Images and Case Reports in Arrhythmia and Electrophysiology

Examination of Explanted Heart After Radiofrequency Ablation for Intractable Ventricular Arrhythmia

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Intractable ventricular tachycardia (VT) and ventricular fibrillation (VF), often referred to as electrical storm (ES), is a life-threatening emergency requiring immediate intervention.[1] Patients presenting with ES often suffer from severe cardiomyopathy but may have structurally normal hearts with ion channelopathy. Common triggers of ES include myocardial ischemia, acute congestive heart failure, electrolyte abnormalities, and drug toxicity. Patients with left ventricular assist devices (LVADs) frequently develop ventricular arrhythmia and ES refractory to antiarrhythmic therapy.[1] One study found that sustained VT or VF occurred in 52% of patients with a continuous-flow LVAD (HeartMate II).[2] Although LVAD therapy can prevent hemodynamic collapse resulting from sustained ventricular arrhythmia, patients may develop hemodynamic instability and decreased flow rates as a result of right ventricular dysfunction. If VT/VF cannot be controlled with antiarrhythmic therapy, then urgent electrophysiological study and ablation are indicated. In this unique report, we describe a successful substrate ablation of recurrent drug refractory ES in a patient with a HeartMate II LVAD awaiting orthotopic heart transplant. After orthotopic heart transplant, gross pathological examination of the explanted heart was performed.

Our Case

A 40-year-old man with nonischemic cardiomyopathy, implantable cardioverter-defibrillator, and a HeartMate II LVAD was admitted to the hospital in anticipation of heart transplantation. On physical examination, the patient was in no acute distress. Bibasilar crackles were noted at both lung bases. ECG revealed normal sinus rhythm with ventricular pacing. On hospital day 2, the patient developed recurrent sustained ventricular arrhythmia. No reversible cause was noted and he was transferred to the intensive care unit for treatment of ES. VF persisted despite treatment with amiodarone and lidocaine infusions. While in VF, LVAD flow rates deteriorated, and defibrillation was frequently required to restore normal sinus rhythm. The patient was referred for urgent electrophysiological study and ablation.

Methods

A transeptal approach was used to access the left ventricle, and an irrigated tip catheter was used for substrate mapping and subsequent ablation. An electroanatomic mapping system (CARTO Biosense Webster) was used to create a 3-dimensional voltage map of the left ventricle during sinus rhythm. During electrophysiological study, frequent

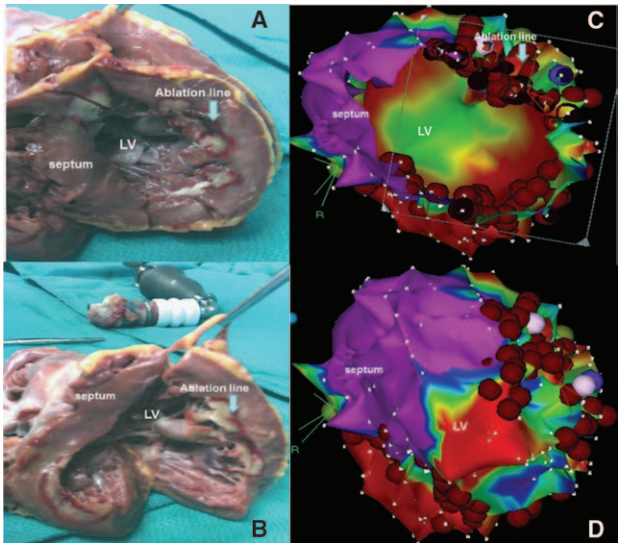

Figure. Macroscopic anatomy and electroanatomic voltage mapping of the left ventricle (LV). Normal voltages (>1.5 mV) are color coded in purple, and abnormal low-amplitude potentials are color coded in blue to red. Scar is defined as red (0.5 mV). Radiofrequency applications (maroon dots) are placed along scar border zones and in areas where late potentials are recorded. **A** and **B**, Macroscopic anatomy of the explanted heart (left anterior oblique [LAO] view equivalent). Note that the lateral edge of the ablation line is interrupted by the insertion of papillary muscle. In (**B**), note the left ventricular assist device HeartMate II inflow cannula in background, with remnant of myocardial tisssue attached. **C**, LAO view of LV. Cutting plane view through the LV demonstrates that ablation lesions correlate with necrosis on gross anatomic specimens. Note that similar to the macroscopic anatomy, there is also interruption of the ablation line at 3'o clock because of papillary muscle insertion. **D**, Same as (**C**) but without the cross-sectional cut.

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monomorphic ventricular premature complexes were not observed. Furthermore, hemodynamic instability during VT/VF precluded efforts to perform entrainment or activation mapping. Standard substrate mapping techniques were used to identify areas of scar border zone and isolated late potentials.[3] The final voltage map of the left ventricle revealed extensive inferobasal, inferolateral, and apical scarring (Figure, C–D). Apical scarring was thought to be secondary to surgical suture lines in the region of the LVAD inflow cannula.

Radiofrequency energy (up to 40 W) was applied around scar border zones and bridging areas between dense scar. The patient tolerated the procedure well and was transferred back to the intensive care unit. After ablation, no sustained ventricular arrhythmia was noted. The patient received orthotopic heart transplant (OHT) 10 days after ablation. Histopathologic examination of the explanted heart revealed punctuate areas of myocardial necrosis. Lesions on electroanatomic mapping correlated with necrotic lesions noted during examination of the explanted heart. Of interest, high-powered radiofrequency lesions delivered using an irrigated catheter failed to produce transmural necrosis (Figure, A–B).

Discussion

In this case, we describe the successful ablation of recurrent, drug refractory ES in a patient with an LVAD and hemodynamic compromise. Shortly after the ablation procedure, the patient underwent orthotopic heart transplant, providing the unique opportunity to examine the explanted heart. Several investigators have reported successful ablation of ES by targeting the ventricular premature complexes that trigger arrhythmia.[4] In our case, substrate mapping and ablation were required because no triggering ventricular premature complexes were apparent. We conclude that ablation of ES is feasible through the use of substrate mapping techniques. Examination of the explanted heart demonstrated that radiofrequency lesions marked on the electroanatomic map correlated well with punctate necrosis found along the endocardium of the left ventricle. These radiofrequency lesions, delivered at high power with an irrigated catheter, did not produce transmural necrosis.

Disclosures

None.

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KEY WORDS: ablation ■ cardiomyopathy ■ devices for heart failure ■ electrophysiology mapping ■ ventricular arrhythmia

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