Cardiac implantable electronic device malfunction after deceleration injury without obvious chest trauma

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Introduction

In patients with cardiac implantable electronic devices, mechanical chest trauma can result in device malfunction.[1,2] Possible mechanisms include lead dislodgment, lead fracture, or device header fracture. Accurate identification of the mechanism of device malfunction is key for selecting the appropriate management strategy: lead reimplantation in case of lead dislodgment vs implantation of a new lead with or without lead removal in case of lead fracture vs generator exchange in case of device header fracture. We describe a case of trauma-related device malfunction and present a stepwise approach for clinical problem solving.

KEY TEACHING POINTS

- Device header fracture can happen as a sequela of mechanical chest trauma and can mimic the findings of lead fracture.
- Device header fracture should be suspected when multiple leads fail simultaneously.
- When device header fracture is suspected, chest imaging and intraoperative findings are confirmatory.

Case report

A 72-year-old man with a history of nonischemic cardiomyopathy, congestive heart failure, and significant atrioventricular conduction disease, who underwent implantation of a Boston Scientific (Marlborough, MA) cardiac resynchronization therapy-defibrillator (CRT-D, INCEPTA model N161) 5 years ago, was referred to our electrophysiology clinic for lead management in the setting of malfunction.

The patient was involved in a car accident 5 months prior to presentation, which led to traumatic injuries requiring prolonged care in the hospital, followed by rehabilitation. He was a restrained driver; he rear-ended another car and sustained head and limb injuries, but he did not suffer visible chest trauma. He was eventually discharged home and subsequently had a routine follow-up in his device clinic 2 months later. Device interrogation revealed arrhythmia alerts consistent with nonphysiologic signals on the right ventricular (RV) lead, triggering inappropriate antitachycardia pacing therapy. No shocks were delivered, and there was no noise on the shock electrogram channel (Figure 1). It was also noted that, around the time of these events, the RV pacing impedance increased abruptly from around 450 to 1800 ohms. The RV lead was not capturing at a maximal output. At this point, he was referred to our clinic for further management.

Stepwise approach for clinical problem solving

Step 1

In a patient who presents with findings consistent with device malfunction possibly caused by mechanical trauma, the differential diagnosis includes lead migration, lead fracture, and device header fracture. Based on the characteristic high-amplitude nonphysiologic electrograms noted, lead migration was deemed to be unlikely.[3] Therefore, a fracture at the level of either the lead or the device header was implicated in the device malfunction in this case.

Step 2

An electrocardiogram was performed that showed an atrially paced rhythm at 60 beats/min with intrinsic conduction to the ventricles. The intrinsic QRS width was 100 ms and the QTC interval was calculated to be 480 ms. We subsequently interrogated his device. Interrogation of the atrial lead showed a P-wave amplitude of 5.7 mV (stable compared to prior measurements) and pacing impedance of 957 ohms (increased from around 400 ohms prior to the event) with a pacing

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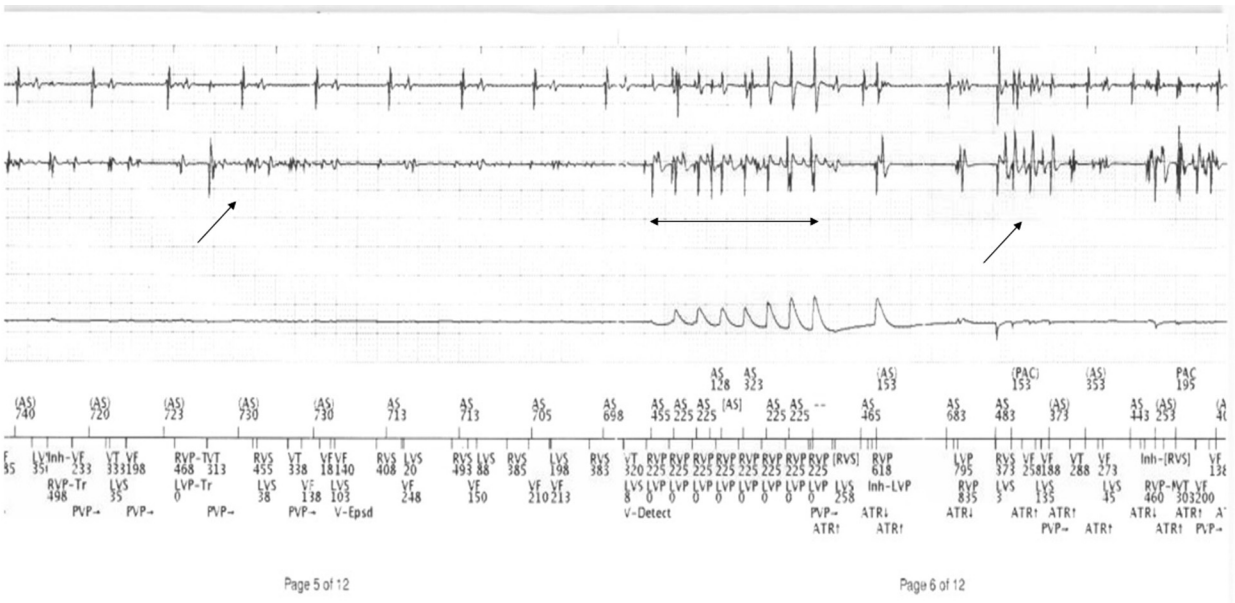

Figure 1 Device interrogation strip showing high-amplitude nonphysiologic electrograms on the right ventricular lead channel (*black arrows*), triggering inappropriate antitachycardia pacing therapy (*black double arrow*). Atrial lead also displays nonphysiologic signals on this tracing.

threshold of 1.8 V at 0.5 ms of pulse width (increased from 0.6 V at 0.5 ms of pulse width prior to the event). RV lead impedance was 2206 ohms, with no capture at 7.5 V at 0.5 ms of pulse width. Left ventricular lead impedance was 1612 ohms, with no capture at 7.5 V at 0.5 ms of pulse width. RV defibrillation impedance was 72 ohms. Temporal trends of pacing impedance in all 3 leads are shown in Figure 2.

Based on the interrogation findings, with a marked abrupt increase in pacing impedance of all 3 leads and failure to capture with RV and left ventricular pacing, the diagnosis would have to be either fracture all 3 leads or device header fracture. For the former to occur simultaneously the location of the fracture would have to be in the extrathoracic portion of the leads, since fracture of the intrathoracic portion of all 3 leads would require a massive traumatic event that would lead to extensive rib fractures and intrathoracic organ damage, which did not happen in this case.

Step 3

A posteroanterior and lateral view chest radiograph (CXR) were performed. There was no evidence of lead fracture on chest imaging. However, as shown in Figure 3A, the lateral CXR showed an abnormal angulation between the device header and the body of the generator.

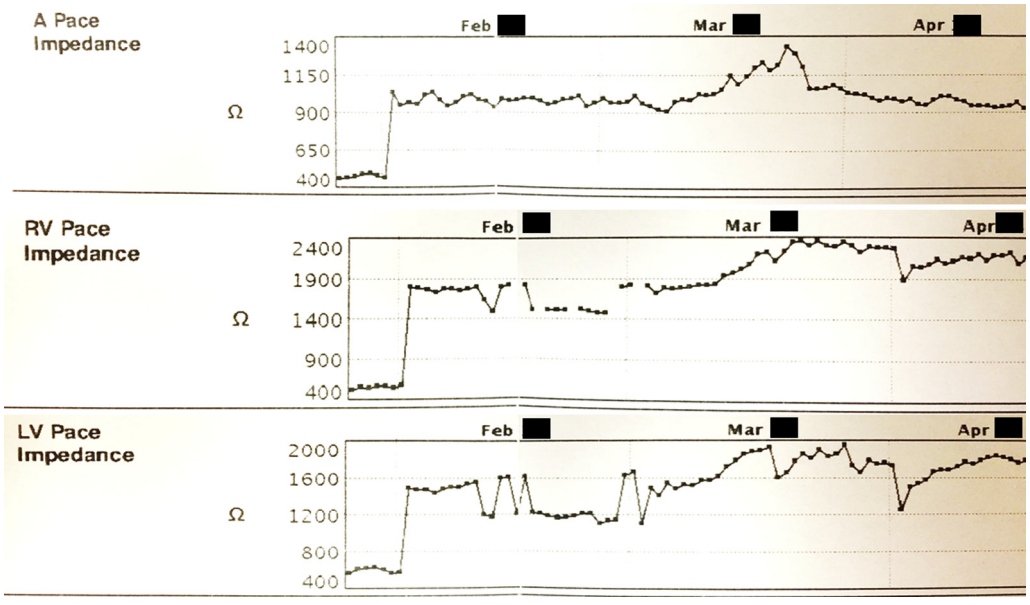

Figure 2 Device interrogation strips showing simultaneous abrupt increase in pace impedance in all 3 leads. A = atrial; LV = left ventricular; RV = right ventricular.

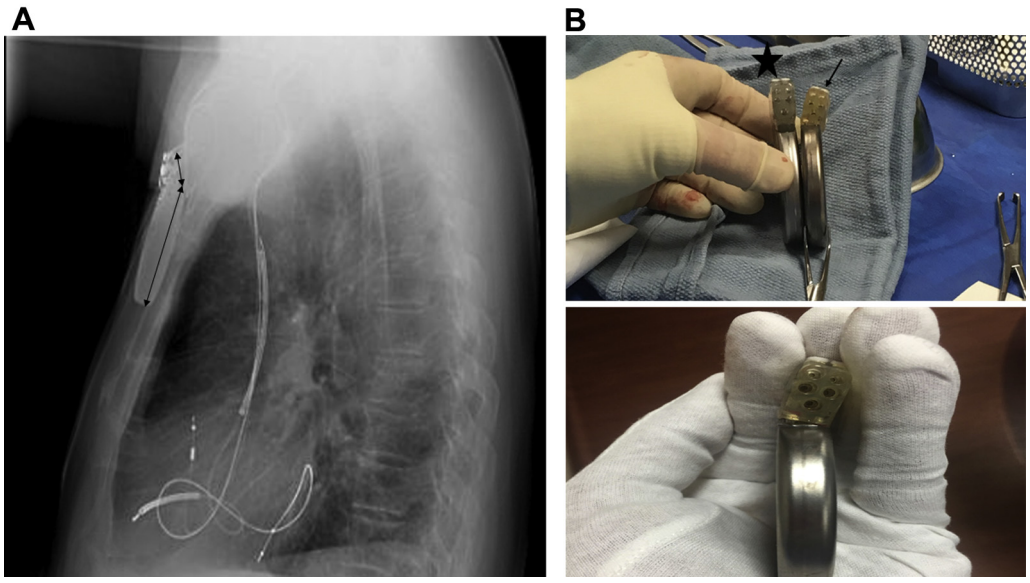

Figure 3 **A:** Chest radiograph (lateral view) showing abnormal angulation between the device header and the body of the generator (*black double arrows*), with no evidence of fracture in any of the leads. **B:** Intraoperative images demonstrating the fractured device header (*black arrow*) in comparison to an intact one from the new generator (*black star*).

At this point, the likely diagnosis was that of a device header fracture. The patient was taken to the electrophysiology lab and his CRT-D battery was explanted. The device header was found to be broken (Figure 3B), confirming our clinical diagnosis. The radiographic and visual appearance of all 3 leads was normal, and electrical testing of the leads through the pacing system analyzer confirmed normal function. A new CRT-D was implanted and connected to his normally functioning leads, resulting in the restoration of normal device function.

Discussion

This case illustrates an extreme presentation of device malfunction following a mechanical trauma to the chest. The patient suffered a deceleration injury while wearing a seat belt with the shoulder strap crossing over the device, and had no apparent major chest injuries. There are limited reports of header fracture in the literature.[4,5] We discuss here a systematic approach to identification of this uncommon entity of device malfunction. Lead dysfunction involving pace-capture failure and impedance rise, in addition to nonphysiologic signals, rules out extracardiac interference, such as from electromagnetic interference. The presence of more than 1 lead involvement in the setting of trauma hints to a limited anatomical location of a potential fracture. Addition of imaging findings, in this case a CXR, revealed an abnormal angulation of the device header, which was strongly suggestive of a header fracture. This was confirmed by further inspection and testing intraoperatively. A stepwise methodical approach to the case allowed accurate clinical diagnosis and avoidance of lead extraction. Device header fracture can be subtle and be often missed. This could lead to a single-lead or multiple-lead involvement based on the extent of the fracture; in this case, the atrial lead was still functioning. A high index of suspicion is critical for diagnosis. It is important, not only for electrophysiologists but for the emergency physicians and trauma surgeons, to recognize that deceleration injury with chest restraint can result in header damage even in the absence of apparent chest trauma. In this case, a routine post-traumatic device interrogation could have helped in the identification of the device malfunction earlier. The patient was not under remote device monitoring, which, if elected could have also aided in earlier detection of the device dysfunction.

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