DEVICE THERAPY

COMPLEX CASE STUDY

Inappropriate ICD Shocks Due to Internal Electromagnetic Interference Generated by Intracoronary Wire Manipulation

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ABSTRACT. A 52-year-old male with a history of severe, symptomatic ischemic cardiomyopathy and primary prevention implantable cardioverter-defibrillator (ICD) placement presented to the Emergency Department with chest pain refractory to medical therapy. During cardiac catheterization, two intracoronary wires were placed in the distal left anterior descending artery, which was located less than 4 mm from the tip of the integrated bipolar ICD lead. During intracoronary wire manipulation, the patient received two defibrillations from his ICD while in sinus rhythm. Review of the intracardiac electrograms showed sinus rhythm with a low voltage, variable frequency signal predominantly seen on the near field channel temporally corresponding precisely with intracoronary wire manipulation, which was detected as ventricular fibrillation (VF) resulting in defibrillation. The patient received inappropriate defibrillation due to intracoronary wire manipulation creating an internal source of electromagnetic interference.

KEYWORDS. artifacts, defibrillators, electromagnetic interference, electromagnetic phenomenal, implantable, inappropriate shock.

Introduction

A majority of inappropriate shocks from implantable cardioverter-defibrillators (ICDs) occur due to supraventricular tachycardia. Other less frequent causes include electromagnetic interference (EMI), lead noise, and oversensing. Regardless of the cause, inappropriate shocks result in significant discomfort and distress for the patient. Close inspection of the events surrounding inappropriate shock is required to determine the etiology. In this report, we describe a patient who received two inappropriate shocks due to EMI originating from an internal source while undergoing cardiac catheterization.

Case summary

A 52-year-old male with a history of severe, symptomatic ischemic cardiomyopathy (left ventricular ejection fraction 25%) and prior three-vessel coronary artery bypass surgery (left internal mammary artery (LIMA) to the mid-left anterior descending artery (LAD), saphenous vein graft to the first obtuse marginal artery, and saphenous vein graft to the second obtuse marginal artery) presented to the hospital with chest pain. Six years prior to presentation he had received a single chamber ICD (Generator-T177 VR Vitality 2, ICD lead-0181 Endotak Reliance SG, Boston Scientific, Natick, MA) for primary prevention of sudden cardiac death. The patient had never received appropriate or inappropriate shocks or antitachycardia pacing.

On arrival to the Emergency Department, a 12-lead electrocardiogram (ECG) was performed which did not show ST-segment changes and his troponin was marginally elevated at 0.33 ng/mL. Owing to persistent chest pain not controlled with aspirin, clopidogrel, heparin, and intravenous nitroglycerin, he was transferred to the cardiac catheterization laboratory for urgent coronary
angiography. The angiogram was performed from his right femoral artery, which identified 100% occlusion of the LIMA and a 70% lesion in the mid-LAD. To assess the hemodynamic significance of the mid-LAD lesion, fractional flow reserve (FFR) was performed. Using a 6 French 3.5 EBU guide catheter (Medtronic, Minneapolis, MN), a Prime Wire Prestige Plus pressure guidewire (Volcano, Inc., San Diego, CA) was advanced across the mid-LAD lesion and into the distal LAD. On fluoroscopy, the FFR guidewire in the distal LAD was noted to be 3.55 mm from the ICD lead tip (Figure 1). The FFR guidewire was then attached to the Volcano system (s5 system, Volcano, Inc, San Diego, CA). During maximal hyperemia, the FFR across the lesion was 0.77. The pressure guidewire was then disconnected from the power source, and a 4.0 x 15 mm drug-eluting stent (Resolute, Medtronic) was passed over the FFR guidewire into the LAD. The stent would not cross the lesion so the stent was removed and the pressure guidewire left in position with the tip of the wire in the distal LAD. A second guidewire (Cougar LS 0.014, Medtronic) was then advanced through the guide catheter and across the lesion. Thirteen seconds after the Cougar guidewire was inserted into the guide catheter, the patient received a defibrillation from his ICD. At the time of shock delivery, he was in sinus rhythm. Following defibrillation, the Cougar guidewire was advanced across the lesion in the mid-LAD. One minute and three seconds following the first defibrillation, as the Cougar guidewire was positioned into the distal LAD, a second defibrillation shock was delivered. A magnet was then placed over the device to disable tachycardia detections. The coronary stent was then passed over the Cougar guidewire into the mid-LAD where it was successfully deployed. 

On device interrogation, he was programmed VVI 40. Stable lead parameters were noted with satisfactory R-wave sensing of 18.5 mV, a right ventricular capture threshold of 1.0 V at 0.4 ms, and high voltage and pacing lead impedance values of 68 ohms and 572 ohms, respectively. The ventricular sensitivity was programmed nominal. For treatment of tachyarrhythmias, he was programmed with two treatment zones: a ventricular tachycardia (VT) zone 200–219 bpm and a VF zone 219 bpm. Review of the stored intracardiac electrograms (EGMs) from the event shows a low voltage, variable frequency signal in the near-field channel which was initially undersensed by the device starting 13 s prior to the first defibrillation (Figure 2a). The near-field channel was then auto-gained by the device, resulting in the variable frequency, variable amplitude signal nearly saturating that channel (Figure 2b). Close review of the far-field channel also shows a very low-voltage signal seen intermittently (Figure 2c). As the auto-gained signal persisted, it met detection criteria for VF and a 31-J shock was delivered. The variable voltage, variable frequency signal persisted following the first shock resulting in redetection in the VT zone. The signal then suddenly terminated and the shock was diverted. The variable voltage, variable frequency signal then reinitiated and was again seen primarily in the near-field channel. The signal persisted and met detection criteria for VF, resulting in a second 31-J shock being delivered. Following the second shock, the high frequency, variable amplitude signal persisted, but spontaneously terminated before redetection occurred. Following this event, we were unable to reproduce the high-frequency signals seen during the event with arm movement, deep inspiration, patient repositioning, or manual pressure on the ICD pocket. There was no change in lead sensing or impedance with any maneuvers. Six months after the event, he has not received another defibrillation. No external source of EMI was identified in the cardiac catheterization laboratory in the vicinity of the patient.

Discussion

In this case, inappropriate defibrillation occurred as a result of EMI. However, on close inspection of the EGM shown in Figure 2, the EMI is seen most prominently in the near field channel (even before automatic gain control amplification). EMI accounts for 4.5% of all inappropriate shocks.1 In ICDs, EMI from an external source is typically most prominent in the far-field channel and is usually present on both the near-field and far-field channels.4 The EMI in the present case was predominantly on the near-field channel, making an external source of EMI less likely. Internal sources of EMI are much less common, but have previously been described.5 We were unable to identify any equipment within the vicinity of the patient creating detectable EMI. At the time of the shocks, the FFR guidewire was disconnected from the power source and therefore could not by itself contribute to the creation of EMI. Finally, the sensed EMI on the ICD was temporally correlated to advancement of the Cougar guidewire adjacent to the FFR wire. The intermittent nature of the EMI also corresponds to when the Cougar guidewire was advanced and repositioned in the LAD. The etiology of the internal EMI was therefore most likely the result of a weak electrical current generated by movement of the Cougar wire against the FFR guidewire. The current was generated within the FFR guidewire by passage of the Cougar guidewire past a breach in the polytetrafluoroethylene (PTFE) outer insulation near the co-pilot of the guide sheath, allowing creation of the electric current which was transmitted to the distal end of the FFR guidewire, which is not coated by PTFU.6 The low-voltage electrical signals were then detected by the ICD lead tip and treated as VF.
Five unique circumstances contributed to detection of the wire-on-wire interaction and inappropriate defibrillation. First, the ICD lead was implanted in the true apex of the right ventricle. While this is an ideal lead position to obtain optimal defibrillation threshold, this position resulted in the ICD lead tip being positioned less than 4 mm from the distal LAD. Second, the Endotak lead utilizes an integrated bipole for sensing. Use of an integrated bipole creates a larger antenna increasing the chance of detecting low-voltage signals emitted from a device within the LAD. Third, the FFR and Cougar guidewires were positioned in the distal LAD. As the Cougar guidewire was advanced through the guide catheter and along the FFR wire, the low-voltage electric current was transmitted down the FFR wire into the distal LAD. Owing to the proximity of the ICD integrated bipolar lead tip to the distal LAD and intracoronary wires, the low-voltage signals created by the Cougar and FFR guidewire interaction were received by the lead. Fourth, automatic gain control amplification of the near-field channel enhanced detection of the otherwise low-voltage (and underdetected) signal, resulting in the EMI being interpreted as VF and inappropriate shock delivery. Finally, the Vitality 2 generation of ICD does not have a noise response algorithm such as the Dynamic Noise Algorithm (present on more recent Boston Scientific ICDs such as Teligen and Cognis) capable of adjusting automatic gain control when EMI is detected.4

### Conclusions

Manipulation of guidewires within the heart including during intracoronary interventions can induce electrical noise that triggers inappropriate ICD therapies. Caution should be exercised in patients with ICDs with guidewire manipulation in the vicinity of the ICD lead.

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### References