Ablation of Ventricular Tachycardia Originating From the Papillary Muscle of the Left Ventricle Early After Heart Transplantation

Vivek Iyer, MD, MSE; Arthur Reshad Garan, MD; Donna Mancini, MD; Hasan Garan, MD; William Whang, MD, MS

Case Presentation
A 46-year-old man 2 months status post heart transplant for cardiac amyloidosis was admitted with recurrent palpitations. There was no reported ventricular dysfunction or history of ventricular arrhythmia in the donor, a 26-year-old man who died in a motorcycle accident.

Monomorphic ventricular tachycardia (VT) was observed in the second week after heart transplant. Echocardiogram revealed preserved graft function with left ventricular ejection fraction of 55%, and endomyocardial biopsy revealed no evidence of rejection. The patient was treated with metoprolol with reduction in his VT burden, but he subsequently signed out of the hospital against medical advice. Three weeks later, he presented to the emergency department with recurrent palpitations. Cardiac monitoring showed frequent ventricular premature complexes and VT (as many as 30 beats), with right bundle branch block morphology QRS configuration and rightward frontal axis—identical to the early postoperative arrhythmia (Figure, panel A). An idiopathic left ventricular VT was suspected. He was referred for electrophysiology study.

A detailed anatomic map of the left ventricle was made using a 64-element phase array intracardiac echocardiography catheter (AcuNav catheter and CARTOsound software, Biosense Webster, Diamond Bar, CA). An electroanatomic contact map revealed no areas of scar. A candidate site with early activation was seen in the anterolateral left ventricle (Figure, panel C) with a sharp prepotential on the local electrogram preceding the QRS complex by 26 ms. The pace map from this site matched only 9 of 12 leads. The catheter tip at this time was clearly visualized using the intracardiac ultrasound catheter on the anterolateral papillary muscle tip. Radiofrequency energy application at this site resulted in transient induction of wide complex tachycardia with similar morphology to the clinical arrhythmia, which terminated with continued radiofrequency delivery. No arrhythmia was inducible after ablation. At 12 months’ follow-up, he remained free of arrhythmia without β-blocker therapy.

Discussion
VT can occur relatively early in the post-transplant state (associated with graft rejection) or late after transplant (eg, in association with transplant vasculopathy). We report a case of post-transplant idiopathic VT originating from the anterolateral papillary muscle.

The papillary muscle has been recognized as a focus for idiopathic VT. Doppalapudi et al1 published the experience of their group consisting of 290 consecutive patients referred for focal ventricular premature complex or VT, with 7 originating from a left ventricular papillary muscle. Our case shares many common features with the published experience with this arrhythmia in native hearts including sharp prepotentials at the successful ablation site,1 a poor pace map at this site,2 and the usefulness of intracardiac ultrasound for identifying effective catheter contact on the complex 3-dimensional surface of the papillary muscle.3

However, we entertained 3 possible factors in the development of idiopathic VT that were unique to our patient given its occurrence in the transplanted heart. First, autonomic denervation was a potentially important factor. With the bivacal technique, both vagal and sympathetic inputs to the heart are interrupted; the altered autonomic state of the transplanted heart may have unmasked a previously silent focus of arrhythmia in the donor. Second, given the donor’s history of motor-vehicle accident, the development of occult myocardial trauma may have contributed to the substrate for clinical arrhythmia, as recently reported in a pediatric patient.4 Finally, we cannot rule out that a substrate for arrhythmia was present in the donor and transplanted into our patient (or indeed, whether a symptomatic episode occurred at the time of the donor’s motor vehicle accident).

Conclusion
We report a case of VT early post–heart transplant, which was mapped to the anterolateral papillary muscle and ablated with intracardiac ultrasound guidance. Clinicians should recognize the potential for idiopathic VT in transplanted hearts and the feasibility of catheter ablation for effective treatment.
Disclosures
None.

References

KEY WORDS: catheter ablation ◼ heart transplantation ◼ papillary muscles ◼ tachycardia, ventricular

Figure. Ventricular tachycardia (VT) early after heart transplantation. A, Twelve-lead ECG of the clinical VT shows frequent monomorphic ventricular premature complexes (VPCs) and short salvos of VT. B, Intracardiac ultrasound demonstrates the ablation catheter as an echo-dense linear structure making contact with the tip of the anterolateral papillary muscle. C, Intracardiac electrogram at this site shows a sharp prepotential and local activation preceding electrocardiographic QRS onset during the VPC by 26 ms. Ablation here led to transient induction of VT followed by termination and noninducibility of the arrhythmia. Abl distal indicates distal ablation catheter bipolar electrogram; and Abl proximal ablation catheter bipolar electrogram.
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