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Case Report

Risk of defibrillation threshold testing in severe heart failure patient: A case of cardiac resynchronization therapy (CRT-D) with acute myocardial infarction

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ABSTRACT

Defibrillation threshold (DFT) testing is usually recommended after device implantation to confirm appropriate implantable cardioverter defibrillator (ICD)/cardiac resynchronization therapy defibrillator (CRT-D) function [1,2]. However, induction of ventricular fibrillation may result in hemodynamic compromise, and cardioversion itself may cause myocardial injury [3,4]. We report on a CRT-D patient with acute myocardial infarction who died due to multiple organ failure 1 day after DFT testing. Our case emphasizes the importance of deciding whether DFT testing should be performed for patients with very severe heart failure in the acute stage of myocardial infarction.

1. Case report

A 59-year-old male was referred to our hospital in cardiogenic shock. A 12-lead electrocardiogram showed abnormal Q-waves in V1–V4 leads and complete atrioventricular block; the patient was diagnosed with acute myocardial infarction (AMI) (Fig. 1). Upon arrival, the patient was unconscious, and his blood pressure was 60 mmHg; therefore, endotracheal intubation and ventilation were immediately performed. Emergency percutaneous coronary intervention (PCI) was performed under temporary pacing for the left main coronary trunk (75% stenosis→0%) and left anterior descending artery (90% stenosis→0%). Left ventricular wall motion showed severe hypokinesis at the anteroseptal region with an ejection fraction of 33%. Sinus rhythm was restored after PCI; however, atrioventricular block frequently occurred, and we considered the patient's condition an indication for permanent pacemaker implantation. Echocardiography showed severely impaired left ventricular wall motion with low EF of 33.6% and cardiac dyssynchrony. The QRS duration was widened to 145 ms and showed a right bundle branch block configuration. The patient was on β-blockade, ACE-inhibitor, and amiodarone. Mexiletine was also given intravenously for frequent ventricular tachycardia (VT). Despite the use of these antiarrhythmic agents, VT still occurred frequently and cardioversion was performed several times. Cardiac resynchronization therapy defibrillator (CRT-D) therapy was selected for this patient and the device was implanted on June 3, 2008, 1 week after PCI. We performed this implantation in the acute stage of MI because the patient had an advanced AV block and needed pacing therapy. At first, the right ventricular (RV) lead was inserted into RV apex. However, RV sensing was not satisfactory (<3.0 mV) and only the RV septum area yielded a slightly better result. After insertion of the RV lead, we performed coronary venography and selected a preferred site for LV pacing in the posterior coronary vein. Ultimately, the atrial pacing lead was located in the right atrial (RA) appendage with sensing 4.1 mV, the RV pacing lead was located in the ventricular septum area with sensing 4.3 mV, and the LV pacing lead was located in the posterior coronary vein (generator: Medtronic CONCERTO C154DWK; RV lead: 6947 Sprint Quatro Secure; LV lead: 4194 Attain; right atrial lead: 5554 CapSure). The RA pacing threshold was 0.2 V at 0.5 ms, RV pacing threshold was 0.6 V at 0.5 ms, and LV pacing threshold was 1.9 V at 0.5 ms without phrenic nerve stimulation. Defibrillation threshold (DFT) testing was not performed because the patient had not fully recovered after PCI. The patient's condition improved gradually after CRT-D implantation and follow up CAG was performed on June 7, 2008. No significant stenosis was seen, including in the region of previous PCI. Echocardiography 1 week after CRT-D implantation showed a slightly increased EF of 41%. On June 11, 2008, DFT testing was planned to confirm the CRT-D system. Interrogation data did not show remarkable change compared to the data at implantation. Ventricular fibrillation (VF) was induced by *T* wave shock; however, defibrillation was not achieved due to VF undersensing (under the setting of RV sensing, 1.2 mV), and sinus rhythm was manually restored after 30–60 s with a shock of 20 J (Fig. 2). We repeated DFT testing after

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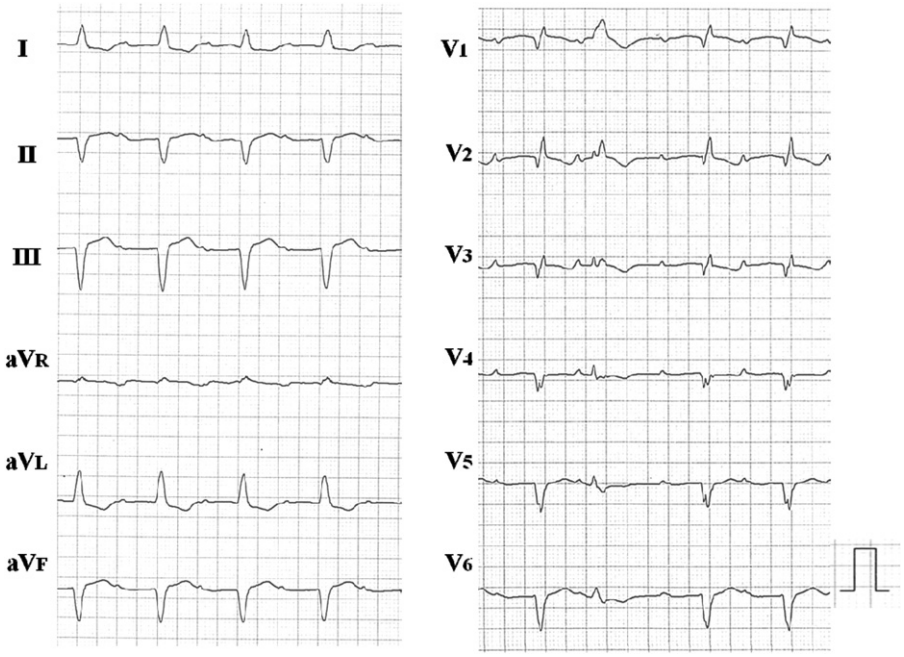

Fig. 1. A 12-lead electrocardiogram after cardioversion showing complete atrioventricular block.

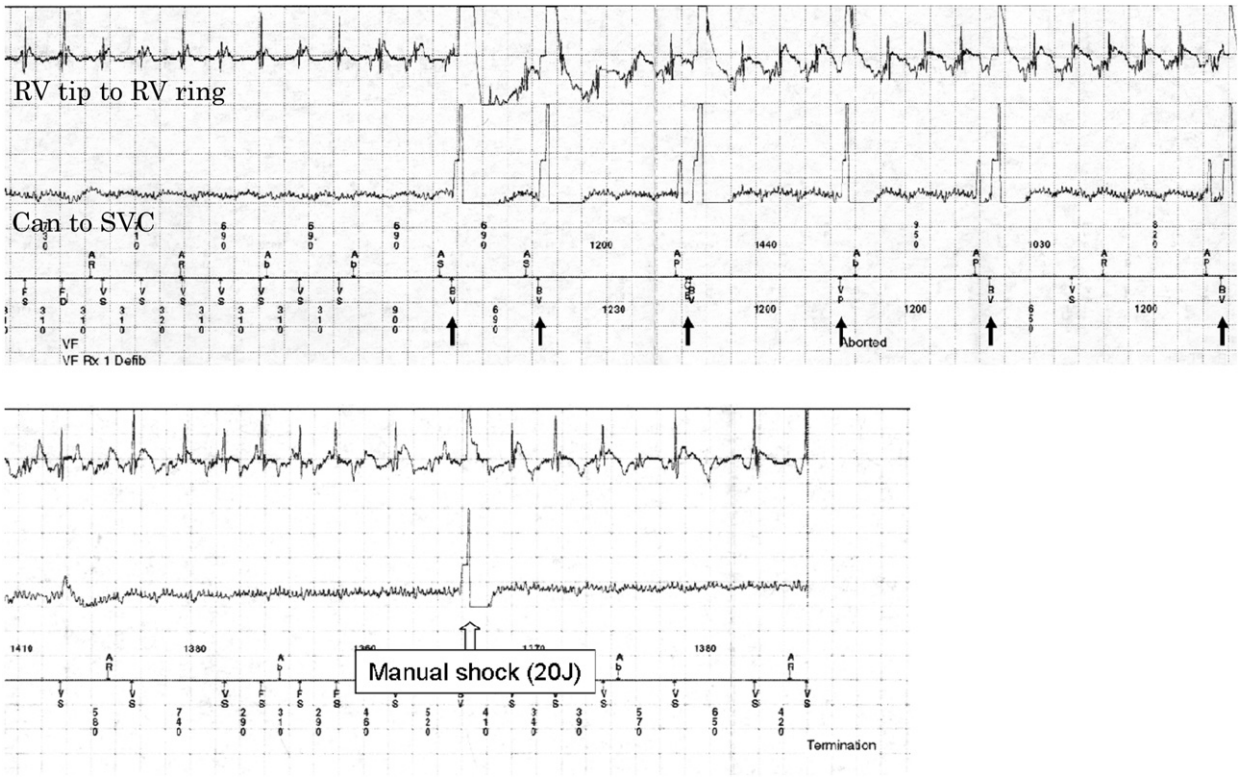

Fig. 2. An intracardiac electrocardiogram recorded during DFT testing. Biventricular pacing was performed (↑BV) because of VF undersensing, and manual shock was delivered (⇑).

changing the RV sensing sensitivity from 1.2 to 0.6 mV. Fine VF wave sensing was obtained, and VF was successfully converted with a shock of 20 J. After DFT testing, the patient complained of headache and nausea, and his blood pressure dropped to 60 mm Hg. Chest X-ray showed significant congestion of the lungs and cardiomegaly. Echocardiography showed severely impaired LV wall motion compared to the results before DFT testing (Fig. 3). One day after DFT testing, laboratory data confirmed multiple organ failure (Table 1). Brain, chest, and abdominal CTs were obtained, but there was no evidence of cerebral infarction, hemorrhage, or aortic dissection. The hemodynamic status and clinical condition of the patient did not improve, and he died after 2 days.

2. Discussion

DFT testing is usually recommended to ensure proper functioning of the ICD/CRT-D and verify that there is no acute lead

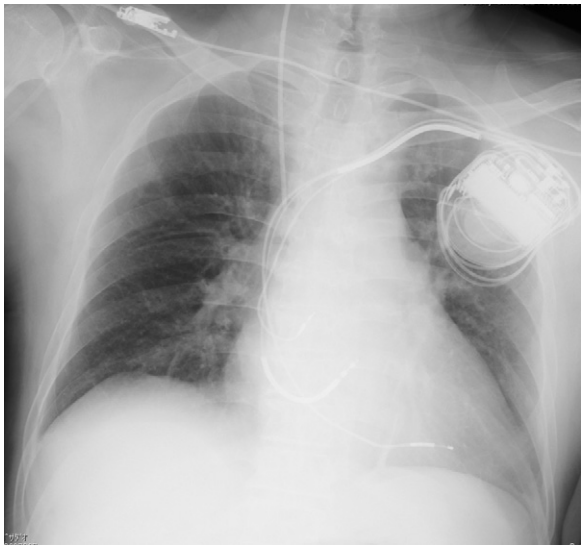

Fig. 3. Chest X-ray after CRT-D implantation. Leads were located in RAA, RV septum, and posterior coronary vein.

Table 1
Laboratory data before and after DFT testing.

Test (reference range)	2008/6/9 (Before DFT testing)	2008/6/12 (1 day after DFT testing)
WBC ($4.08.0 \times 10^9$/L)	13.8	15.4
HBG (14.0–17.0 g/dL)	11.4	10.8
PLT ($150–350 \times 10^9$/L)	363	355
BUN (8.0–19.0 mg/dL)	31.4	51.7
Creatinine (0.80–1.30 mg/L)	1.13	1.40
T-Bil (0.30–1.20 mg/dL)	1.32	1.21
AST (8–38 U/L)	81	1252
ALT (4–44 U/L)	57	1138
LDH (106–220 U/L)	647	2739
CK (54–253 U/L)	637	662
CK-MB ($<25\%$)	43	34
CRP (<0.20 mg/dL)	1.65	4.68
FDP (<10 μg/mL)	17.0	32.8
D-dimer (<1.0 μg/mL)	4.1	14.2

WBC: white blood cell count; HBG: hemoglobin; PLT: platelet; BUN: blood urea nitrogen; T-Bil: total bilirubin; AST: aspartate aminotransferase; ALT: alamine aminotransferase; LDH: lactate dehydrogenase; CK: creatinine kinase; CK-MB: creatinine kinase MB isoenzyme; CRT: C-reactive protein; FDP: fibrin degradation products.

dislodgement [1,2]. For patients with impaired cardiac function, VF induction and shock delivery can cause an unstable hemodynamic condition and clinical deterioration [3,4]. Gasparini et al. proposed delayed DFT testing for patients with impaired left ventricular function with a CRT-D [5]. The benefits of delayed DFT testing are listed as follows: patients with heart failure treated with CRT-D show marked clinical improvement, and the risk of lead dislodgement is also reduced. The data obtained from the Sudden Cardiac Death in Heart Failure Trial (SCD-HeFT) have shown that DFT testing is not beneficial for prediction of long-term mortality or shock efficacy [6], and there are several reports proposing that there is some uncertainty regarding DFT testing in patients with severe heart failure from the point of view of safety [7,8]. Bianchi et al. compared clinical outcomes between 2 patient groups: those who underwent DFT testing and those who did not, and they also demonstrated that there was no significant difference in the clinical outcome for 2 years after ICD implantation [9].

We encountered several difficulties in our case. First, the patient was recovering after myocardial infarction and cardiogenic shock, and he might have been unable to tolerate DFT testing. Current ACC/AHA/ESC guidelines recommend a delay of 40 days as the minimum time prior to ICD implantation [10]. These guidelines are based on the results of the DINAMIT trial [11], which evaluated the effectiveness of early implantation (6–40 days after AMI) and failed to demonstrate the efficacy of ICD therapy to reduce total mortality. However, there remains a possibility of significant reduction of arrhythmic death in some patients in the early period after AMI. Second, shock delivery was delayed during DFT testing because of VF undersensing, and it took some time to restore sinus rhythm. Third, we repeated DFT testing after changing the RV sensing sensitivity; this extended DFT testing may have led to hemodynamic instability for this patient, and his clinical condition deteriorated rapidly into fatal cardiogenic shock. There might have been preferable options for this patient, such as: (1) not performing the DFT testing; (2) postponing the DFT testing until the patient recovered completely after the AMI; (3) maintaining the temporary pacing until the clinical condition was more stable and then implanting the ICD or CRT-D. This case served as an important warning on the management of CRT-D in patients with severe heart failure.

In conclusion, our case emphasizes the importance of the decision whether or not to perform DFT testing as a routine protocol for patients with severe heart failure. Careful consideration must be given to whether DFT testing should be performed.

Conflict of interest

All authors have no conflicts of interest to declare.

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