

CASE REPORT

Cardiac Resynchronization Therapy and Proarrhythmia: Weathering the Storm

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ABBREVIATIONS:

CRT = cardiac resynchronization therapy
CRT-D = cardiac resynchronization
therapy-defibrillator
ECG = electrocardiogram
ICD = implantable cardioverter
defibrillator
LBBB = left bundle branch block
LV = left ventric-le(-ular)
VT = ventricular tachycardia
VTS = ventricular tachycardia storm

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ABSTRACT

In patients with significant left ventricular (LV) dysfunction and congestive heart failure despite optimal medical therapy, implantation of cardiac resynchronization therapy-defibrillator (CRT-D) devices has been shown to improve symptoms and diminish ventricular tachyarrhythmia susceptibility. We herein describe the case of a patient with dilated cardiomyopathy who developed ventricular tachycardia storm (VTS) one month after the implantation of a CRT-D device. The storm was initially controlled with pharmacotherapy, allowing the patient to continue with biventricular pacing. Two months later the patient was readmitted due to multiple episodes of polymorphic ventricular tachycardia. This time VTS was refractory to various intravenous antiarrhythmic drugs and it was finally controlled only when LV pacing was turned off. In patients with heart failure treated with CRT-D, VTS can occur and may occasionally be best managed by turning off LV pacing. Our report raises an important and concerning issue of biventricular pacing causing 'proarrhythmia' in rare instances.

INTRODUCTION

Cardiac resynchronization therapy (CRT) is an established therapy of heart failure, especially in patients with medically refractory heart failure and evidence of left ventricular (LV) dyssynchrony. Multiple studies have demonstrated the benefit of biventricular pacing on hemodynamics, quality of life, morbidity, and mortality.¹⁻⁸ Nevertheless, electrophysiological effects of CRT are still poorly understood.

CASE REPORT

A 64-year-old Caucasian man with a history of dilated cardiomyopathy was referred to our department for CRT. His functional status was New York Heart Association (NYHA) class III on optimal medical treatment. The patient had a history of frequent ventricular extrasystoles along with runs of nonsustained monomorphic ventricular tachycardia (VT) with left bundle branch block (LBBB) morphology and inferior axis, i.e. positive QRS in leads II, III and aVF and negative in lead aVL on 24-hour

Holter monitoring or resting ECGs. Neither sustained VT nor syncope was previously noted. Baseline ECG showed first degree atrioventricular block (PR interval at 268 ms), QRS complex with LBBB morphology and prolonged duration (152 ms), left axis deviation and prolonged QTc interval (536 ms, Fig. 1A). The patient had severe LV systolic dysfunction with an ejection fraction of 20%.

In view of continuing symptoms, a cardiac resynchronization device with defibrillation capability (CRT-D) was implanted for primary prevention. The procedure was uneventful without any complications. Sensing and pacing thresholds were optimal. Biventricular pacing was initiated at the time of implantation, causing a further QTc interval prolongation (634 ms, Fig. 1B). Within a month post-operatively, the patient developed a VT storm (VTS) with multiple episodes of spontaneous sustained monomorphic and polymorphic VT that could be terminated only transiently by implantable cardioverter defibrillator (ICD) therapies. The majority of monomorphic

VT episodes were interrupted with antitachycardia pacing, whereas most of polymorphic VT episodes were self-terminated and thus, untreated (Fig. 2A). The remaining episodes were interrupted by ICD shocks (Fig. 2B). The diagnosis of both monomorphic and polymorphic VT was made using 12-lead electrocardiography and device intracardiac electrograms (Fig. 2 & 3). Monomorphic VT during VTS was similar to the non-sustained monomorphic VT episodes before CRT-D implantation, i.e. with LBBB morphology and inferior axis.

The storm was initially controlled with intravenous amiodarone, allowing the patient to continue with biventricular pacing. There was no documentation of myocardial ischemia, metabolic or electrolyte disorders. Two months later the patient came to our emergency department again, complaining for progressively deteriorating dyspnea and palpitations. Interrogation of the device showed VTS with multiple episodes of both monomorphic and polymorphic VT treated by anti-tachycardia pacing or ICD shocks. Again, other than CRT-D

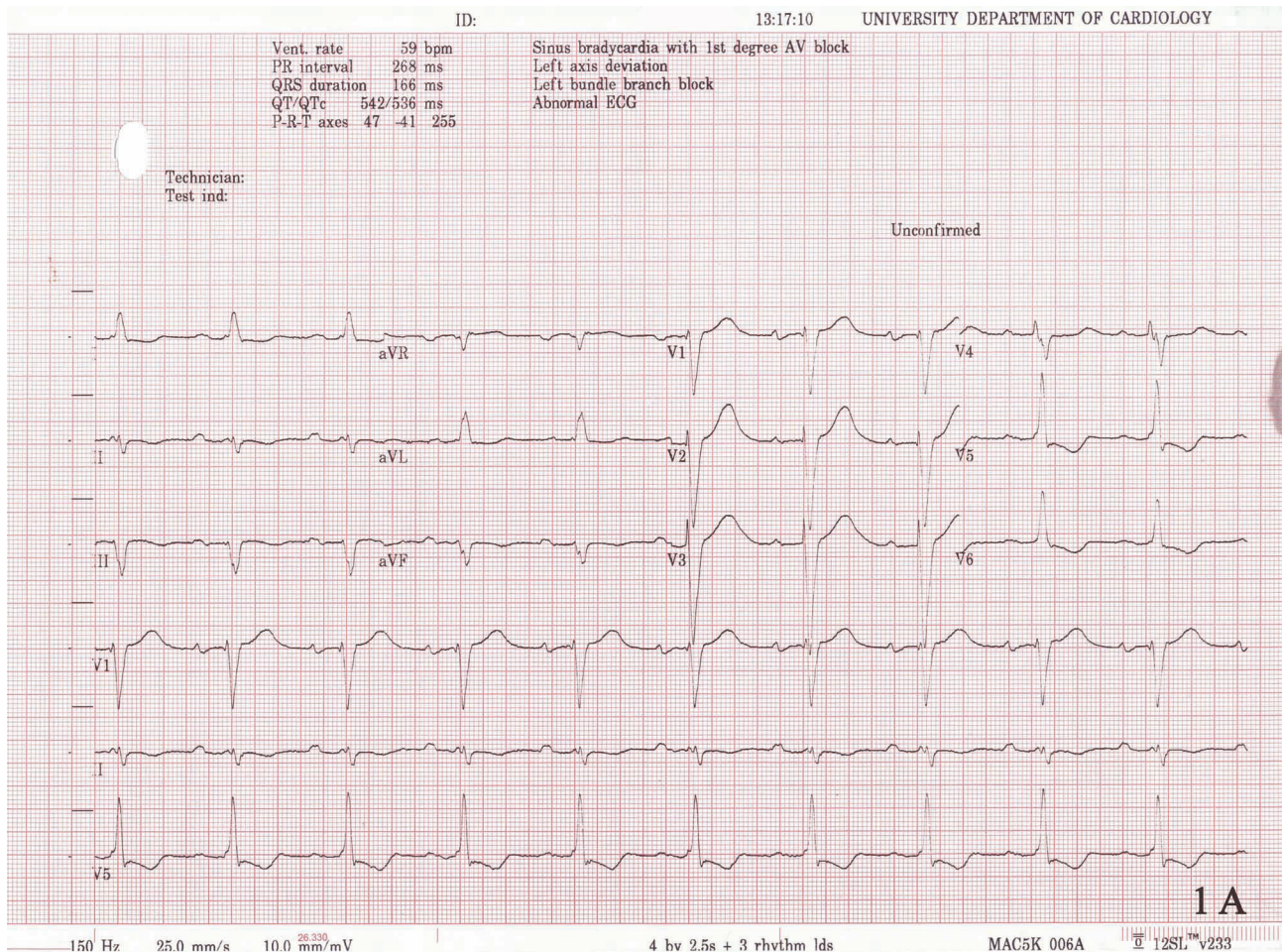


FIGURE 1A. 12-lead resting ECG showing 1st degree AV block and prolonged QRS duration with LBBB morphology and left axis deviation. AV = atrioventricular; LBBB = left bundle branch block.

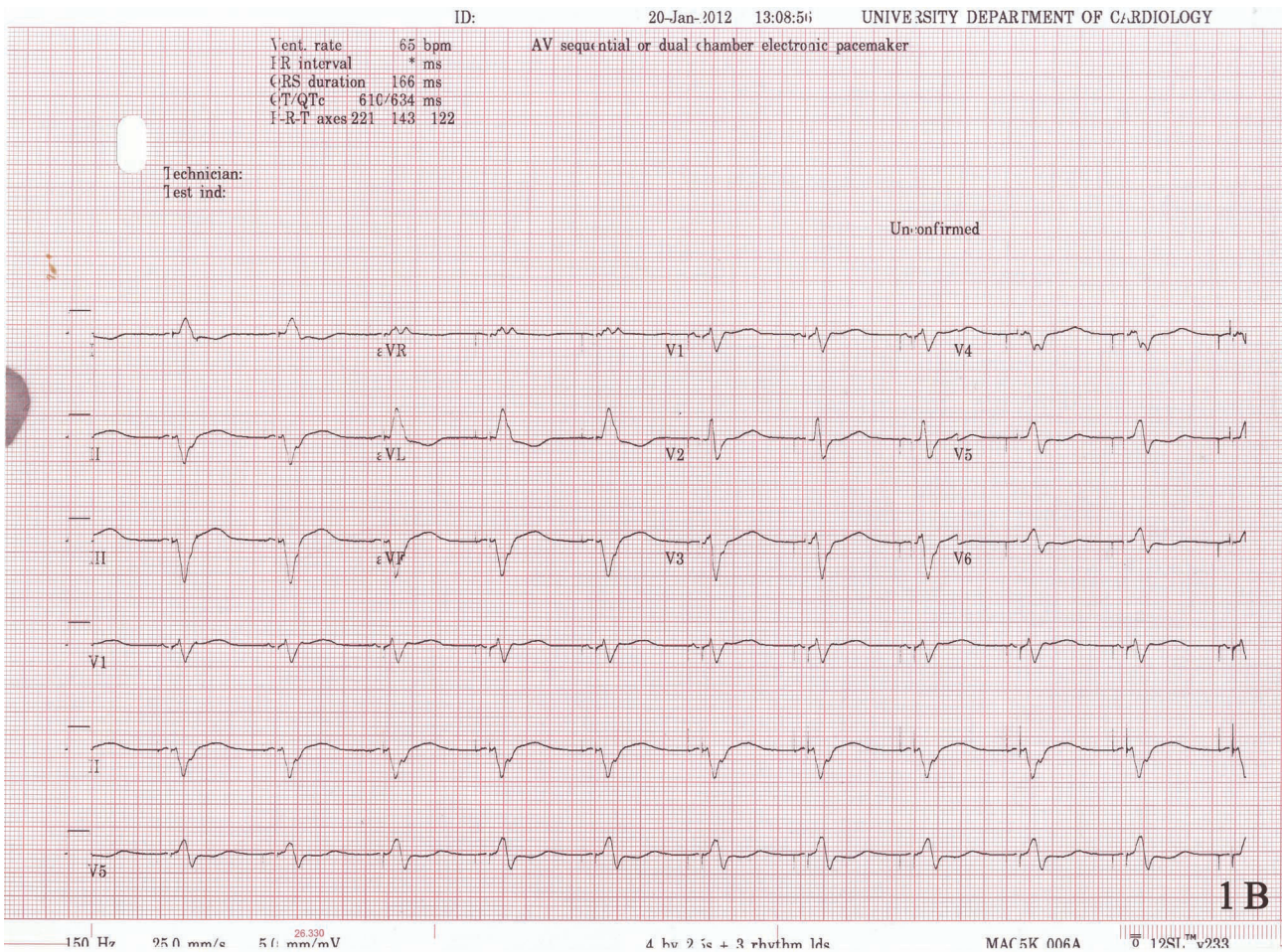


FIGURE 1B. 12-lead resting ECG showing biventricular pacing after the CRT-device implantation. The already prolonged QTc interval was further increased to 634 ms after the procedure. CRT = cardiac resynchronization therapy

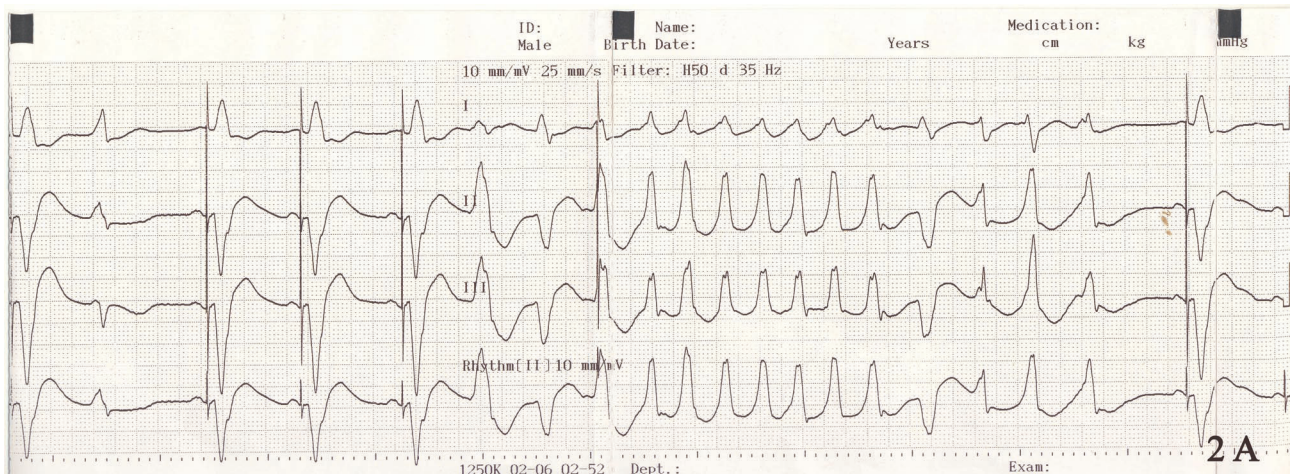


FIGURE 2A. Resting ECG showing self-terminating polymorphic ventricular tachycardia.

Episode: VT (176 min⁻¹ / 340 ms) (Continued)

VT/VF Episode 8 of 17
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1: A Sense Amp AutoGain (1.8 mm/mV)
2: Can-RV-coil AutoGain (2.9 mm/mV)

3: Markers

Sweep Speed: 25 mm/s

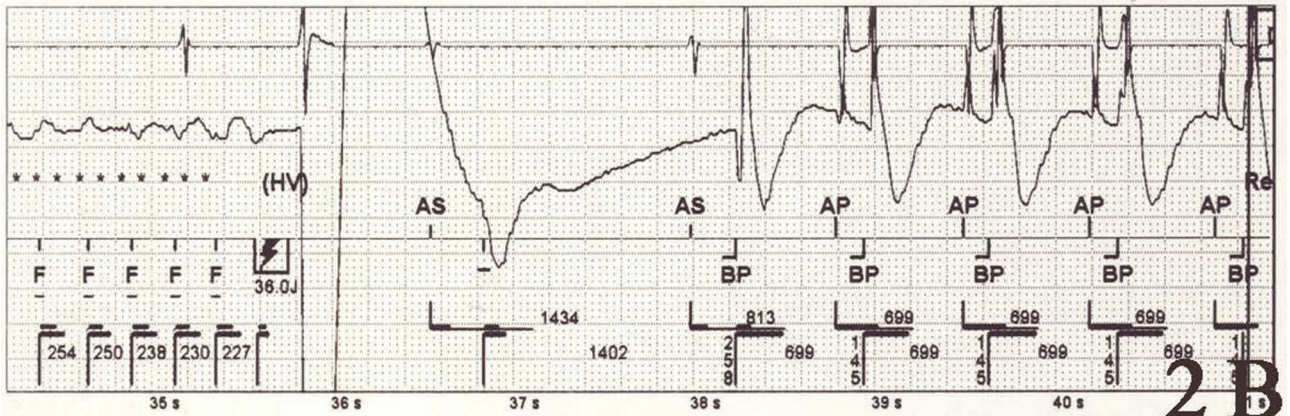
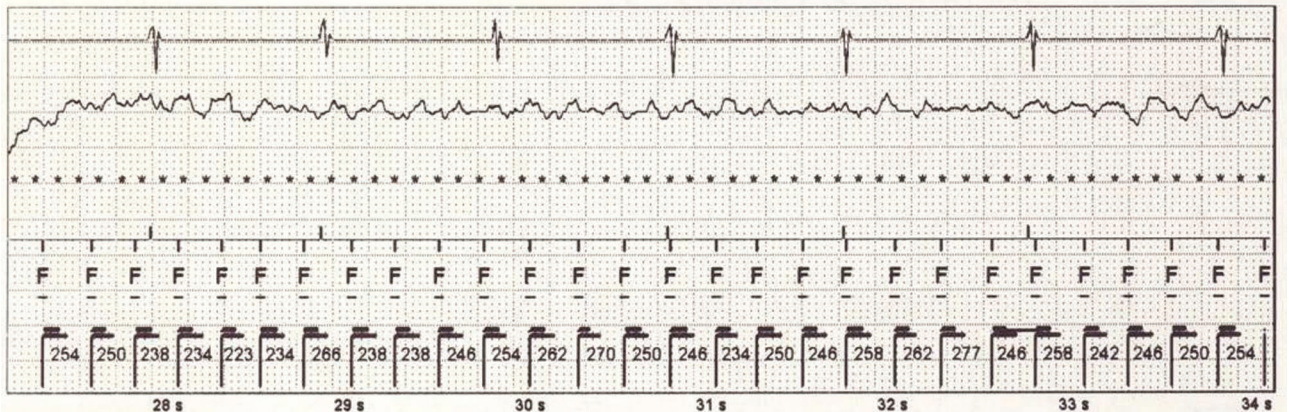
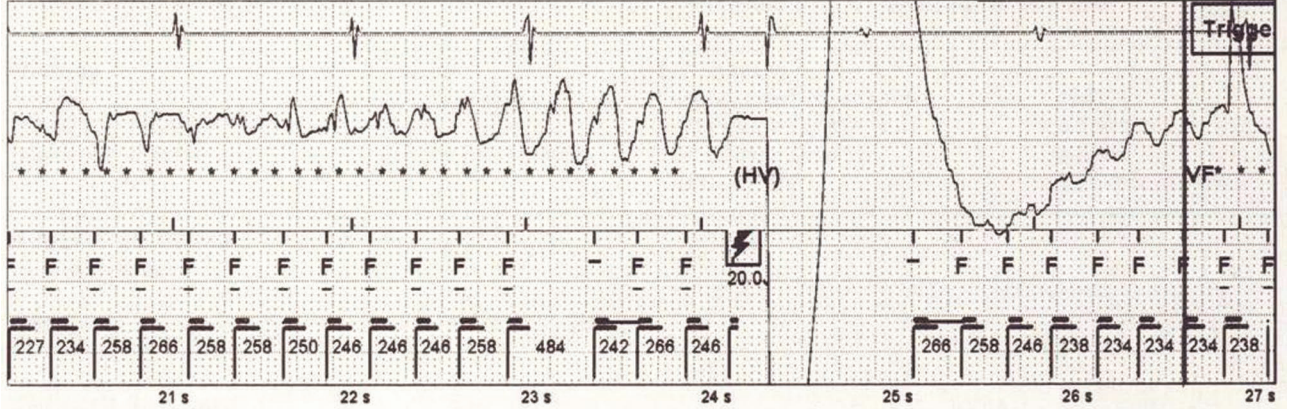


FIGURE 2B. Intracardiac electrogram showing polymorphic VT, which degenerated into VF after the first shock delivery and it was finally stopped by a second shock. VF = ventricular fibrillation; VT = ventricular tachycardia

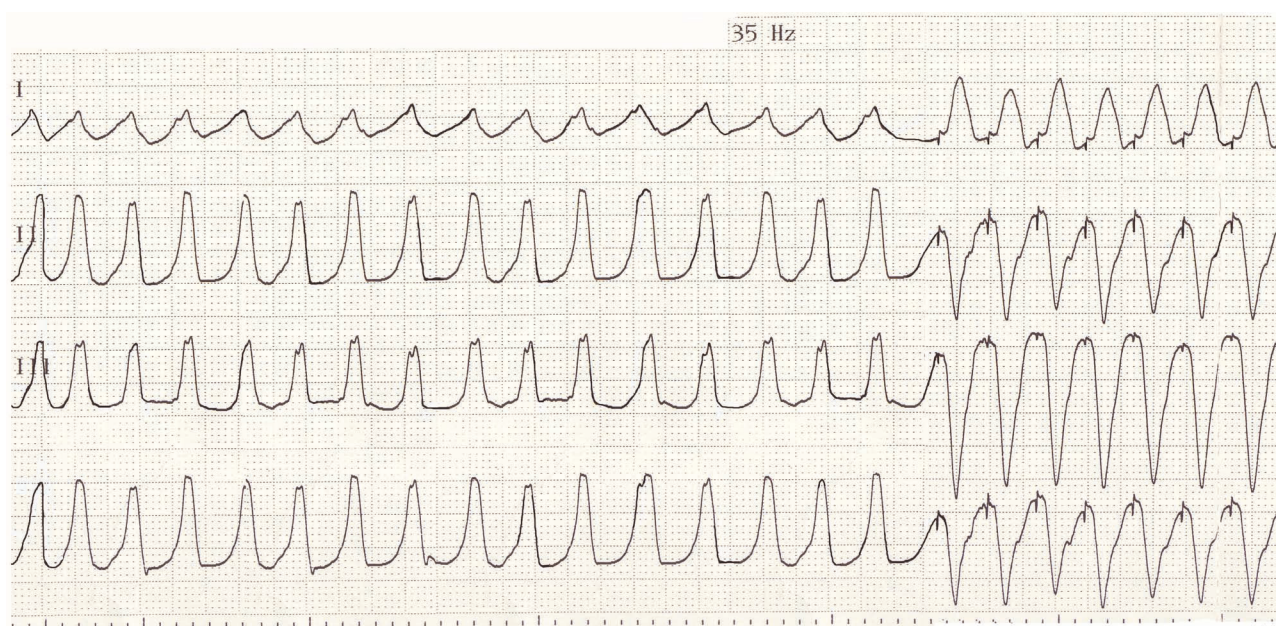


FIGURE 3A. Resting ECG showing rapid monomorphic VT and the subsequent antitachycardia attempt by the ICD. ICD = implantable cardioverter defibrillator; VT = ventricular tachycardia

implantation, no readily identifiable precipitating factor for the VTS was elicited. The patient was managed with intravenous antiarrhythmic drugs (amiodarone and subsequently lidocaine), resulting in a modest decrease in but no elimination of VT. Finally, LV pacing was turned off, with resolution of VT. All attempts to resume LV pacing resulted in VT recurrence. Three months after biventricular pacing was suspended, the patient remains in good health without any major arrhythmic events.

DISCUSSION

Although the delayed or halted progression of cardiac dysfunction due to CRT may be sufficient to prevent malignant ventricular tachyarrhythmias, there is still lingering uncertainty regarding the presence and magnitude of antiarrhythmic effects of CRT per se. Furthermore, there is experimental as well as clinical evidence that LV pacing may have proarrhythmic potential. Several reports have described a ventricular proarrhythmia effect.⁹⁻¹⁷ It occurs frequently within the first hours or days after initiation of biventricular pacing. The incidence reported in limited single series is low, between 3.4% and 4% during the first few days, with a predominance in ischemic cardiomyopathy.^{11,17} Either polymorphic or monomorphic VT have been described. What is interesting in our case is that both VTs were present: episodes of nonsustained monomorphic VT which deteriorated after biventricular pacing and de novo appearance of polymorphic VT, apparently due to excessive QTc

interval prolongation. Our patient received no antiarrhythmic medication during the time period after CRT-D implantation and before the first VTS. During VTS we used intravenous amiodarone and subsequently lidocaine. Although amiodarone may result in QT prolongation, it has a very low incidence of torsades de pointes, while lidocaine has no effect on both QT interval and risk of proarrhythmia. As a result, we believe that antiarrhythmic agents played an insignificant role in the arrhythmia recurrence. It seems that QTc interval prolongation was entirely and solely due to biventricular pacing. Ultimately, the only intervention, which proved to be effective in eliminating VT episodes, was inactivation of LV pacing.

Most patients could be managed by temporary discontinuation of biventricular pacing. In almost all cases, turning off LV pacing completely suppressed VT. Catheter ablation in combination with long-term antiarrhythmic medication is frequently proposed. Despite eliminating VT, most patients have a poor outcome with a low response to CRT. In our case, we tried, after the first VTS, to manage our patient with conventional intravenous antiarrhythmic therapy. Arrhythmia recurrence, two months later, made us abandon that idea. Only cessation of biventricular pacing completely suppressed VT and attempts to resume LV pacing resulted in VT recurrence.

There are several reported mechanisms by which biventricular pacing may alter the myocardial substrate and promote ventricular arrhythmias. It has been found that single epicardial and biventricular pacing in patients with congestive heart failure increased the QT interval and the transmural dispersion of repolarization, creating the substrate for reentrant poly-

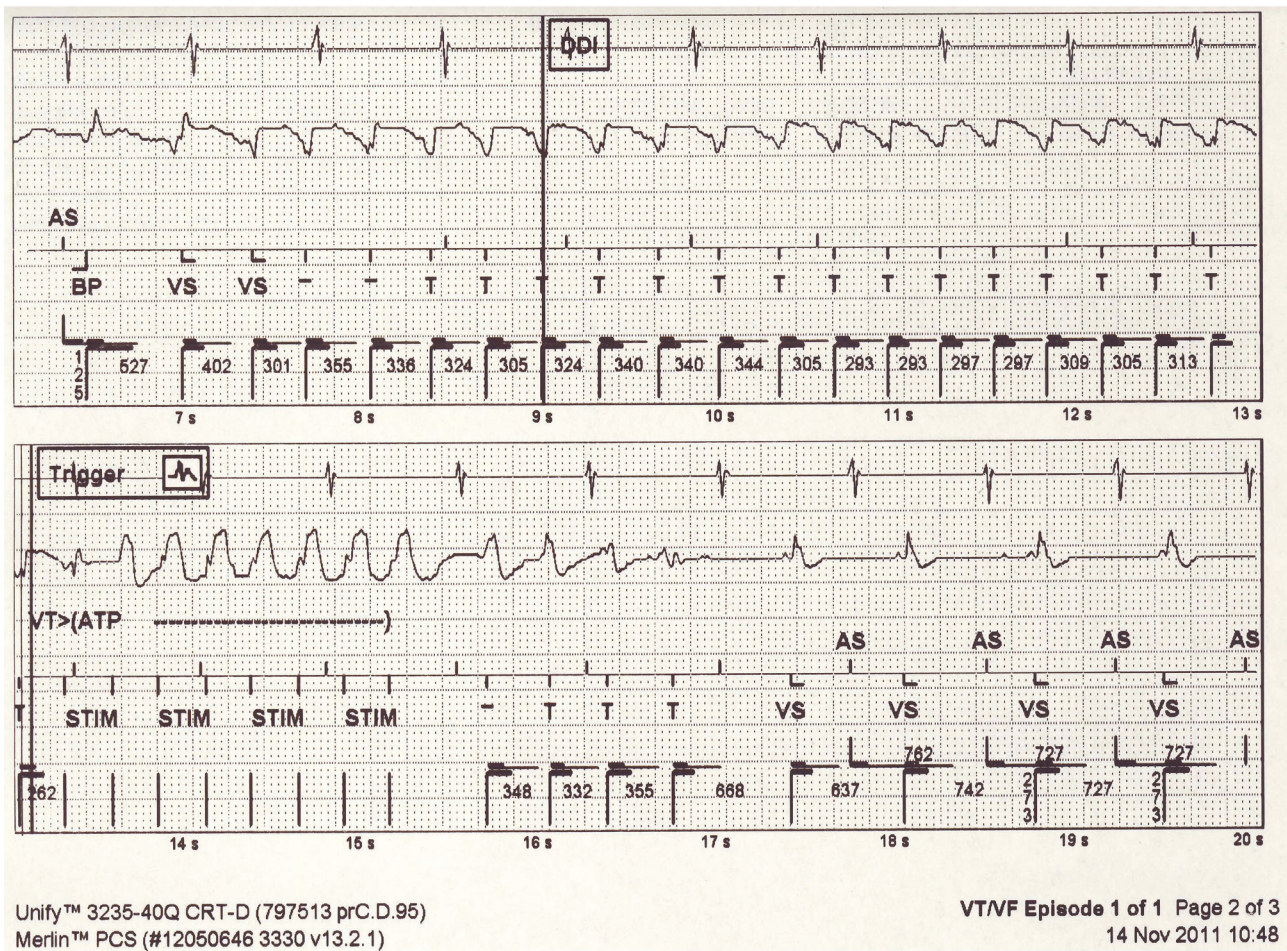


FIGURE 3B. Intracardiac electrogram showing a monomorphic VT episode which was correctly identified by the device and successfully interrupted by the first antitachycardia pacing attempt. VT = ventricular tachycardia

morphic VT.^{18,19} Moreover, LV pacing significantly modifies the activation pattern within and around myocardial scars.¹²

On the other hand, there is strong evidence that cardiac structural and contractile reverse remodeling following CRT can result in a favourable antiarrhythmic effect.^{20,21} Specifically, Dilaveris et al demonstrated a significant improvement in several arrhythmogenic indices after CRT in both ischemic and dilated cardiomyopathy patients. Similarly, in a subanalysis of MADIT-CRT trial,²¹ among those patients with a high response to biventricular pacing there was a significantly lower incidence of malignant ventricular tachyarrhythmias.²¹

CONCLUSION

In line with other reports, our case raises an important and concerning issue of CRT causing proarrhythmia in rare instances. It also raises an important concern regarding the

monitoring period after CRT device implantation, especially in patients receiving such a system without an ICD backup. The reason why certain patients go on to develop monomorphic or polymorphic VT after CRT therapy remains poorly understood. It would be prudent for physicians to be cognizant of this possible proarrhythmic effect of CRT.

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