First direct human evidence of a probable implantable cardioverter-defibrillator lead-related scar serving as a substrate for ventricular tachycardia

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Introduction

It is well known that after the initial months of implantable cardioverter-defibrillator (ICD) implantation, there is a peak in the incidence of appropriate ICD therapies because of the local irritation effect of the lead on myocardial tissue. Moreover, there is growing evidence of the proarrhythmic effects of pacemaker or ICD leads long after implantation. The pathophysiologic mechanism can be lead fracture, epicardial perforation, local scar formation around the tip, or a change in the activation pattern of the ventricles caused by epicardial or endocardial pacing.[1–4] Proof of lead-related arrhythmogenesis is based on indirect evidence such as identical pace-map from the lead tip, activation or voltage mapping, or noninducibility of the tachycardia after lead removal.[2] The aim of this report is to present the first direct human pathologic evidence of a probable ICD lead-related scar causing ventricular tachycardia (VT).

Case report

A 50-year-old man with a history of myocardial infarction in 1991 and coronary artery bypass graft surgery 1 year later underwent dual-chamber ICD implantation for secondary prevention in 2003. He was suffering from chronic heart failure of ischemic origin and recurrent VTs, so VT ablation was performed at another institution in 2008. The ICD system was removed completely because of a generator change-related pocket infection in 2008, and a new dual-chamber ICD system was implanted from the opposite side. In the 2 years after reimplantation, he presented with multiple, appropriate, effective ICD therapies because of sustained monomorphic VTs with at least 5 different morphologies having different cycle lengths (300, 480, 420, 540, and 560 ms). Left ventricular ejection fraction was 26%. Coronary angiography revealed occluded native coronaries and intact bypass grafts. The patient was placed on the waiting list for orthotopic heart transplantation in 2009. Because the VTs were drug resistant despite administration of amiodarone and mexiletine and were causing multiple ICD discharges, radiofrequency catheter ablation (RFCA) was performed at our institution in 2010. Multiple inducible VT morphologies were observed during the study, and electroanatomic mapping identified an extensive low-voltage area corresponding to the anteroseptal–apical scar tissue of the remote myocardial infraction. After isolated late potentials and exit points were successfully targeted by RFCA, only 1 sustained VT morphology remained.

The ultimate VT morphology closely resembled the paced QRS morphology. It also was clinically relevant according to a previous ECG of a spontaneous VT episode with a cycle length of 560 ms (Figure 1). Further pace-mapping located this VT close to the screw-in tip of the steroid-eluting defibrillator lead (Guidant Endotak Reliance G 0185, Boston Scientific, St. Paul, MN) in the apical aspect of the interventricular septum. The area around the lead tip was then approached from both the left and right sides of the interventricular septum. A magnetic navigation enabled catheter was used in the left ventricle,[5] but the ablation attempts were not successful (Figure 2). A new ventricular pace/sense electrode was implanted at the end of the procedure because of sensing failure of the shock lead after ablation. Three weeks after ablation, the patient underwent orthotopic heart transplantation. This provided us the unique opportunity to investigate the explanted heart with the electrode tip *in situ* and to directly visualize the tip-related scar (Figure 3), which corresponded to the site of origin

KEYWORDS Implantable cardioverter-defibrillator lead proarrhythmia; Ventricular tachycardia substrate; Pathology specimen

ABBREVIATIONS ICD = implantable cardioverter-defibrillator; **RFCA** = radiofrequency catheter ablation; **VT** = ventricular tachycardia (Heart Rhythm Case Reports 2015;1:10–12)

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KEY TEACHING POINTS

- Implanted ICDs can have a clinically relevant proarrhythmic effect, which can manifest long after device implantation. Although rare, scar formation around the lead tip can serve as a substrate for ventricular tachycardia.
- Comparing the ECG of the clinical ventricular tachycardia to the paced QRS morphology can be the clue to correct diagnosis. Pace-mapping can help localize the source of origin.
- Pathologic examination of the explanted heart directly clarified the substrate of this particular ventricular arrhythmia. Even use of a steroid-eluting lead could not prevent significant scar formation around the tip.

previously identified by pace-mapping. As clearly demonstrated by the pathology specimen, the location of the scar was unreachable by any ablation catheter, thus explaining why RFCA was unsuccessful in this area. Moreover, this scar was well separated from the infarct-related scar, which was unrelated to the ultimate tachycardia (Figure 3).

Discussion

To the best of our knowledge, this likely tip-related scar is the first direct human pathologic evidence of lead-related arrhythmogenesis, which was identified as a substrate of a clinically relevant VT. One can speculate that the lead tip probably caused local damage to a tributary coronary branch with consequent hemorrhage and scar formation around the tip, and this could be the pathophysiologic mechanism. Unfortunately, we do not have straightforward imaging evidence that this scar did not exist previously and that the scar developed only after ICD lead implantation, but we believe that our data are quite compelling. In the setting of unexplained VTs or VT storm, this possible lead complication deserves more attention. Given the possible mechanism, even a steroid-eluting lead design cannot prevent significant scar formation around the tip.[4]

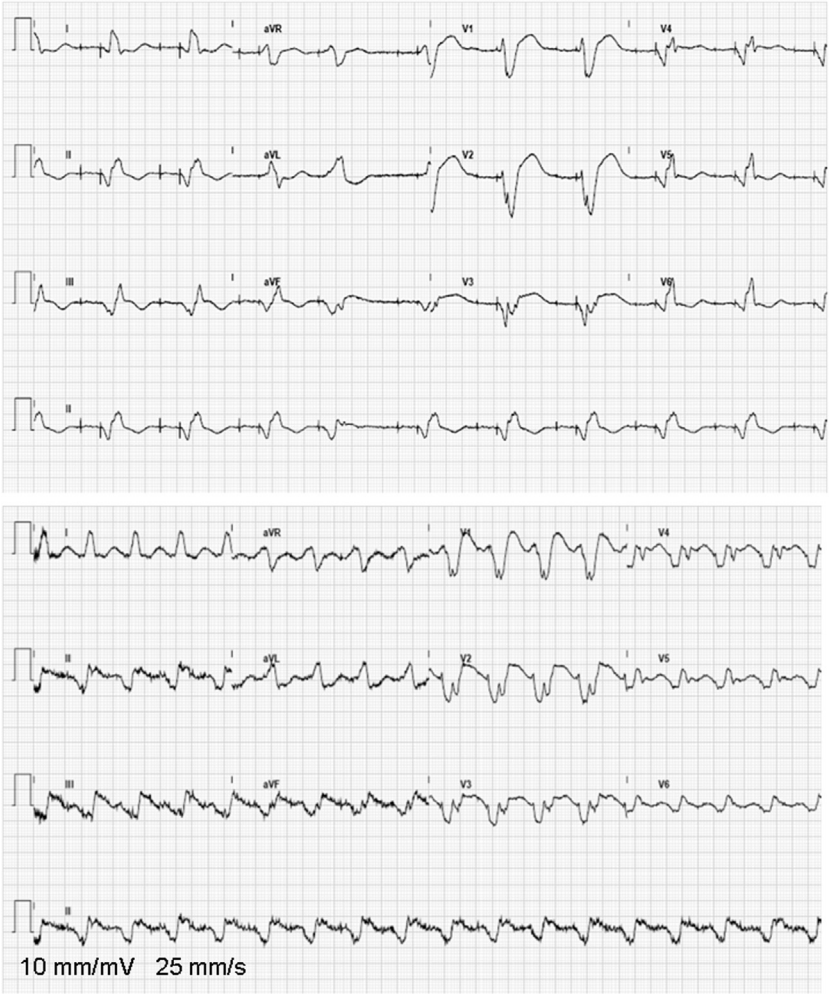

Figure 1 Twelve-lead ECGs with paced QRS complexes and during a spontaneous sustained ventricular tachycardia (VT) episode with a cycle length of 560 ms. Note that the ventricular pacing morphology, albeit not fully identical, is closely related to the morphology of the VT. The slight difference in QRS morphologies is explained by the anatomic location of the scar (see Figure 3 for details).

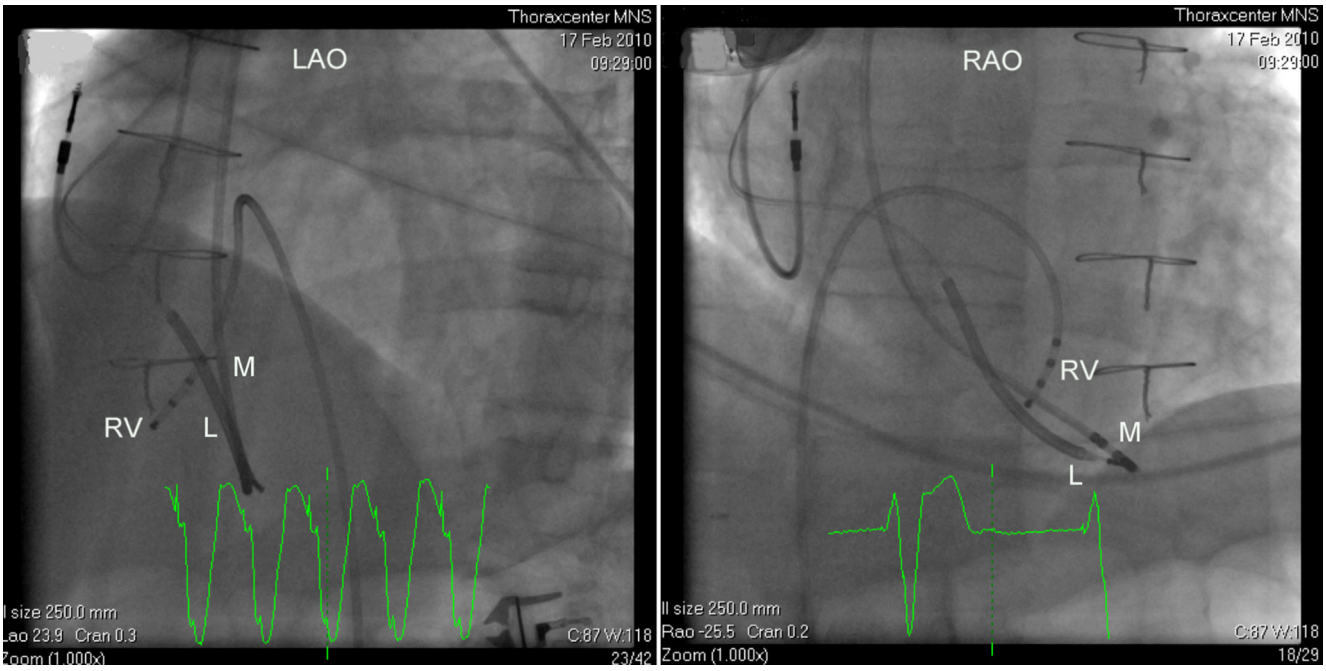

Figure 2 Fluoroscopic images in the left anterior oblique (LAO) and right anterior oblique (RAO) projections depicting the mapping/ablation catheter (M) placed in the right ventricle via transjugular access. The site of origin of the ventricular tachycardia is in close proximity to the tip of the shock lead (L). RV = diagnostic catheter in the right ventricle.

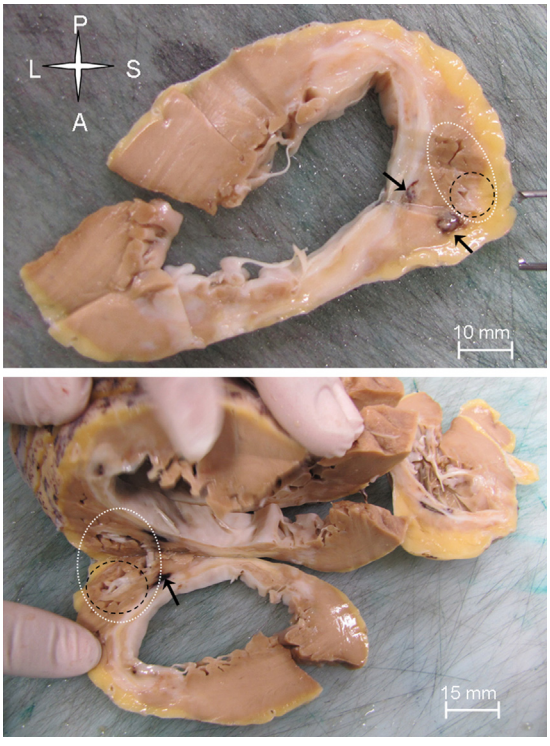

Figure 3 **Top:** Oblique cross-sectional macroscopic pathology specimen of the explanted heart at the apical level of the right ventricle *(white dotted circle)* demonstrating scar tissue *(black dotted circle)* around the tip of the explanted implantable cardioverter-defibrillator lead and the 3-week-old necrotic, hemorrhagic lesions of 2 radiofrequency applications *(arrows)*. One lesion was created from the left ventricle and the other from the right ventricle. The location of the scar explains why a fully identical pace-map could not be achieved, because this region was unreachable by any of the catheters. Note that the extensive scar of the old myocardial infarction is separated from this scar. **Bottom:** Another aspect of the explanted heart showing the distinct scar caused by the tip and the extensive infarction scar tissue. Orientation: A = anterior; L = lateral; P = posterior; S = septal.

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