentially fatal arrhythmias could be induced by frequent right ventricular stimulation.

DISCUSSION

This patient presented with severe acute myocardial infarction. He suffered cardiogenic shock and incessant potentially fatal arrhythmias after treating the acute myocardial infarction. Generally, both IABP and PCPS are effective treatments for myocardial infarction with cardiogenic shock.[1-4] However, they were not effective for this patient and his physical condition was exacerbated, because the incessant arrhythmias and cardiogenic shock were sustained. Our initial treatment was PTCA, which is a standard treatment for acute myocardial infarction. However, PTCA was unsuccessful due to distal embolism, and was the main reason for this patient to fall into this miserable state.

As a result, we were obliged to follow a double strategy. Firstly, cryosurgery was performed to treating the incessant arrhythmias and both partial left ventricular volume reduction and CABG were performed to treat the cardiogenic shock. The most effective treatment was cryosurgery, which controlled the incessant arrhythmias immediately. Arrhythmias after acute myocardial infarction have been treated by catheter ablation in the treatment of VT in selected patients with healed myocardial infarction.[5-9] Catheter ablation can now be used to fatal arrhythmias caused by non-healed myocardial infarction, but few case reports have described fatal arrhythmias with non-healed myocardial infarction.[10,11] In our patient, mapping of the VT focus showed that cryosurgery would be effective.

Given that partial left ventricular volume reduction was performed in the subacute phase of myocardial infarction, the extensive dyskinetic area of the myocardium was impossible to reduce completely. In the chronic phase, left ventricular contractility did not improve despite partial left ventricular volume reduction and patent coronary bypass graft. This showed the border of the acutely infarcted myocardium was impossible to differentiate accurately. Whether partial left ventricular volume reduction was effective is doubtful. On the other hand, our patient came out of cardiogenic shock, and survived without PCPS and IABP. We could not use aspiration therapy and nifekalant, which are common treatments for such a patient. However, we believed that our treatment was the best at that time and his survival depended on all medical treatments.

This case indicates that the first principle is to be able to control incessant fatal arrhythmias, the second principle is to improve cardiogenic shock using partial left ventricular volume reduction and CABG. Our patient had suffered from incessant arrhythmias and multiple organ failure for several days, but survived and was discharged on foot. This is a remarkable survival in a patient with severe complications of acute myocardial infarction treated by cryosurgery, left ventricular volume reduction, and CABG.

要　約

重度合併症を発症した急性心筋梗塞患者救命の1例

高間　典明　　星崎　洋　　内藤　滋人

大島　茂　　谷口　興一　　倉林　正彦

症例は急性心筋梗塞に罹患した50歳，男性．心臓カテーテル検査では左前下行枝(分節 6)が完全閉塞であった．経皮的冠動脈形成術を施行したものの，最終的には遠位部に血栓性閉塞をきたした．その結果，心原性ショックと持続性心室頻拍ならびに心室細動が出現し状態悪化の原因となった．最終的には不整脈をコントロールする目的で，カテーテルアブレーションならびに冷凍外科療法を施行し，心機能改善する目的で左室縮小術と冠動脈バイパス術を施行した．心機能の改善は認められなかったが，持続性の心室性不整脈はコントロールすることができた．臨床経過は著明に改善し，最終的には独歩で退院することができた．急性心筋梗塞に伴う重度合併症を持つ症例を救命することができたので報告した．

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