Clusters of Ventricular Fibrillation in a Patient With an Implantable Cardioverter Defibrillator Treated With Amiodarone

Tetsuro Emori, MD; Keiko Ohta, MD; Kengo Kusano, MD; Hiroshi Morita, MD; Hiromi Matsubara, MD; Shunji Sano, MD*; Tohru Ohe, MD

A 44 year-old man with severe left ventricular dysfunction resulting from an old myocardial infarction developed clusters of ventricular fibrillation (VF). Although coronary bypass surgery was performed and heart failure was well controlled, the VF recurred during amiodarone therapy. Despite multiple deliveries of shocks by an implantable cardioverter defibrillator, the electrical storm could not be terminated. Some substrate for rapid ventricular tachyarrhythmias, refractory to class III drugs, can lead to death from arrhythmia. (Circ J 2003; 67: 163–165)

Key Words: Electrical storm; Implantable cardioverter defibrillator

Implantable cardioverter defibrillators (ICDs) have a high success rate in terminating ventricular tachycardia and ventricular fibrillation (VF), although studies have shown that 10% of patients with ICD receive multiple shocks during a short period of time, which is one of the most demanding clinical problems during the follow-up of an ICD patient. This condition is known as arrhythmic or electrical storm and class III agents, amiodarone, sotalol and β-blockers, should prevent such an arrhythmic storm.1,2

We report on a patient who developed intractable arrhythmic storm despite amiodarone therapy.

Case Report

A 43-year-old man was admitted to the emergency room because of cardiac arrest. Cardiopulmonary resuscitation was performed and VF recovered to normal sinus rhythm after cardioversion with 300 J. The chest X-ray showed cardiomegaly (cardiothoracic ratio, 62%) and the electrocardiogram (ECG) recorded after conversion to sinus rhythm showed a QS pattern in the precordial leads compatible with the presence of an old anterior myocardial infarction. The QTc interval was 0.44 s and no significant ST change was observed. The pulmonary capillary wedge pressure was 8 mmHg and the cardiac index was 3.0 L·min⁻¹·m⁻². Mild metabolic acidosis was corrected by the infusion of sodium bicarbonate and the serum potassium level was kept at 4.0 mmol/L or higher. However, VF recurred incessantly requiring 30 episodes of DC shocks for termination in spite of the continuous infusion of magnesium sulfate and amiodarone (Fig 1). Emergency coronary angiography revealed stenoses and occlusion at multiple sites of the major arteries: total occlusion of the left anterior descending artery, subtotal occlusion (90%) of the circumflex artery and 75% stenosis at the proximal right coronary artery.

The patient had been treated for schizophrenia since 13

Fig 1. The incessant recurrent ventricular fibrillation during 15 min of continuous ECG recording was successfully terminated by external DC shocks.

(Received September 7, 2001; revised manuscript received November 16, 2001; accepted December 13, 2001)

Departments of Cardiovascular Medicine and *Cardiovascular Surgery, Okayama University Graduate School of Medicine and Dentistry, Okayama, Japan

Mailing address: Tetsuro Emori, MD, Department of Cardiovascular Medicine, Okayama University Graduate School of Medicine and Dentistry, 2-5-1 Shikata-cho, Okayama 700-8558, Japan. E-mail: emorit@md.okayama-u.ac.jp
years of age and had been admitted to other hospital because of congestive heart failure 2 years ago. Coronary risk factors included smoking and poorly controlled diabetes (hemoglobin A1c = 8.2%). Echocardiography during the previous admission revealed a segmental abnormality of the left ventricular (LV) wall compatible with an old extensive anterior myocardial infarction as well as diffusely reduced contraction of the posterior and inferior walls. The LV diastolic dimension, systolic dimension and ejection fraction were 68 mm, 59 mm and 29%, respectively. Diuretics and carvedilol were started.

After the initial intensive therapy, 200 mg of amiodarone and 300 mg of mexiletine were started on the 5th day of admission. His hemodynamic condition was stable and serum electrolytes were normal. The VF episodes decreased spontaneously, but clusters still developed every 3 or 4 days for 1 month in spite of the oral amiodarone therapy. Coronary bypass surgery was performed to improve the myocardial ischemia of the inferior and posterior walls and prevent VF: a saphenous vein graft was bridged between the aorta and posterior descending artery, and the left internal thoracic artery was extended by a free radial graft and connected to the left circumflex artery. However, VF recurred after coronary bypass surgery and so an ICD (Medtronic MICRO JWELLTM II) was implanted. Tachycardia with a cycle length less than 320 ms was defined as VF and cardioversion with 30 J was set up for therapy. He was transferred to a hospital for rehabilitation and psychiatric therapy after coronary angiography performed 1 month postoperatively confirmed that the coronary bypass grafts were patent. Myocardial scintigraphy with dipyridamole showed that there was no ischemia but there was a fixed perfusion defect in the anterior wall and reduced uptake in the inferior and posterior walls. Drug therapy for cardiac arrhythmia and heart failure (200 mg amiodarone, 300 mg mexiletine, 5 mg carvedilol, 4 mg candesartan, 80 mg furosemide, and 100 mg spironolactone) as well as for schizophrenia (1.5 mg haloperidol and 1 mg biperiden), which was the same prescription used in the university hospital has continued. Diabetes was well controlled by diet without medication.

He did not complain of any symptoms but he was in an apathetic state of schizophrenia. There was no sign of congestive heart failure or myocardial ischemia by physical examination, ECG or chest X-rays carried out at during the regular monthly examination. The chest X-rays showed a gradual decrease in the cardiothoracic ratio, reaching 55% by the 16th month after the first arrhythmic storm. He stayed at the hospital and his medication was completely managed by nurses. The QT interval was not prolonged despite amiodarone therapy of 200 mg a day (QTc = 0.45 s) (Fig 2). The blood concentration of amiodarone was not evaluated but the serum level of thyroid stimulating hormone was persistently between 6.0 and 8.0 μU/ml, indicating amiodarone-induced hypothyroidism. A proarrhythmic effect of the major tranquilizer was not seen in conjunction with electrolyte disturbance or amiodarone. He developed several VF clusters in February and May 2000 without apparent triggering factors (Fig 3), and once the VF developed, it recurred incessantly for 5 to 10 minutes but then the arrhythmic storm spontaneously lost its force without requiring any additional medication. He had only 3 isolated episodes of VF, which were successfully terminated by the first ICD shock of 30 J and was in a stable condition since May 2000. In the 16th month after the first arrhythmic storm, a total of 63 ICD shocks were incessantly delivered within 50 min. Although each VF episode was successfully terminated by the first or second shock, the recurrent VF cycle length exceeded the initially programmed VF detection interval, finally resulting in the cessation of ICD therapy (Fig 4). The VF could not be converted by external cardioversion and he suffered a cardiac arrest despite cardiopulmonary resuscitation attempts.

**Discussion**

Even in the era of epicardial patch electrodes with thoracotomy devices, a postoperative arrhythmic storm is a serious complications in 10% of patients with an ICD! In the majority of cases, myocardial ischemia, congestive heart failure, electrolyte disturbance or drug intoxication is the
triggering factor. Although the present patient had severely depressed left ventricular function, he never developed congestive heart failure during the arrhythmic storm and a gradual decrease in the cardiothoracic ratio indicated a stable compensated state. Thus, we consider that ischemic myocardium of the inferior and posterior walls might have been the substrate for VF because 1 month of ral amiodarone therapy did not prolong the cycle length of the arrhythmia or suppress the VF cluster. However, coronary bypass surgery failed to suppress the development of VF. Increased sympathetic activity is also known to contribute to arrhythmic storms and although there was no direct evidence, adrenergic drive seems a possible cause for the VF clusters in this patient, because no other triggering factor was detected. The VF was concentrated into a short span of time and the arrhythmic storm spontaneously lost its force without any additional medication especially later after his transfer, suggesting some functional modulating factors such as autonomic tone. In the last VF cluster, which differed from the other VF episodes in the late period, the VF recurred persistently. Congestive heart failure and the proarrhythmic effects of drugs can be eliminated as underlying factors because there no significant change was detected at the regular examination immediately before the last VF cluster. Amiodarone or β-blockers are generally accepted as the best available drugs for prevention of arrhythmic storm. A class III agent, sotalol, has been reported to be effective in reducing the risk of death and the delivery of ICD shocks in a randomized trial? The clinical course of the present patient can be divided into 2 parts: the early period during which intensive care was required for frequent VF clusters until his transfer at 2 months after amiodarone therapy began, and the late period in which VF clusters were less frequent. The relatively calm state that existed until the last VF cluster in the late period can partly be attributed to amiodarone therapy, although electrical instability remained. The slight prolongation of the QT interval after the introduction of amiodarone suggests the existence of an area of myocardium with short refractoriness, which became a fixed substrate for intractable ventricular tachyarrhythmias. It is possible that the dose of amiodarone was not enough to suppress the ventricular tachyarrhythmias. However, considering the patient’s light body weight (40 kg) and severe left ventricular dysfunction, the dose of 200 mg was considered sufficient and the subclinical hypothyroidism would seem to support that, although the blood concentration of amiodarone was not evaluated.

This case warns us that an intractable arrhythmic storm, which cannot be managed by amiodarone, is a serious side effect in the era of increasing use of ICD.

References