A Shocking Case of Far-Field Atrial Oversensing in Giant-Cell Myocarditis

Tanuka Datta, MD, Stephen Melnick, DO, Bharaniabirami Rajaram, MD, Behzad B. Pavri, MD

**ABSTRACT**

We report a unique case of delivery of inappropriate implantable cardioverter-defibrillator therapies related to a "perfect storm": presence of an integrated lead, insufficient lead slack related to right heart dilation resulting in shock coil misplacement, myocarditis with loss of R waves, and the concomitant occurrence of an incessant atrial tachycardia.

**HISTORY OF PRESENTATION**

A 35-year-old male with a history of nonischemic cardiomyopathy from giant-cell myocarditis (GCM) with a HeartMate 3 left ventricular assist device (Abbott Cardiovascular, Plymouth, Minnesota) and a single-chamber implantable cardioverter-defibrillator (VIGILANT EL ICD D232/254693, Boston Scientific, Marlborough, Massachusetts) presented after receiving a shock. His vital signs showed blood pressure of 110/89 mm Hg, pulse of 69 beats/min, and oxygen saturation of 99% on room air. Echocardiography showed that his left ventricle, even when fully unloaded by the left ventricular assist device with normal flows, was barely contractile. The right ventricle was also severely dilated and hypokinetic, suggesting single-ventricle physiology with the right ventricle acting as a passive conduit. Telemetry and electrocardiographic review showed an atypical atrial flutter or atrial tachycardia with predominantly 2:1 atrioventricular block (Figures 1A and 1B).

**DIFFERENTIAL DIAGNOSIS**

The differential diagnosis for shock therapy included ventricular tachycardia (VT), ventricular fibrillation (VF), and inappropriate sensing.

**INVESTIGATIONS**

Interrogation revealed device programming for VVI pacing at 40 beats/min. Tachytherapies were programmed in 2 zones: VT at 200 beats/min (10 s) and VF at 220 beats/min (2.5 s). Therapies for arrhythmias in the VT zone were programmed to 2 rounds of antitachycardia pacing (ATP) followed by 36-J shocks;
therapies for arrhythmias in the VF zone were programmed for 1 try of ATP followed by shocks. The device logs showed that the patient had experienced 38 episodes of “VT,” with 22 episodes triggering ATP and 3 episodes resulting in shocks; all treated episodes were declared to have “successful termination” (Figure 2), all occurring over a short time period. The stored intracardiac trends disclosed that over the preceding 2 months, as his steroids were tapered, the sensed R-wave amplitude had diminished to the point at which the defibrillator was no longer able to sense native R waves even at maximum autogain (Figure 3). However, the far-field atrial electrograms during ongoing atrial tachycardia were sensed by the integrated defibrillator lead, leading to delivery of inappropriate therapies.

**MANAGEMENT**

In the setting of GCM, the progressive reduction in R-wave amplitudes was likely due to direct involvement of the myocardial tissue in the right ventricle, where his defibrillator lead was located, because of disease progression. Furthermore, Boston Scientific defibrillator leads have an integrated bipolar configuration; right ventricular (RV) dilation and the septal lead position (placed at an outside hospital) likely contributed to the insufficient slack seen on chest radiography, resulting in the proximal end of the shocking coil (used as the anode for sensing) being in the right atrium (Figure 4). The automatic sensing threshold of his defibrillator had dropped down to the lowest level because of small native QRS complexes, which resulted in detection of atrial signals during the ongoing

**FIGURE 1 Telemetry and Electrocardiography of Atypical Atrial Flutter**

(A) Telemetry showing 2:1 atrial tachycardia. Visible atrial deflections are marked with arrows; atrial rate is about 210 to 220 beats/min. (B) 12-Lead electrocardiogram showing atypical atrial flutter with 2:1 atrioventricular conduction; atrial rate is approximately 216 beats/min, and ventricular rate is about 108 beats/min. Electrocardiogram is displayed at twice gain with 20-Hz filter applied.
atrial tachycardia at a rate of approximately 210 beats/min (Figure 5). This arrhythmia was detected as “VT,” resulting in inappropriate ATP followed by a shock, terminating the atrial tachycardia briefly.

We tested for true R-wave sensing at maximum sensitivity of 0.15 mV, but the device continued to sense only atrial signals. In the setting of inability to detect true R waves, and inappropriate shocks due to far-field atrial oversensing during incessant atrial tachycardia, the device was completely deactivated for both tachycardia and bradycardia therapies.

**DISCUSSION**

Inappropriate shocks can be the result of oversensing atrial depolarization during an atrial arrhythmia such as flutter or tachycardia (1–3). Clinical scenarios in
which these occur are summarized in Table 1 (4–6). RV lead positioning becomes especially important with integrated bipolar leads; placement in the RV apex with the distal coil lying entirely within the RV cavity will minimize the probability of atrial oversensing (7,8). Remote monitoring has the potential to recognize early changes and potentially prevent such outcomes (9).

**FOLLOW-UP**

The patient successfully underwent heart transplantation in the following months and is doing well clinically.

**CONCLUSIONS**

Inappropriate shocks in the setting of inflammatory cardiomyopathy can be the result of disease progression as well as RV lead factors such as bipolar configurations and lead placement. Cardiologists must be able to distinguish between true VT and inappropriate sensing of atrial arrhythmias in this unique setting.

**FUNDING SUPPORT AND AUTHOR DISCLOSURES**

The authors have reported that they have no relationships relevant to the contents of this paper to disclose.
| Cause of Oversensing                                                                 | Image                                                                 | First Author (Ref. #)                        |
|-------------------------------------------------------------------------------------|----------------------------------------------------------------------|---------------------------------------------|
| RV lead-only devices                                                                 |                                                                     |                                             |
| Lead dislodgement to the atrium or atrioventricular junction                         | [Image](#)                                                          | Brüggemann et al. (4)                       |
| Integrated bipolar lead positioning near tricuspid annulus                           | [Image](#)                                                          | Kossaify (5)                                |
| Unintentional lead implantation into coronary sinus                                  | [Image](#)                                                          | Gunderson et al. (6)                        |

Continued on the next page
TABLE 1 Continued

| Cause of Oversensing                                                                 | Image | First Author (Ref. #) |
|--------------------------------------------------------------------------------------|-------|-----------------------|
| Insulation defect in atrial portion of the lead causing sensing of atrial activity |       | Gunderson et al. (6)   |

Dual-chamber devices

Atrial lead to RV lead interaction with the atrial lead contacting the RV lead during atrial systole, thereby producing a signal that is sensed by the RV lead

Gunderson et al. (6)

REFERENCES

1. Grimm W, Menz V, Hoffmann J, et al. Complications of third-generation implantable cardioverter defibrillator therapy. Pacing Clin Electrophysiol 1999;22:206-11.

2. Kowalski M, Ellenbogen KA, Wood MA, Friedman PL. Implantable cardiac defibrillator lead failure or myopotential oversensing? An approach to the diagnosis of noise on lead electrograms. Europace 2008;10:914-7.

3. Daubert JP, Zareba W, Cannom DS, et al., for the MADIT II Investigators. Inappropriate implantable cardioverter-defibrillator shocks in MADIT II: frequency, mechanisms, predictors, and survival impact. J Am Coll Cardiol 2008;51:1357-65.

4. Brüggemann T, Dahlke D, Chebbo A, Neumann I. Tachycardia detection in modern implantable cardioverter-defibrillators. Herzschrittmacherther Elektrophysiolog 2016;27:171-85.

5. Kossaify A. Sensing and detection functions in implantable cardioverter defibrillators: the good, the bad and the ugly. Acta Cardiol Sin 2020;36:308-17.

6. Gunderson BD, Patel AS, Bounds CA, Shepard RK, Wood MA, Ellenbogen KA. An algorithm to predict implantable cardioverter-defibrillator lead failure. J Am Coll Cardiol 2004;44:1898-902.
7. Müller D, Hoffman E, Fiek M, Grünewald A, Steinbeck G. Bedeutung der Speicherelektrogramme zur Differenzierung adäquater und inadäquater ICD-Therapien [Stored electrograms to differentiate between adequate and inadequate ICD therapy]. Herzschrittmacherther Elektrophysiol 1997;8:39–45.

8. Barold SS, Kucher A. Far-field atrial sensing by the left ventricular channel of a biventricular device. Pacing Clin Electrophysiol 2014;37:1624–9.

9. Varma N, Michalski J, Epstein AE, Schweikert R. Automatic remote monitoring of implantable cardioverter-defibrillator lead and generator performance: the Lumos-T Safely Reduces Routine Office Device Follow-Up (TRUST) trial. Circ Arrhythm Electrophysiol 2010;3:428–36.

KEY WORDS cardiac pacemaker, cardiomyopathy, supraventricular arrhythmias, ventricular tachycardia