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Case Report

Electrical storm after cardiac resynchronization therapy in a patient with nonischemic cardiomyopathy: Signal-averaged vector-projected 187-channel electrocardiogram-based risk stratification for lethal arrhythmia

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ABSTRACT

We describe treatment of atrial flutter and electrical storm presenting as incessant ventricular tachycardia (VT) after implantation of a cardiac resynchronization therapy defibrillator (CRT-D) in a patient with dilated cardiomyopathy. No prior arrhythmic event had occurred. Our treatment strategy, including amiodarone administration, was guided in part by signal-averaged vector-projected 187-channel electrocardiogram (SAVP-ECG)-based risk stratification for ventricular arrhythmia. Corrected recovery time (RTc) dispersion and Tpeak-end dispersion were used to evaluate transmural dispersion of repolarization. RTc and Tpeak-end dispersion increased during the period of electrical storm. Values were improved 2 years after CRT-D implantation, and the amiodarone was discontinued. The VT has not recurred despite discontinuation of the antiarrhythmic agent. SAVP-ECG-based risk stratification for ventricular arrhythmia proved useful for the management of antiarrhythmic therapy.

1. Case report

A 77-year-old man with dilated cardiomyopathy visited our hospital in December 2010 and reported dyspnea on effort. Echocardiography revealed cardiac dyssynchrony with a low ejection fraction of 29%, and the patient was diagnosed with New York Heart Association class III heart failure. The plasma N-terminal pro-B-type natriuretic peptide (NT-pro BNP) level was 3691 pg/mL. The electrocardiogram (ECG) QRS complex (136 ms) was widened to 136 ms, with a left bundle branch block conturation (Fig. 1). The patient was being treated with spironolactone (aldactone), β-blocker (carvedilol), and angiotensin II receptor antagonist (captopril). Implantation of a cardiac resynchronization therapy defibrillator (CRT-D) was scheduled and performed in May 2011, without any complications. After implantation of the right ventricular (RV) lead in the RV apex, coronary venography was performed, and a suitable lateral branch was identified as a candidate vessel for left ventricular (LV) lead implantation. The LV lead was positioned at the midportion of the lateral branch. The LV pacing threshold was 0.5 mV at 0.5 ms without phrenic nerve stimulation. The right atrial (RA) lead was then positioned at the RA appendage. The following device and leads were used: Promote RF generator, Durata 7120Q RV defibrillation lead, QuickFlex 1158T LV lead, and Tendril STS RA lead (St. Jude Medical, St. Paul, Minnesota, USA). The RV pacing threshold was 0.75 V at 0.4 ms, and the RA pacing threshold was 0.5 V at 0.4 ms. The device was programmed with a ventricular tachycardia (VT) zone set to ≥166 bpm (therapies = antitachy pacing (ATP) × 3, shock 10 J, 25 J, 36 J × 4) and a ventricular fibrillation (VF) zone set to ≥230 bpm (therapies = shock 15 J, 36 J, 36 J × 4).

After CRT-D implantation, the QRS duration decreased to 122 ms (Fig. 2), and the cardiac dyssynchrony improved. However, 5 days after implantation, atrial flutter (AFL) and electrical storm presenting as frequent VT were seen. As shown in Fig. 3A, the VT was initiated by a premature ventricular complex. The CRT-D intracardiac tracing showed both AFL and VT. Appropriate shocks were delivered, and both the AFL and VT were terminated (Fig. 3B); however, incessant VT developed after restoration of sinus rhythm. A total of 9 VT zone shocks were delivered. Biventricular pacing was discontinued, and intravenous administration of amiodarone was initiated to inhibit VT. The VT abated within 1 week, and CRT was restarted. Oral amiodarone was administered to prevent VT recurrence. To determine the risk of ventricular arrhythmia, we evaluated the corrected recovery time (RTc) dispersion and Tpeak-end dispersion on a signal-averaged vector-projected 187-channel electrocardiogram

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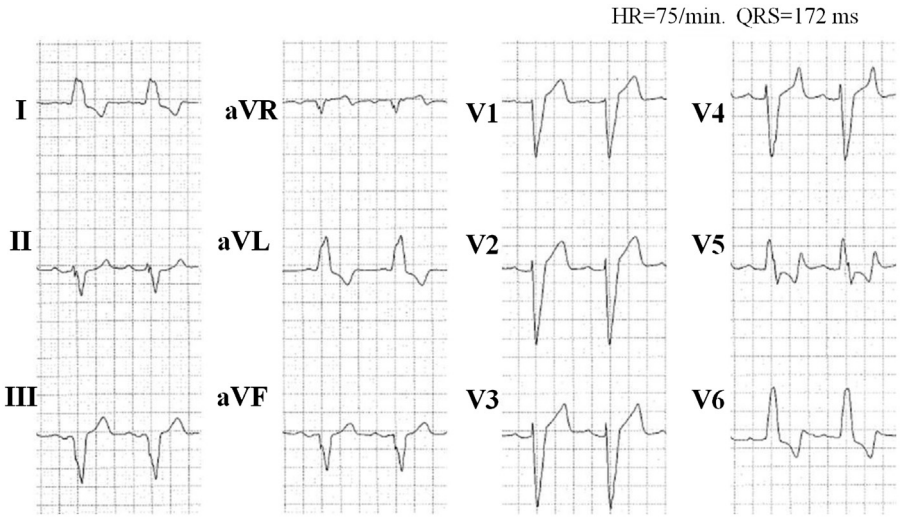

Fig. 1. Twelve-lead electrocardiogram obtained before CRT shows a wide QRS complex with left bundle branch block.

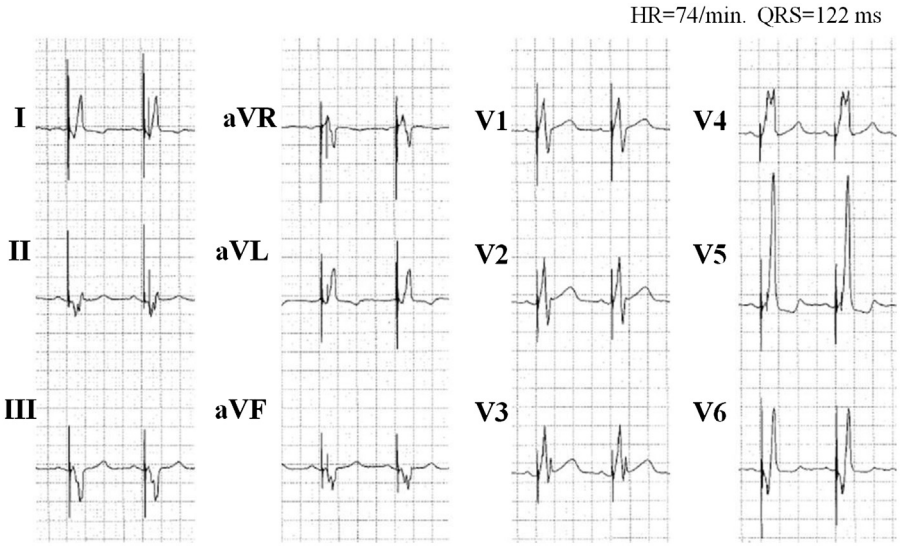

Fig. 2. Twelve-lead electrocardiogram obtained after CRT shows narrowing of the QRS complex compared to that before CRT.

(SAVP-ECG). RTc and Tpeak-end dispersion increased during the period of electrical storm (average, 15 ms and 48 ms, respectively; Fig. 4A). These findings suggest that transmural dispersion of repolarization increased in our patient, leading to ventricular proarrhythmia. Two years after implantation of CRT-D, follow-up SAVP-ECG showed decreased augmentation of RTc dispersion and Tpeak-end dispersion (17 ms and 13 ms, respectively; Fig. 4B). It remains unclear whether this improvement was the result of time or the administration of amiodarone. We halved the dose of amiodarone before withdrawing the drug altogether. No VT recurrence occurred despite discontinuation of the antiarrhythmic agent.

2. Discussion

Proarrhythmic events after CRT have been reported in 5–10% of CRT recipients [1–4]. Gasparini et al. investigated the incidence of electrical storm in patients with heart failure treated with CRT and reported an increased incidence in patients with nonischemic cardiomyopathy in whom a CRT-D was implanted for secondary prevention [5]. In most cases, the arrhythmia can be managed by administration of an antiarrhythmic agent and/or discontinuation of LV pacing within 1 month after implantation of the CRT system. Kantharia et al. reported a case of electrical storm induced by CRT. The VT did not disappear even after extraction of the LV lead, and catheter ablation was performed to control the VT [6]. In another case, VT was induced by biventricular pacing and controlled by triple-site biventricular pacing and atrioventricular node ablation [7]. In contrast, CRT has been reported to suppress arrhythmias in some cases [8–10]. These reports suggest that the suppression is not due to the effects of pacing itself. Rather, reverse remodeling with CRT can decrease the AFL burden and frequency of ventricular arrhythmias.

The mechanism underlying the proarrhythmic effect of CRT is not well understood. One explanation is that transmural dispersion of repolarization increases with LV pacing. Bai et al. studied the effects of LV epicardial pacing and biventricular pacing in a canine model of dilated cardiomyopathy [11] and showed that both LV epicardial pacing and biventricular pacing prolonged the ventricular repolarization time and increased transmural dispersion of repolarization. Prolonged transmural dispersion occurred parallel to augmentation in the Tpeak-end interval. According to Scott et al., CRT with transseptal

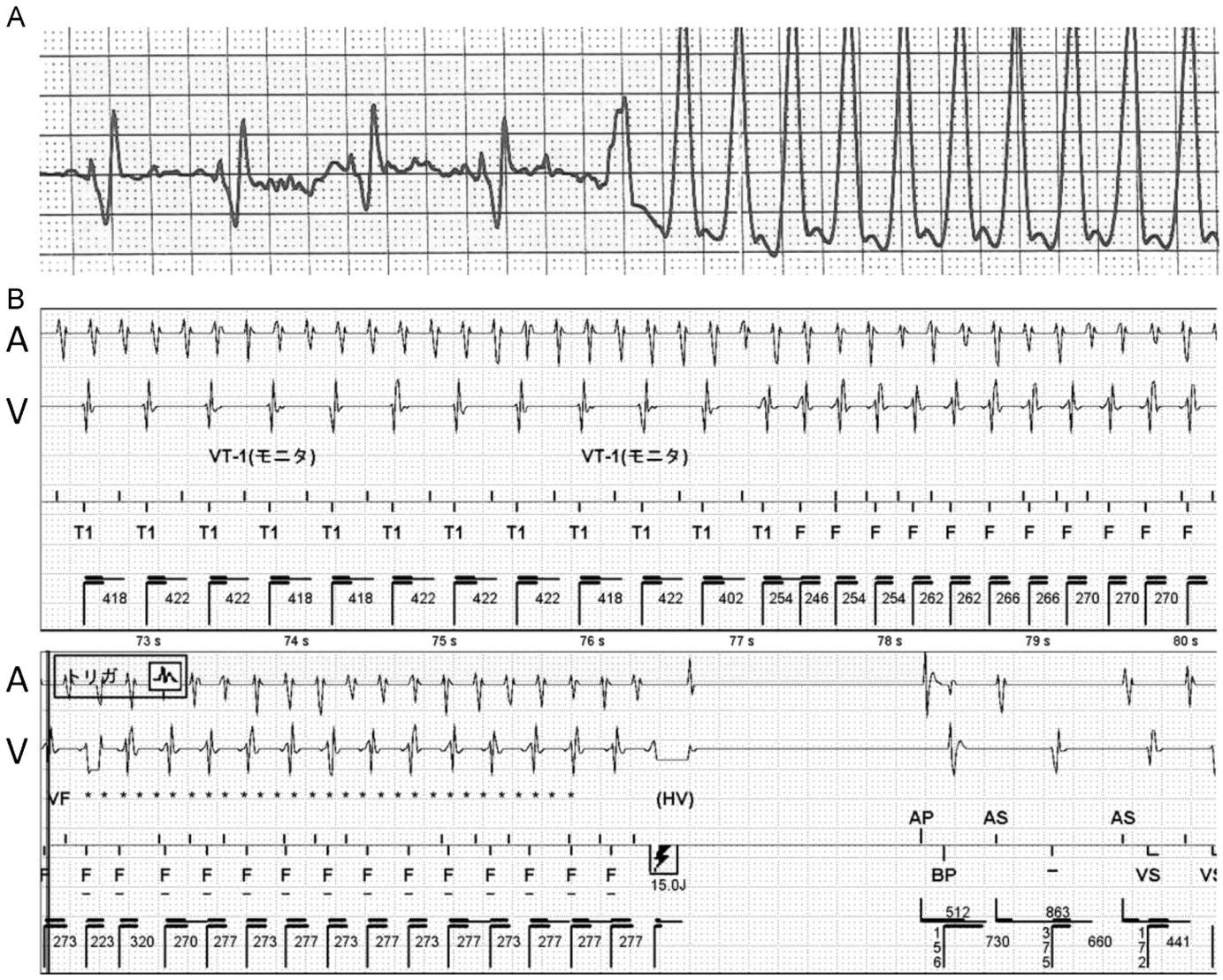

Fig. 3. Cardiac monitor tracing and intracardiac electrocardiogram shows atrial fibrillation and ventricular tachycardia episodes 5 days after CRT-D implantation.

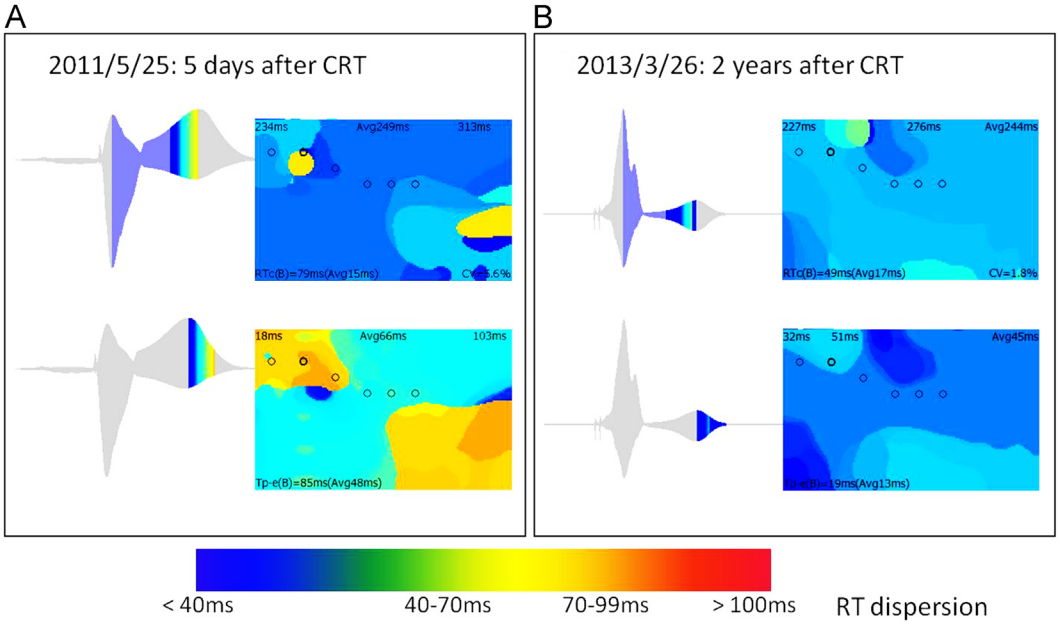

Fig. 4. RTc and Tpeak-end dispersion assessed on SAVP-ECG obtained 5 days and 1 year after CRT implantation. RTc and Tpeak-end dispersion were improved 2 years after CRT implantation.

endocardial LV pacing (in comparison with epicardial LV pacing) reduced QTc and Tpeak-end dispersion, and these authors concluded that transseptal LV pacing may be less arrhythmogenic [12]. Barbhaiya et al. looked at the relationship between ventricular arrhythmia, the QT interval, and Tpeak-end dispersion and found that increases in Tpeak-end dispersion and Tpeak-end/QT ratio were associated with an increased incidence of ventricular arrhythmia in patients with a CRT-D [13]. Another group also reported an association between Tpeak-end dispersion and major arrhythmic events [14].

Nakai et al. showed that RTc and Tpeak-end dispersion can be used to evaluate the spatial distribution of myocardial repolarization [15,16]. We measured RTc and Tpeak-end dispersion in the acute and chronic periods in the case reported herein. Both variables were increased during the acute period after CRT, suggesting that repolarization heterogeneity was augmented before being modified by amiodarone. These changes are consistent with previous reports of prolongation of the Tpeak-end interval with LV pacing. On the follow-up SAVP-ECG, RTc and Tpeak-end dispersion had decreased to within safe ranges. There is no standard index for use of antiarrhythmic agents, and it is difficult to make the decision to stop an antiarrhythmic agent once it is started, even if the patient is free of arrhythmia. In the present case, RTc/Tpeak-end dispersion increased in the acute phase after CRT, suggesting a potential substrate for ventricular arrhythmia, but both measures decreased to within normal range with amiodarone administration and with time. We controlled the dose of amiodarone in response to the low risk for ventricular arrhythmia indicated by the SAVP-ECG. Our experience in this case highlights the importance of risk stratification for lethal arrhythmia after CRT.

3. Conclusion

We treated a patient who experienced electrical storm after CRT-D implantation. Our treatment strategy was guided in part by SAVP-ECG-based risk stratification for ventricular arrhythmia. Our SAVP-ECG findings indicate that the proarrhythmic effect of CRT may be due to repolarization heterogeneity induced by LV pacing. The SAVP-ECG findings allowed us to both administer and withdraw the antiarrhythmic agent effectively; therefore, we suggest use of SAVP-ECG as a risk stratification tool in cases of CRT-induced electrical storm.

Conflict of interest

There is no conflict of interest related to this report.

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