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Case Report

Anti-tachycardia pacing degenerated fast ventricular tachycardia into undetectable life-threatening tachyarrhythmia in a patient with non-ischemic dilated cardiomyopathy

Kei Yunoki (MD)[a,*], Nobuhiro Nishii (MD)[a], Hiroshi Morita (MD)[a], Jun Iwasaki (MD)[b], Satoshi Nagase (MD)[a], Kazufumi Nakamura (MD)[a], Yoshiki Hata (MD)[a], Kunihisa Kohno (MD)[a], Kengo F. Kusano (MD, FJCC)[a], Hiroshi Ito (MD, FJCC)[a]

[a] *Department of Cardiovascular Medicine, Okayama University Graduate School of Medicine, 2-5-1, Shikata-cho, Kita-ku, Okayama City, Okayama 700-8558, Japan*
[b] *Department of Cardiology, Iwakuni Medical Center, Yamaguchi, Japan*

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Summary A 45-year-old man with dilated cardiomyopathy was admitted to our hospital due to congestive heart failure (CHF). Despite the optimal medical treatment, his condition had not improved because of severe left ventricular dysfunction. Because he experienced non-sustained ventricular tachycardia (VT), a biventricular implantable cardioverter-defibrillator (Bi-V ICD) was implanted for reduction of dyssynchrony and primary prevention of lethal tachyarrhythmia. After discharge, he developed CHF and was transported to our hospital by ambulance. In the ambulance, monomorphic sustained VT with 200 bpm suddenly occurred. The ICD detected it as fast VT and anti-tachycardia pacing (ATP) was delivered. After the ATP therapy, RR intervals of VT became irregular and prolonged. Ventricular fibrillation-like electrical activity was recorded by a far-field electrogram from the defibrillator, but the tachycardia cycle length exceeded 400 ms which is under the tachycardia detection rate. The device failed to deliver a shock and the patient had to be rescued with an external shock. This is a rare case of fast VT that degenerated into undetectable life-threatening tachyarrhythmia by ATP.

Introduction

An implantable cardioverter-defibrillator (ICD) has become widely used for therapy in various patients with high-risk arrhythmia and reduced mortality in primary and secondary

* Corresponding author.
Tel.: +81 86 235 7351; fax: +81 86 235 7353.
E-mail address: yunokei@yahoo.co.jp (K. Yunoki).

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Table 1 Implantable cardioverter-defibrillator parameters.

	Pacing threshold at implantation (3 months follow-up)	Sensing threshold at implantation (3 months follow-up)	Lead impedance at implantation (3 months follow-up)
Atrial lead	0.8 V/0.5 ms (0.8 V/0.5 ms)	4.1 mV (2.0 mV)	529 Ω (506 Ω)
RV lead	0.8 V/0.5 ms (1.0 V/0.5 ms)	10.8 mV (7.0 mV)	763 Ω (767 Ω)
LV lead	1.2 V/0.5 ms (1.2 V/0.5 ms)	7.9 mV (6.5 mV)	876 Ω (907 Ω)

Detection	Threshold	Detect beats	Duration	Therapies
VF	220 bpm	8 of 10	1 s	41J × 6
FVT (via VF)	180 bpm	8 of 10	2.5 s	ATP (1 sequence), 41J × 5
VT	150 bpm	8 of 10	2.5 s	ATP (3 sequences), 41J × 3

RV, right ventricular; LV, left ventricular; VF, ventricular fibrillation; FVT, fast ventricular tachycardia; VT, ventricular tachycardia; ATP, anti-tachycardia pacing.

prevention. An ICD can terminate ventricular tachyarrhythmia by cardioversion or anti-tachycardia pacing (ATP). However, some patients who suffer from frequent discharges of the ICD experience anxiety and depression [1]. Several studies have shown a correlation between poor quality of life (QoL) and frequent ICD shocks [2,3]. Recent studies have shown that empirical ATP for fast ventricular tachycardia (FVT) is an effective, painless therapy and improves QoL [4,5]. Moreover, subsequent shocks can terminate tachyarrhythmia even though ATP fails to terminate it. However, we experienced a patient with dilated cardiomyopathy (DCM) in whom ATP degenerated FVT into life-threatening polymorphic VT that could not be detected by an ICD.

Case report

A 45-year-old man with DCM was admitted to our hospital due to congestive heart failure (CHF) in April 2008. He experienced severe dyspnea with minor exertion [New York Heart Association (NYHA) functional class III]. Echocardiography showed that the left ventricular ejection fraction (LVEF) was 10%. He experienced non-sustained VT but did not have sustained lethal arrhythmia. A biventricular (Bi-V) ICD was implanted for reduction of dyssynchrony and primary prevention of lethal tachyarrhythmia [ICD generator: Contak Renewal 4 HE (Boston Scientific Corp, St Paul, MN, USA), atrial lead: Capsure Fix Novus 5076-52 (Medtronic Inc, Minneapolis, MN, USA), right ventricular lead: Linox TD 65/18 (Biotronik GmbH, Berlin, Germany), left ventricular lead: Easytrak2 4543 (Boston Scientific Corp)]. The ICD was programmed with three tachycardia zones (Table 1). FVT therapy consisted of initial ATP [8 autodecremental pulses at 88% cycle length (CL)] and subsequent shocks. The sensing level was set nominal ($\geq$0.27 mV) at the time of device implantation (pass filter: 20 Hz). ICD shock of 21 J could terminate induced ventricular fibrillation (VF) during a defibrillation threshold (DFT) test. Unfortunately, because Bi-V pacing was not effective for CHF, the patient was placed on the waiting list for a heart transplant in Japan. After discharge, he developed CHF and was transported to our hospital by ambulance in October 2008. His metabolic status at the time of transportation did not include hyper- or hypo-potassemia, or hypo-magnesemia, but slight renal dysfunction. He had taken diuretics, beta-blockers, and angiotensin II type 1 receptor blockers, but not amiodarone, because of lung side effects. In the ambulance, monomorphic sustained VT with 200 beats per minute (bpm) suddenly occurred (Fig. 1). The ICD detected it as FVT and an ATP was delivered. After the ATP therapy, the RR intervals of VT became irregular and prolonged. VF-like electrical activity was recorded by a far-field electrogram, which was recorded by bipolar between the ICD generator and defibrillation coil, and mimics a body surface electrocardiogram in the limb lead. Although each RR interval of the tachyarrhythmia encountered a VF zone, FVT zone, or slow VT zone, the ICD could not deliver shock because the CL of the tachyarrhythmia did not satisfy any of the criteria of VF zone and FVT zone. Finally, the tachycardia met slow VT criteria, and the ICD delivered a second ATP. The second ATP did not terminate the tachycardia but prolonged CL and it became over 400 ms (Fig. 1). The patient collapsed and resuscitation was started. ICD shock was not delivered because the CL was out of range of ICD detection. Because the electrocardiogram of cardiac monitoring in the ambulance showed a VF waveform, we attempted defibrillation of 360 J from an external defibrillator and the tachycardia was successfully terminated (Fig. 2).

Discussion

ATP for FVT sometimes induces VF which has short CL, and subsequent ICD shock terminates VF. Occasionally, ATP for FVT also induces slow VT with long CL, in which patients usually do not collapse. However, in the present case, ATP for FVT initiated slow but life-threatening VF-like electrical activity that could not be detected by an ICD and resulted in deteriorated hemodynamics. Whether this life-threatening arrhythmia was slow polymorphic VT or VF would be a key point in this case. ATP could introduce multiple block and zones of slow conduction in the severely injured myocardium. Multiple block and slow conduction resulted in random reentry, and then ''monomorphic sustained VT'' would convert to ''slow polymorphic VT''. In fact, after the ATP therapy, RR intervals of VT became irregular and prolonged over 400 ms (<150 bpm) in a near-field electrocardiogram and we therefore concluded that

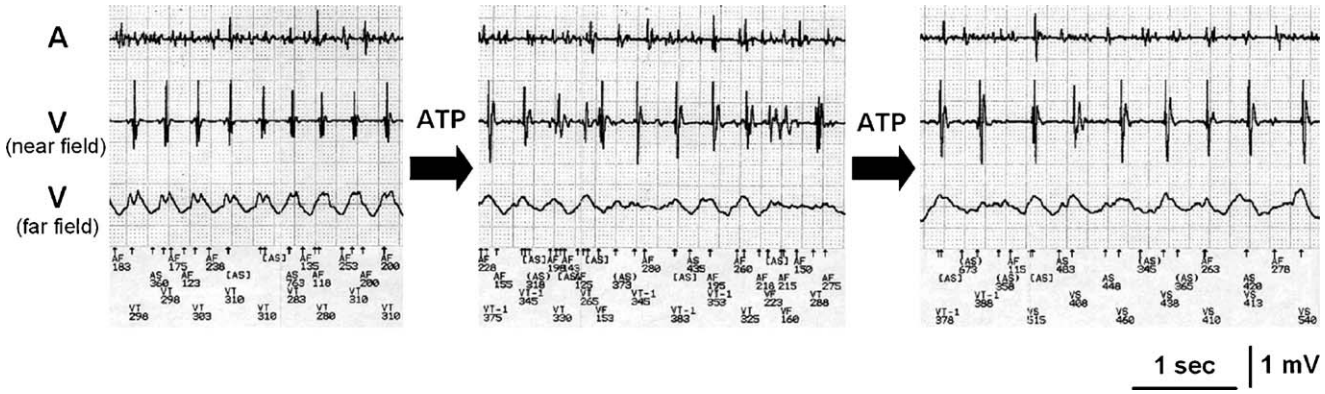

Figure 1 Monomorphic sustained ventricular tachycardia (VT) with a cycle length (CL) of 300 ms occurred. The implantable cardioverter-defibrillator (ICD) detected it as fast VT and anti-tachycardia pacing (ATP) was delivered. After the ATP therapy, the RR intervals of VT became irregular and prolonged. Ventricular fibrillation (VF)-like electrical activity was recorded by a far-field electrogram. Each RR interval encountered a VF zone, fast VT zone or slow VT zone. The tachycardia met slow VT criteria, and second ATP was delivered. After the second ATP therapy, the CL became over 400 ms, which was out of the range of ICD detection and ICD shock was not delivered. A: atrial electrogram; V: ventricular electrogram; near-field, bipolar electrocardiogram of tip of the defibrillation lead; far-field, bipolar electrocardiogram between the ICD generator and defibrillation coil.

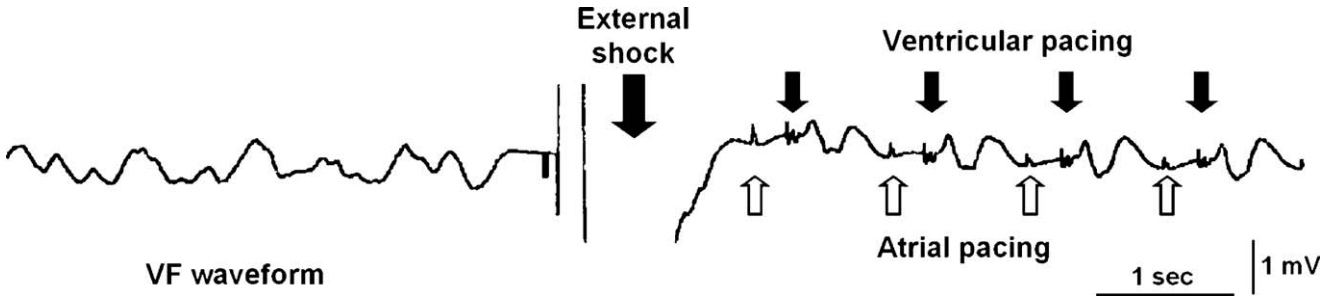

Figure 2 Electrogram of cardiac monitoring in the ambulance showed a ventricular fibrillation (VF) waveform. Defibrillation of 360 J from an external defibrillator was attempted and the tachycardia was successfully terminated by the first external defibrillation.

this arrhythmia was ''slow polymorphic VT'' because the rate of VF is usually between 150 and 500 bpm. However, in a far-field electrogram of an ICD or ambulatory monitoring, this arrhythmia seemed to be VF-like electrical activity due to multiple block. This is a rare case with severe cardiac dysfunction in which ATP could have been lethal to the patient.

The PainFREE RXII and EMPIRIC trials have demonstrated that empirical ATP for FVT is as useful as ICD shocks in primary and secondary prevention for patients without indication for a Bi-V ICD [4,5]. More recently, the ADVANCE CRT-D trial demonstrated that ATP is safe and effective for heart failure patients with Bi-V ICDs [6]. When compared with these trials, the efficacy of ATP to terminate VTs was less effective in patients with Bi-V ICD than in patients without indication for a Bi-V ICD. In fact, the PainFREE RXII trial demonstrated that deteriorated LVEF reduced successful termination of VTs by ATP [4]. Our patient had low cardiac function and it would have been associated with unsuccessful ATP for FVT. In addition to low cardiac function, it has been reported that the defibrillation efficacy in VT/VF is significantly dependent on the VT/VF duration and that a long duration of VT/VF (>15 s) increased DFT [7]. Empirical ATP for FVT has an advantage in most patients with an ICD; however, ATP for FVT is sometimes harmful for patients who have low LVEF, high NYHA functional class, and frequent recurrence of CHF, and early shock therapy is recommended to avoid the succeeding unstable hemodynamics.

In conclusion, this is a rare case of FVT that degenerated into undetectable life-threatening ventricular tachyarrhythmia by ATP. This case showed us practical implications relating to the programming of contemporary ICD devices.

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