Fibrous encapsulation of defibrillation electrode and elevated high-voltage impedance in patients with a subcutaneous implantable cardioverter-defibrillator

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Introduction

The use of conventional transvenous implantable cardioverter-defibrillators (ICDs) is well established for prevention of sudden cardiac death (SCD) from ventricular tachyarrhythmias.[1,2] However, transvenous lead failure, venous thrombosis, and infections may complicate follow-up.[3] An entirely subcutaneous ICD (S-ICD) is an option for some patients at SCD risk,[4] whenever there is a desire to avoid transvenous leads or prior transvenous system complications have occurred. The safety and effectiveness of S-ICDs have been demonstrated both in clinical trials and in noncontrolled registration studies; however, such systems are not immune from adverse events.[5–8] Herein, we report 2 cases of fibrous encapsulation of the S-ICD defibrillation shock electrode at 2 years after implantation and its potential impact on the high-voltage impedance and defibrillation test (DT).

Case report
Case #1

A 61-year-old man with ischemic cardiomyopathy was referred for S-ICD implantation for primary SCD prevention. Past cardiac history included ST-elevation acute myocardial infarction with primary angioplasty and stenting of the left circumflex coronary artery (Promus DES; Boston Scientific Inc, St Paul, MN) and left ventricular dysfunction (ejection fraction 30%) under optimized medical therapy. Other medical history included type 2 diabetes mellitus, chronic kidney disease stage 3, hypertension, chronic obstructive pulmonary

KEY TEACHING POINTS

- Fibrous encapsulation on the shock electrode of a subcutaneous implantable cardioverter-defibrillator (S-ICD) system can occur in some individuals

- Increased high-voltage impedance at failed defibrillation test can be corrected by removing the fibrous encapsulation with minimal change in both S-ICD and lead system.

- Impact of fibrous encapsulation on the S-ICD electrode on the lead performance needs to be further studied.

disease, stage 1 lung adenocarcinoma with expected life expectancy >1 year, and obesity with body mass index (BMI) of 34. S-ICD implantation was performed according to the standard protocol, but with the 2-incision technique. The pulse generator (EMBLEM S-ICD, model A209; Boston Scientific Inc, St Paul, MN) and the lead (Q-TRAK SQ 3400; Boston Scientific Inc, St Paul, MN) were used. DT was deferred owing to the presence of a left ventricular apical thrombus. At 2 years post implantation, DT failed with 65 J and 70 J shock on 2 separate ventricular fibrillation (VF) inductions, with high-voltage lead impedances (HVLI) of 153 ohms and 138 ohms, respectively. On both occasions, 200 J biphasic shocks with an external defibrillator were used successfully in terminating VF. Consequently, system revision was undertaken.

At the time of lead revision, there was a dense fibrous tissue encapsulating the shock electrode. Histological findings of cross-section specimens showed connective tissue with collagen fiber arranged in parallel without inflammatory cells (Figure 1). We manually peeled away the fibrous tissue from the lead. The original lead was repositioned slightly toward the right side of the sternum. DT was successful with 65 J
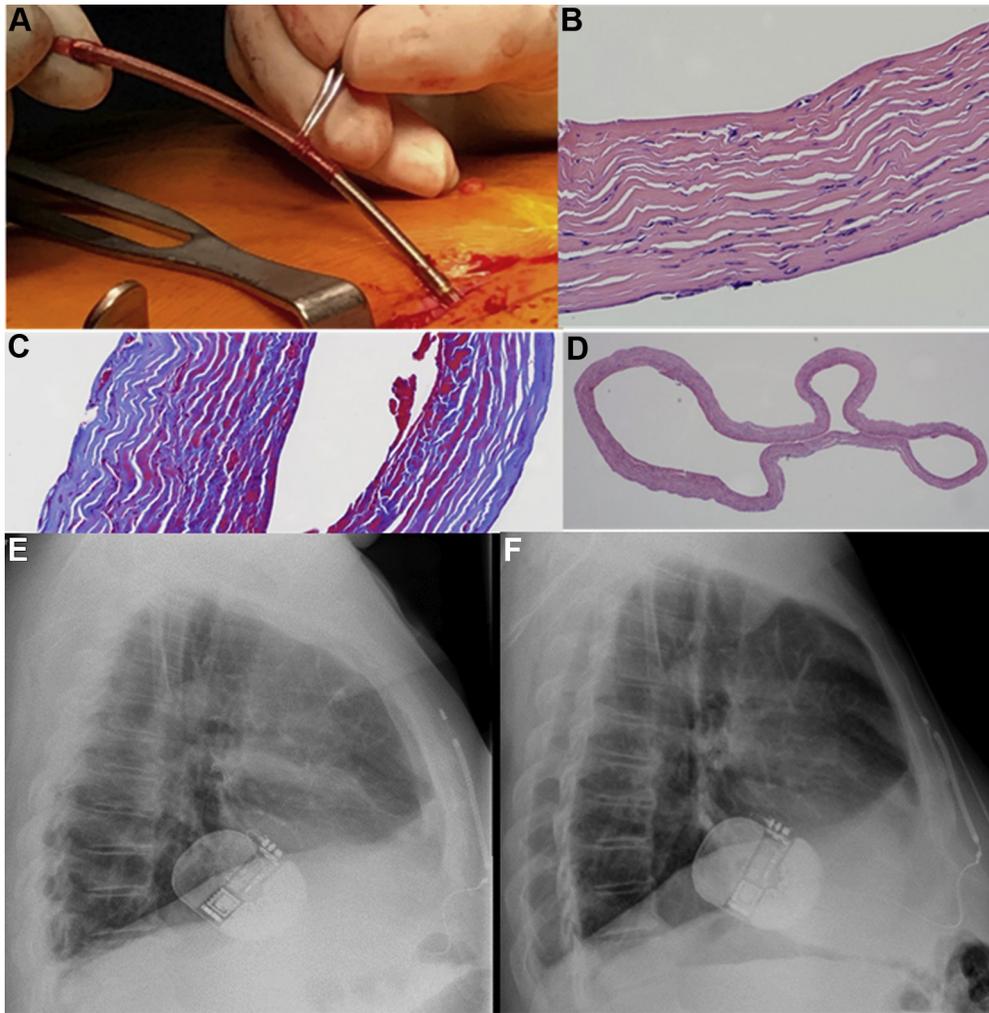
Figure 1   Gross and microscopic view of fibrous sheath around defibrillator shock electrode and lead position. **A:** The defibrillation shock electrode was completely covered with a translucent fiber sheath. **B:** Microscope features: A dense fibrous tissue with collagen fibers in parallel (pink) and fibroblast (blue), hematoxylin–eosin (H-E) stain, 400×. **C,D:** Full cross-section of fibrous sheath in microscope, H-E stain, 40×. **E,F:** Chest radiograph of lead and device positions before (E) and after (F) lead revision.

with a high-voltage impedance of 85 ohms. During follow-up of 2.5 years since implant, there has been no S-ICD therapy for ventricular arrhythmia. The patient died 6 months after S-ICD generator revision from unknown cause.

Case #2

A 58-year-old man with noncompaction cardiomyopathy and left ventricular ejection fraction of 30%–35% received an S-ICD for primary SCD prevention. Past medical history included obstructive sleep apnea and nonischemic dilated cardiomyopathy with BMI of 31. S-ICD implantation was performed according to the standard protocol with the 2-incision technique. The pulse generator (EMBLEM S-ICD, model A209; Boston Scientific Inc, St Paul, MN) and the lead (Q-TRAK SQ 3401; Boston Scientific Inc, St Paul, MN) were used. A successful DT was performed with 65 J shock and HVLI of 98 ohms at implant. Owing to the concerns of S-ICD lead fibrosis and its potential impact on DT as observed in case 1, we performed DT at the 2-year

follow-up. The HVLI was 185 ohms with a 10 J shock from S-ICD. VF was then induced with 50 Hz pacing; however, VF was under-sensed by the device (Figure 2, top left). A 65 J shock with HVLI of 170 ohms from the S-ICD was manually delivered, without success. He was successfully rescued with an external defibrillator at 200 J biphasic shock.

During lead revision, the shock electrode was removed by the simple traction technique and a dense fibrous capsule over the shock electrode was observed, similar to that seen in case 1. Before we removed the shock electrode, a long suture was attached to the suture ring at the distal end of the electrode. When the electrode was pulled out, the suture attached to the lead tip remained in the subcutaneous tunnel with its free end secured outside the wound (Figure 2, top right). After the removal of the fibrous capsule, we could easily reposition the electrode back along the same track by pulling on the suture up along the sternum (Figure 2, bottom). The S-ICD wound was closed. There was no blood or saline injection to the subcutaneous tunnel during this lead revision. A successful DT was performed at 65 J shock with HVLI of
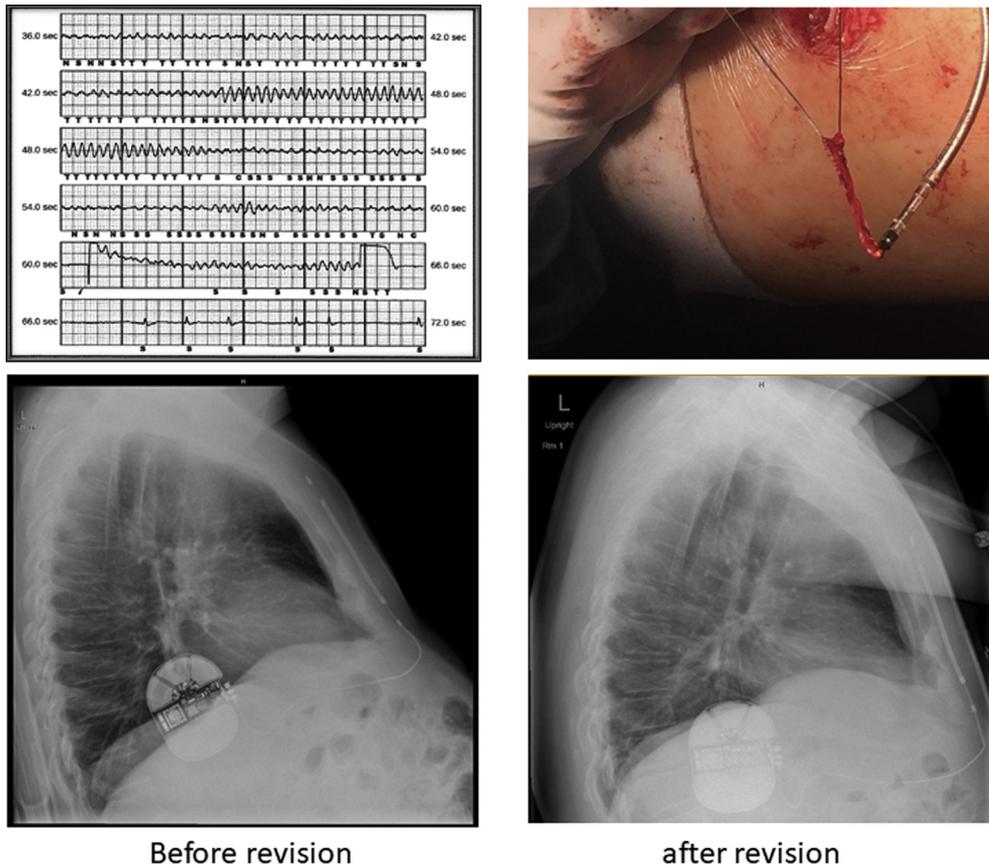
Figure 2 Peeling off the fibrous encapsulation and repositioning the electrode into the same location through a string preattached to the tip of the electrode. **Top left:** Failure to sense ventricular fibrillation by the device, and external defibrillator rescue. **Top right:** The defibrillation shock electrode was completely covered with a fiber sheath. **Bottom:** Chest radiograph to verify the same lead position before and after revision.

77 ohms. During his 1-year follow-up after lead revision, a successful DT was performed with stable HVLI of 87 ohms and 79 ohms at 65 J, respectively. HVLIs measured directly by high-voltage shock for both patients at initial S-ICD implant and before, during, and after S-ICD system revision are summarized in Table 1.

Discussion

We observed dense fibrous encapsulation of defibrillation electrodes in 2 patients with S-ICD after 2 years post system implantation. High HVLIs were seen in both patients with failed DT. Removal of fibrous tissues encapsulating the electrodes with minimal or no changes of S-ICD generator and

Table 1 Clinical characteristics and high-voltage lead impedance measured by high-voltage shock at initial implant and before, during, and after subcutaneous implantable cardioverter-defibrillator system revision

	Case 1	Case 2
Age	61	58
Sex	Male	Male
Indication	Primary prevention ICM	Primary prevention NICM
BMI	34	31
LVEF	30%	30%
LV thickness (septum/posterior wall)	1.2 cm/1.2 cm	1.1 cm/1.1 cm
Renal function (GFR, mL/min/1.73 m^2)	34	>60
DT at initial implant	Not done	98 ohms @ 65 J
Failed DT at 2 years after implant	153 ohms @65J 138 ohms@70J	185 ohms @ 10 J 170 ohms @ 65 J
Revision with successful DT	85 ohms @65J	77 ohms @ 65 J
Pocket revision	No	No
Follow-up after revision	Died of unknown cause at 6 months	Successful DT at 1 year 87 ohms @ 65 J 79 ohms @ 65 J

BMI = body mass index; DT = defibrillation test; GFR = glomerular filtration rate; ICM = ischemic cardiomyopathy; LV = left ventricle; LVEF = left ventricular ejection fraction; NICM = nonischemic cardiomyopathy.

shock electrode position resulted in marked reduction of HVLIs and successful DT. We proposed here that fibrous tissue formation around the defibrillation electrode in some individual patients may cause HVLI elevation and potentially affect S-ICD performance.

Collagen as a biological insulator under physiological conditions has been previously reported.[9] Dense layers of collagen tissues observed in our 2 patients might have resulted in shock energy being wasted, thereby increasing DT. Significant HVLI changes were reported in 41% of transvenous ICD shock electrodes after 3 months of implantation, with changes >12 ohms and >50 ohms in 8% and 0.4% patients, respectively.[10] While factors such as lead orientation, recoiling, and lead contact to myocardium were responsible for HVLI changes in the acute phase, fibrous tissue formation over shock electrodes was considered the main reason for HVLI changes in the weeks and months after implant. HVLI changes of 6–12 ohms could alter the characteristics of the shock waveform, responsible for the 15% increase in DT over time.[10]

No previous studies have examined the impact of fibrous tissue formation on the HVLI in an S-ICD system. The pacing pulse test for calculating shock lead impedance was used for transvenous ICD systems to monitor HVLI changes over time. The test consists of a 4 V positive pulse (20 μS) between the electrode and the can and then a second pulse at 4 V in the opposing polarity.[11] However, this method has not been systematically validated for the S-ICD system. Based on the characteristics of the electric circuit and the calibration constant during manufacturing, engineers formulated a best-fit quadratic equation to improve the accuracy of calculating impedance measurements (personal communication). The HVLI weekly recordings from these 2 patients were downloaded from the S-ICD system; the highest monthly impedances are shown in Figure 3. There was no difference in overall trends when we compared the trend of using the highest monthly impedance and using the weekly impedance. The trends of HVLI changes from both patients appeared to share the following common characteristics. First, there was an initial rise of impedance, which peaked around 3 months after implant. Second, there was a plateau phase from 3 months to 18–20 months. This was followed by a second impedance rise before they peaked at 22–24 months. Third, after lead revision with removal of fibrous tissue, the impedance remained stable at 4–8 months after revision. The mechanisms of early (3 months) and late (22–24 months) HVLI increases remain unknown. Previous animal studies for other biocompatible materials implanted subcutaneously indicated that total fibrous encapsulation occurred by 90 days after implant.[12] Of note, there was an excellent correlation between the impedance calculated by the pacing pulse method and HVLI directly measured by 65 J shock around the time of DT. Long-term follow-up is needed to determine if significant HVLI changes occur later in device life.

Factors influencing DT in patients with S-ICD have not been well studied. In a retrospective study,[13] high S-ICD DT were associated with increased BMI, body surface area, and septal or posterior wall thickness. A high-voltage impedance of >138 ohms appeared to predict patients with high DTs in a univariate analysis. In our cases, failed DTs were observed with HVLI of 138 ohms and 170 ohms, respectively. In a computer modeling study[14] on the determinants of S-ICD efficacy, addition of sub-coil and sub-generator fat increased shock impedance. Calculated DT were increased 3-fold with the addition of 10 mm sub-coil fat. Fibrous encapsulation of the shock electrodes seen in our
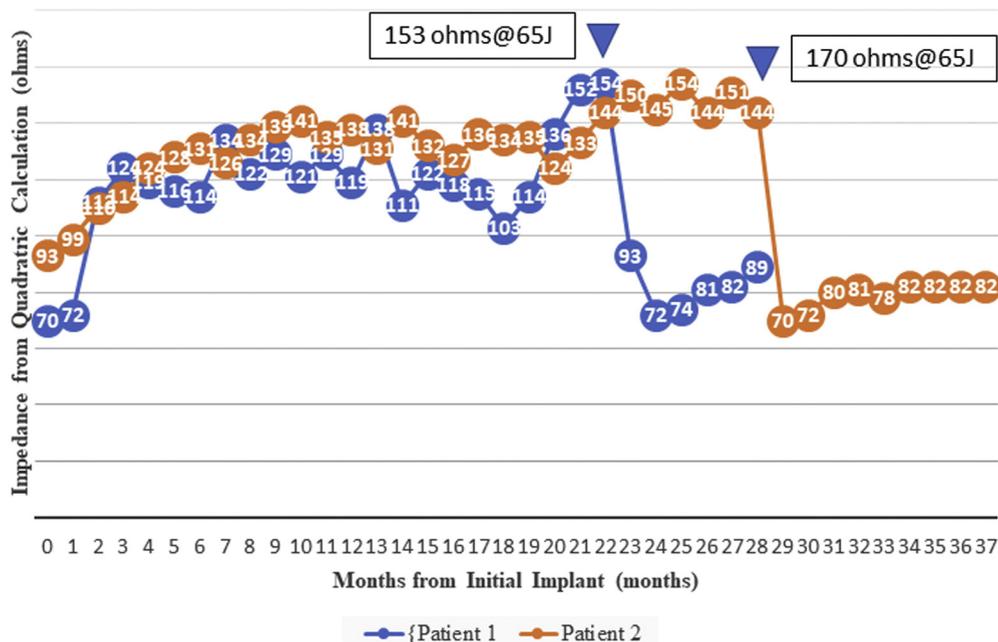

Figure 3   Trends of high-voltage impedance measured by low-voltage pacing pulse during 25 months of follow-up. Arrow indicates the high-voltage impedance with 65 J shock at the time of failed defibrillation test.

cases may function as a "biological insulator" just like the sub-coil fat, to increase the shock impedance and DT. In case 2, we showed marked decrease of HVLI impedance to the baseline level after the removal of the fibrous encapsulation and repositioning of the shock electrode to the same location. This suggests that the fibrous encapsulation was responsible for most of the HVLI rise after implant for this patient. Owing to the subcutaneous location of the shock electrode, there is minimal blood or body fluid pooling from the lead extraction to account for the impedance decrease. Nevertheless, the probabilistic nature of defibrillation added to the complexity and difficulties in determining the true effect of the fibrous encapsulation on the defibrillation threshold.

Limitations

We could not draw any conclusion about whether the finding from these 2 cases is common to other shock leads. A prospective registry on lead impedance over time will help to confirm if this observation is clinically significant in a large population of S-ICD implants.

Conclusion

In conclusion, we showed here that fibrous encapsulation of the S-ICD defibrillation shock electrode occurred in 2 patients after S-ICD implant, which caused both an elevated high-voltage impedance and failed DT. Further studies are needed to determine the frequency with which this phenomenon occurs and whether fibrous encapsulation may adversely affect long-term performance of S-ICD defibrillation systems.

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