CASE REPORT

Electrical storm of monomorphic ventricular tachycardia after a cardiac-resynchronization-therapy-defibrillator upgrade

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In patients with significant left ventricular dysfunction and congestive heart failure despite optimal medical therapy, implantation of cardiac resynchronization therapy-defibrillation (CRT-D) devices has been shown to improve symptoms and mortality. In this report, we describe a case of a patient with ischaemic cardiomyopathy who developed incessant ventricular tachycardia (VT) after undergoing an upgrade from an implantable cardioverter defibrillator to a CRT-D device. The patient required multiple anti-arrhythmic agents, removal of the coronary sinus lead, and radiofrequency ablation to control VT. Thus, in rare patients, the CRT devices may potentially cause 'proarrhythmia' with serious consequences.

KEYWORDS
Cardiac resynchronization therapy; Ventricular tachycardia; Cardiomyopathy; Congestive heart failure; Anti-arrhythmic agents; Radiofrequency ablation; Proarrhythmia

Cardiac resynchronization therapy (CRT) devices capable of bi-ventricular (Bi-V) pacing, or those that additionally provide defibrillation function (CRT-D) are now recommended for patients with congestive heart failure (CHF). However, little is known about the possible adverse ‘proarrhythmia’ that may result as a rare complication of these devices. In this case report, we describe a patient who developed incessant ventricular tachycardia (VT) after undergoing an upgrade from an implantable cardioverter defibrillator (ICD) to a CRT-D device.

Case report

A 73-year-old man with prior myocardial infarctions, coronary artery bypass surgery, and VT had implantation of an ICD with a dual-chamber pacing capability. Following therapy with amiodarone, the index VT had become slower at rates between 155 and 166 bpm and could be effectively terminated with antitachycardia pacing (ATP) therapies. The patient had no clinical episodes of VT or shocks for the next 3.5 years, but continued to have New York Heart Association (NYHA) class III-CHF. In view of continuing symptoms despite optimal medical therapy, severe left ventricular (LV) systolic dysfunction with an ejection fraction of 15%, electrocardiogram with first degree atrioventricular block with PR interval measuring 260 ms, and left bundle branch block (LBBB) with QRS complexes measuring 187 ms, the system was upgraded to CRT-D at the time of elective ICD generator change for battery depletion. A new unipolar coronary sinus (CS) lead, model 1056 K and a new generator, model V-340 (St Jude Medical, Sylmar, CA, USA) were implanted (Figure 1). The right ventricular (RV)-paced QRS, the LV-paced QRS, and the Bi-V-paced QRS measured 240, 220, and 147 ms, respectively. The sensing and pacing thresholds were optimal. The procedure was uneventful without any complications; in addition no ventricular arrhythmias were observed during manipulation of the guide wire or the CS lead in the posterolateral vein or when the lead was tested. No change or prolongation in the QT interval was noted post-operatively. The device was programmed as follows: (i) VT detection = 132–150 bpm for monitoring purposes only; (ii) VT detection = 151–181 bpm, therapies = ATP / C2 / 3, 25 J / C2 / 3 and (iii) VF detection = 182 bpm, therapies = 36 J / C2 / 6.

Within 12 h post-operatively, the patient developed incessant VT with multiple episodes of spontaneous sustained monomorphic VT with PR interval measuring 260 ms, and left bundle branch block (LBBB) with QRS complexes measuring 187 ms, the system was upgraded to CRT-D at the time of elective ICD generator change for battery depletion. A new unipolar coronary sinus (CS) lead, model 1056 K and a new generator, model V-340 (St Jude Medical, Sylmar, CA, USA) were implanted (Figure 1). The right ventricular (RV)-paced QRS, the LV-paced QRS, and the Bi-V-paced QRS measured 240, 220, and 147 ms, respectively. The sensing and pacing thresholds were optimal. The procedure was uneventful without any complications; in addition no ventricular arrhythmias were observed during manipulation of the guide wire or the CS lead in the posterolateral vein or when the lead was tested. No change or prolongation in the QT interval was noted post-operatively. The device was programmed as follows: (i) VT detection = 132–150 bpm for monitoring purposes only; (ii) VT detection = 151–181 bpm, therapies = ATP / 3, 25 J / 1, 36 J / 3 and (iii) VF detection = 182 bpm, therapies = 36 J / 6.

Within 12 h post-operatively, the patient developed incessant VT with multiple episodes of spontaneous sustained monomorphic VT and resultant ICD shocks. A monomorphic VT of LBBB morphology with superior axis at a rate of 167 bpm (Figure 2A) was appropriately detected and treated by the device (Figure 2B). Most ATP therapies, however, accelerated VT, and therefore, ATP was inactivated. The patient was treated with intravenous amiodarone, and subsequently required lidocaine, procainamide, oral amiodarone, and mexiletine. Although no triggering
premature ventricular complexes were observed, initially the LV pacing was turned off, and subsequently the CS lead was also removed to avoid focal mechanical irritation close to the re-entrant circuit that may lead to further VT. He continued to have monomorphic VT albeit at a slower rate of 96 bpm. The QRS complexes widened and the morphology changed to right bundle branch block (RBBB). The wavefront of VT was thought to exit and activate in the posterolateral area of the LV (site of the epicardial CS lead), which was supported by a QS pattern in lead I, aVL, and dominant R up to V6 (Figure 3A). The patient subsequently underwent a radiofrequency ablation procedure using a substrate-mapping technique. Using the electroanatomical CARTO™ mapping system, based on electrograms of low amplitude (<0.5 V), a large scar extending from the septal basal to the lateral area of the LV was delineated. A narrow isthmus between scar areas was identified. Pace mapping from potential exit sites at the border zones between scar and healthy tissue was also performed. Linear RF ablations were then applied at the isthmus and exit points of the circuit, and from the edge of the scar to the mitral valve annulus (Figure 4). During brief episodes of non-sustained VT, presystolic

Figure 1 Fluoroscopic view in the left anterior oblique projection shows the right atrial active screw-in lead placed in the lateral wall, the RV pace/sense and defibrillation lead placed in the RV apex, and the CS lead placed through the main CS body into the posterolateral vein.

Figure 2 A 12-lead ECG of VT of LBBB morphology and inferior axis at a rate of 167 bpm is shown in (A). In (B), the intracardiac electrograms of the VT that were recorded from the device are shown. The top channel electrograms are from the right atrium and the bottom channel electrograms are from the RV. The middle channel annotates markers. A rapid VT (cycle length 350–340 ms) is correctly identified and treated with a shock of 850 V.
potentials (88 ms before the onset of QRS) were recorded at the mid-isthmus (Figure 3B).

Although the patient subsequently remained stable from the arrhythmia standpoint, after 3 weeks he died from intractable heart failure.

Discussion

In most patients with significant LV dysfunction and CHF, despite optimal medical therapy, implantation of CRT-D devices has been shown to improve symptoms and mortality. Furthermore, reduction in both appropriate ATP therapy and ICD shocks, as observed in the study by Ermis et al., suggests that CRT diminishes ventricular tachyarrhythmia susceptibility and burden in these patients. However, there have been a few reports of induction of both polymorphic and monomorphic VT by Bi-V/LV pacing related to CRT.

The reasons why certain rare patients go on to develop monomorphic or polymorphic VT after CRT therapy remains unclear. Recent reports suggest that LV epicardial pacing...
can be proarrhythmic leading to polymorphic VT by reversal of the normal activation sequence, prolongation of QT interval, and creation of transmural dispersion of repolarization.4,8 The mechanism of development of monomorphic VT after CRT is also poorly understood. In fact, in a small study by Kowal et al.,9 induction of VT by programmed electrical stimulation using Bi-V pacing was significantly reduced when compared with RV pacing. It has been hypothesized that in Bi-V pacing, pre-excitation of the area of slow conduction occurs which abolishes or minimizes the conduction delay of a premature complex that is necessary for the re-entrant arrhythmia. However, it is also possible that creation of unidirectional block and the dispersion of refractoriness in zones of slow conduction within the scar tissue located in the LV by LV pacing promote re-entrant VT. In an era when invasive EP studies were more widely performed, it was common to perform pacing in the LV to induce VT.10 In previously reported cases, monomorphic VT occurred in response to LV pacing.5,6 Although in one case, the VT was inhibited by activation of Bi-V pacing,5 in another case, the only intervention that eliminated VT was inactivation of LV pacing.6 In fact, in the latter case, despite subsequent regimen of multiple anti-arrhythmic agents all subsequent attempts to reactivate LV pacing resulted in prompt VT recurrence.6 In our patient, VT of incessant nature was initiated by Bi-V pacing. Despite discontinuing LV pacing and LV lead removal, the patient continued to have VT. We believe that the use of multiple anti-arrhythmic drugs that were necessary to suppress the VT led to a marked alteration in the inherent electrophysiological properties of the myocardium and facilitated adverse re-entry causing further VT.

In line with other reports, our case raises an important and concerning issue of CRT causing ‘proarrhythmia’ in rare instances. It would be prudent for physicians instituting CRT to be cognizant of the rare possibility of proarrhythmic effects.

References