CASE REPORT

Increase in Internal Defibrillation Threshold During Acute Myocardial Infarction

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Abstract

A 67-year-old man suffered an acute anteroseptal myocardial infarction complicated by multiple episodes of ventricular fibrillation, which were not systematically defibrillated by maximum, internal 35-J shocks delivered by an implanted cardioverter defibrillator (ICD). He had suffered from acute inferior myocardial infarction 6 years earlier, complicated with sustained polymorphic ventricular tachycardia (VT). Due to inducibility of sustained VT on an electrophysiologic study, an ICD was implanted. Defibrillation testing performed after healing of anteroseptal infarction was successful with a 10-J safety margin, suggesting that acute myocardial ischemia transiently elevated the internal defibrillation threshold.

Key words: defibrillation threshold, acute myocardial infarction, myocardial ischemia, implantable cardioverter-defibrillator


Introduction

Several experimental studies have suggested that acute myocardial ischemia increases the ventricular defibrillation threshold (DFT) (1). However, few studies of the internal DFT during myocardial ischemia in humans have been published. We report a case of transient elevation of the DFT during the acute phase of myocardial infarction in a recipient of implantable cardioverter defibrillator (ICD).

Case Report

A 67-year-old man was admitted to our hospital after the sudden onset of anterior chest pain, followed by 7 shocks delivered by a GEM 7227 single chamber ICD (Medtronic Inc., Minneapolis, MN). He had suffered from acute inferior myocardial infarction 6 years earlier. Reperfusion of the proximal right coronary artery was unsuccessful with emergency percutaneous coronary intervention. Since sustained polymorphic ventricular tachycardia (VT) without unconsciousness developed on the 6th, 8th and 12th day after onset (Fig. 1), oral administration of amiodarone was initiated.

Left ventriculography revealed akinesis of inferior wall motion and almost normal systolic function with left ventricular ejection fraction of 62%. Electrophysiologic study under the administration of amiodarone demonstrated the inducibility of polymorphic VT (Fig. 2), suggesting that the mechanism of the VT might be scar-related reentry. Thus, the pulse generator and an integrated, screw-in, Sprint model 6943-65 single-coil lead (Medtronic) introduced via the left subclavian vein, with its tip placed at the right ventricular apex, had been implanted. The device delivered maximum 35 J biphasic shocks with 65%/65% tilt, through a 120-μF capacitance. Defibrillation testing at the time of implantation was successful with a minimum 10-J safety margin. Treatment with amiodarone, 200 mg p.o. daily, continued until this episode.

On admission, a 12-lead electrocardiogram showed elevation of the ST segment in leads I, aVL, and V1-V6, consistent with acute anteroseptal and antero-lateral myocardial infarction (Fig. 3). Emergent coronary angiography showed a total occlusion of the proximal left anterior descending artery, which was successfully treated by coronary angioplasty approximately 2 h after the onset of chest pain. The peak serum concentration of creatine phosphokinase was 12,264 IU/l. Interrogation of the ICD revealed a total of 5 episodes of
Figure 1. 12-lead ECG during polymorphic ventricular tachycardia. Note spontaneous termination of the tachycardia during recording of lead V1,3.

Figure 2. Induction of polymorphic ventricular tachycardia following single extra stimulus of a S1-S2 coupling interval of 210 ms with a S1-S1 basic cycle length of 400 ms delivered from right ventricular outflow tract (RVOF). I, II and V1 = 12-lead ECG, 3-4 to 1-2 HBE = the proximal to distal His-bundle electrogram.
ventricular defibrillation occurring over an 18 minutes period, starting approximately 30 minutes after the onset of chest pain (Fig. 4). The 2nd, 4th and 5th episodes were successfully defibrillated with a single 35-J shock, and an active can/superior vena cava coil → right ventricular coil current vector. However, the 1st and 3rd episodes were not terminated with a first 35-J shock, though were successfully defibrillated with a second 35-J shock delivered with a current vector in the reverse direction (Fig. 5), suggesting that the DFT was approximately 35 J. Defibrillation testing performed 111th day after the onset of anteroseptal myocardial infarction confirmed a minimum 10-J safety margin, suggesting that acute myocardial ischemia had transiently elevated the DFT. Left ventriculography performed 2 years after the onset of anterior myocardial infarction revealed akinesia of anterior and apical wall motion, severe hypokinesis of inferior wall motion, moderate hypokinesis of septal wall motion and moderately reduced systolic function with left ventricular ejection fraction of 47.7%. It is noteworthy that neither was amiodarone discontinued, nor was the shock polarity reversed or other electrodes added, in an attempt to lower the DFT.

**Discussion**

This case showed the occurrence of a transient increase in the energy required to defibrillate the heart with an ICD during the acute phase of a myocardial infarction. It has been suggested that the two mechanisms may cause failure to defibrillate (2-4). First, the shock may have been too weak to interrupt the fibrillatory wave fronts. Second, VF may have been terminated, but a new episode may have been initiated by a critical shock strength or a focal mechanism. An insufficient shock strength would explain the immediate, though not the delayed recurrence of VF. Both mechanisms are possible to explain the increase in DFT in the present case.

In contrast to the several reports of successful ICD treatment of VF complicating acute ischemia (5, 6), the present case suggests that, while a first ICD shock delivered for VF at an energy 10 J below maximum may be successful under stable testing conditions, it may not be strong enough during acute ischemia. It further suggests that, in ICD recipients suffering from coronary artery disease, special measures may be needed, such as the implant of a high energy device, the insertion of an additional lead, or special programmable functions for high DFT.

Electrical storm of ventricular fibrillation occasionally develops in the acute phase of myocardial infarction. Acute ischemia may be one of the factors responsible for VF in-

![Figure 4](image-url)
Figure 5. Interrogation of the 1st episode of ventricular tachyarrhythmia stored in the ICD.
A. The first 35-J shock (*) fails to terminate VF. B. The second 35-J shock (**) terminates VF.

Initiation, since it can induce several facilitating changes, such as increased sympathetic activity (7, 8), hyperkalemia (9), and acidosis (10), which increase ventricular vulnerability. Recent reports have highlighted a genetic preposition to the development of arrhythmia associated with acute myocardial infarction. Hu et al described sodium channel mutation to be associated with the development of an arrhythmic storm during acute ischemia (11). Interestingly, all VF episodes were associated with ST-segment changes and were initiated by short-coupled extrasystoles, similar to those in the present case. Furthermore, we speculate that the development of recurrent VF in the present case might have been associated with global myocardial ischemia which was caused by acute myocardial ischemia in one major territory of the coronary artery in addition to healed myocardial infarction in another major territory.

References