Successful catheter ablation of recipient left ventricular tachycardia 26 years after heterotopic heart transplantation



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Introduction

In heterotopic heart transplantation (HHTx), the donor heart is connected to the recipient heart in a parallel fashion, acting as a biological biventricular assist device. This technique has been developed before the cyclosporine era to reduce early postoperative death related to graft failure. HHTx is associated with a lower long-term survival compared with orthotopic heart transplantation and is now only used for selected patients with high pulmonary vascular resistance as an alternative to heart-lung transplantation or in case of major donor-to-recipient size mismatch.¹ HHTx survivors are exposed to a risk of ventricular arrhythmias (VAs) originating from the native heart. Only scarce literature exists regarding their management, which is crucial to prevent hemodynamic compromise and thrombus formation. Here, we report a case of successful recipient ventricular tachycardia (VT) catheter ablation and we discuss the clinical challenges that arise from the particular setting of HHTx.

Case report

A 71-year-old man was referred to our institution for chest pain and progressive dyspnea on effort, 26 years after HHTx. He is now the last survivor who underwent HHTx at our center and probably one of the longest-living worldwide. In 1990, the HHTx was performed for end-stage coronary artery disease resulting from a large anterior myocardial infarction in 1987. According to the description of the biventricular assistance configuration of HHTx by Frazier,² donor and recipient aorta and pulmonary arteries were anastomosed end-to-side after anastomosis of donor and

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recipient left atria. In 1999, the patient experienced a lateral myocardial infarction of the native heart and underwent stenting of the native left main and circumflex arteries. He remained asymptomatic until 2009 when a first VT originating from the high lateral wall of the left ventricle of the recipient heart was successfully ablated by radiofrequency (RF) through a retroaortic approach. Following ablation, electrocardiogram (ECG) of the native heart showed sinus rhythm with first-degree atrioventricular block (AVB), complete right bundle branch block, and left anterior fascicular block with anteroseptal and high lateral Q waves. At that time, the recipient heart was providing most of the total cardiac output. The procedure allowed full recovery of the previous functional status, and left ventricular ejection fraction (LVEF) of the recipient heart measured by echocardiography and magnetic resonance imaging remained stable at 20% on further evaluations.

In 2016, the patient experienced angina pectoris. Hemodynamic status was normal with no signs of congestive heart failure. Twelve-lead ECG revealed 2 dissociated rhythms: a regular, monomorphic, wide QRS-complex tachycardia with right bundle branch morphology at 136 beats per minute (bpm); and a narrow QRS-complex rhythm at a rate of 65 bpm, the 2 rhythms representing the superimposed ventricular activation patterns of the recipient and donor heart, respectively (Figure 1A). Echocardiography showed severely depressed LVEF of the native heart and normal LVEF of the graft. Pharmacologic treatment with amiodarone failed to terminate VT durably and caused paroxysmal asystole of the donor heart, which resulted in temporary clinical deterioration and led to repeat catheter ablation.

Considering the unusual anatomy, transseptal puncture of the recipient interatrial septum was performed under careful transesophageal echocardiography and radioscopic guidance, to achieve stable catheter positioning on the lateral basal and apical left ventricle (LV) endocardium. A conventional retrograde aortic approach via the right femoral artery was also used for LV outflow tract, septum, and lateral wall mapping.

Recipient LV endocardial mapping was performed with a PentaRay NAV catheter and a SmartTouch open-irrigated

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KEY TEACHING POINTS

- Interpretation of heterotopic transplant electrocardiography can be challenging owing to the presence of 2 superimposed rhythms.
- Ventricular arrhythmia originating from the native heart is a common complication that can lead to functional deterioration and thrombus formation.
- Echo-guided transseptal approach in the recipient heart is feasible and facilitates ventricular tachycardia radiofrequency ablation, whereas the biventricular assistance configuration provides hemodynamic stability during tachycardia that allows complete activation mapping.

ablation catheter, using the electroanatomic CARTO 3 mapping system (Biosense Webster, Diamond Bar, CA). Voltage mapping revealed a large endocardial ischemic scar area in the anteroapical and laterobasal walls of the recipient LV (Figure 1B-C). Hemodynamic tolerance and stability allowed for complete activation mapping of the clinical VT. A critical isthmus containing fragmented, slow-conducting, and prolonged mid-diastolic potentials was identified within the scar (basal to mid segment of the lateral wall, Figure 2A). Propagation mapping revealed a dual-loop figure-8-type macroreentry with a cycle length of 440 ms (Video 1, available in Online Data Supplement). The clinical VT was terminated after 3 RF applications at 35 W within the protected isthmus (Video 2, available in Online Data Supplement). Programmed ventricular stimulation induced 3 other VTs arising from the inferolateral, anteroseptal, and basal anterior walls, all terminated by RF applications in the presence of diastolic potentials. Programmed ventricular stimulation failed to induce any further tachycardia. After VT termination, asystole of the recipient heart owing to complete infrahisian AVB was initially observed, followed by a slow ventricular escape rhythm (Figure 2B). An increase of 15 mm Hg in mean arterial pressure was obtained by recipient right ventricular pacing and when ventricular escape was present. The next day, 12-lead ECG showed normal sinus rhythm of the donor heart and apparition of a wide 30-bpm QRS activity, corresponding to a ventricular escape rhythm of the recipient heart (Figure 2C). A few days after catheter ablation, the patient was asymptomatic and was discharged. He had no VT recurrence after 2 months.

Discussion

Atrial tachyarrhythmias are well-known complications of orthotopic heart transplantation and abundant literature exists regarding their management. However, management of arrhythmia occurring in HHTx has been mainly described with individual observations.^{3–7} Among 16 patients who underwent HHTx at their center, Tagusari and colleagues⁸

reported a 25% cumulative incidence of VAs after HHTx at a mean follow-up of 4 years. However, in a more recent historical cohort of 20 HHTx recipients with a 25-month mean follow-up, only 1 patient experienced VA.¹ Nonetheless, most VAs in HHTx are related to the underlying recipient heart disease⁹ and are clinically challenging because the recipient heart often remains functionally important. HHTx, which was originally performed as a biological LV assist device,¹⁰ evolved into a biventricular assist configuration after the report of 2 patients who experienced heart failure related to recipient heart ventricular fibrillation.¹¹ In the later configuration, recipient VAs are often well tolerated and their diagnosis requires careful physical examination searching for the disappearance of the irregular pulse pattern related to donor heart parasystole. Discrimination of the 2 superimposed rhythms on standard 12-lead ECG can be difficult, and some authors used echocardiography to identify which heart was concerned by the arrhythmia.¹² In our case, diagnosis of VT originating from the normally positioned recipient heart was strongly suggested by the left precordial leads and was confirmed by a magnetic resonance imaging study during VT (not shown). Right bundle branch, right axis QRS morphology indicated a lateral LV wall origin, a finding consistent with the history of lateral myocardial infarction. Available therapies included antiarrhythmic drugs, implantable cardioverter-defibrillator therapy, and catheter ablation. Considering amiodarone failure and despite the moderate impact of VT on functional status, we thought that a redux catheter ablation procedure of the recipient heart VT would improve hemodynamics and prevent thrombus formation.

To our knowledge, this is the first report of successful ablation of recipient heart VT originating from the LV by a transseptal approach. Acting as a biventricular assist device, the donor heart allowed for detailed activation mapping of the recipient heart's unstable VT and consequently its termination by RF applications on the critical isthmus, with no hemodynamic compromise during the procedure.

After a short period of asystole, complete recipient AVB with ventricular escape rhythm was observed. Despite reports suggesting a potential benefit of linked pacing of donor and recipient hearts,^{13,14} the risk of superior vena cava or donor right atrium obstruction owing to the presence of pacing leads was a major concern for this patient regarding the narrowness of his venous channels. Consequently, and because we thought that the escape rhythm was hemodynamically efficient, we decided to delay evaluation for pacing indication that might include leadless ventricular pacing of the recipient heart. Moreover, good hemodynamic tolerance of the arrhythmia, potential risks of donor heart farfield R-wave sensing, and donor heart ventricular fibrillation induced by electrical cardioversion accidentally delivered on the donor-heart T wave made implantable cardioverterdefibrillator therapy not a beneficial option.

Although orthotopic heart transplantation is almost exclusively used worldwide, HHTx may still represent a viable alternative to heart-lung transplantation for the growing

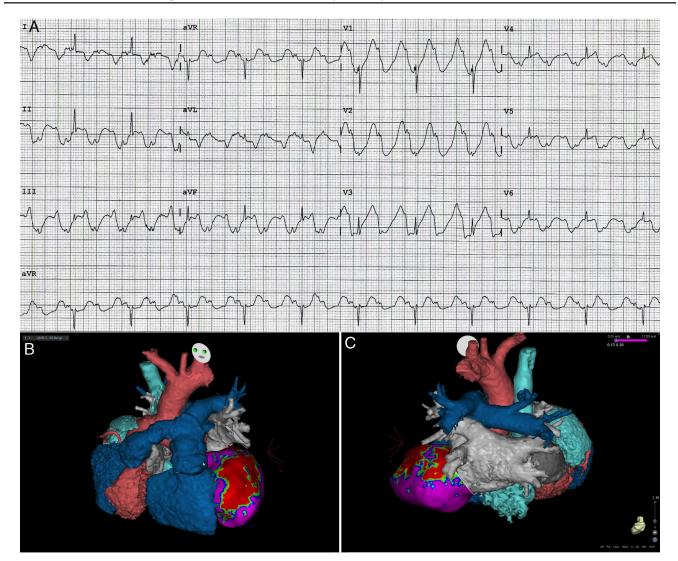


Figure 1 Initial electrocardiogram (ECG) presentation and merging of the computed tomography scanner and voltage electroanatomic mapping of the native left ventricle. **A:** Standard 12-lead ECG showing 2 distinct superimposed patterns of ventricular activation: 1 wide QRS-complex tachycardia of right bundle branch block morphology at 136 beats per minute (bpm), consistent with ventricular tachycardia originating from recipient left ventricle, and a narrow QRS-complex rhythm at 65 bpm corresponding to the donor heart sinus rhythm. Negativity of the wide QRS complex in leads I, aVL, and V₅ and V₆ suggests left lateral origin. **B:** Three-dimensional computed tomography reformation of a left anterior oblique view of the heart merged with the bipolar voltage map of the recipient left ventricular (LV) endocardium using the electroanatomic CARTO 3 mapping system. Heterotopic heart transplantation is in its biventricular assist configuration: both donor aorta (light red) and pulmonary artery (deep blue) are directly connected to the corresponding recipient vessels. The native heart, at the right of the image, is markedly dilated compared with the donor heart. Mapping is achieved by both a transseptal approach under careful transesophageal echocardiography guidance and a retrograde aortic approach. Purple indicates voltage >0.25 mV, corresponding to viable endocardium, and red indicates low voltage <0.12 mV, corresponding to dense scar. A broad low-voltage scar area of the LV endocardium is identified on anteroseptoapical and lateral walls and corresponds to the infarcted territory. **C:** Posteroanterior view. Donor (white) and recipient (gray) left atria are widely anastomosed. Recipient right atrium receives the native inferior and superior vena cava (light blue). Note the presence of a protected isthmus within the lateral LV scar, bordered by anterolateral dense scar and laterolateral mitral annulus.

number of candidates with high pulmonary vascular resistance¹ or in case of size mismatch between a small graft and the recipient in order to avoid marginal grafts underuse in graft shortage situations. Knowing that catheter ablation of recipient heart VT is feasible might be encouraging for further development of HHTx.

Conclusion

This case illustrates the feasibility of catheter ablation of recipient LV reentrant tachycardia in HHTx via transseptal puncture of the native interatrial septum. VT mapping was facilitated by the presence of the donor heart, which acted as a biological biventricular assist device.

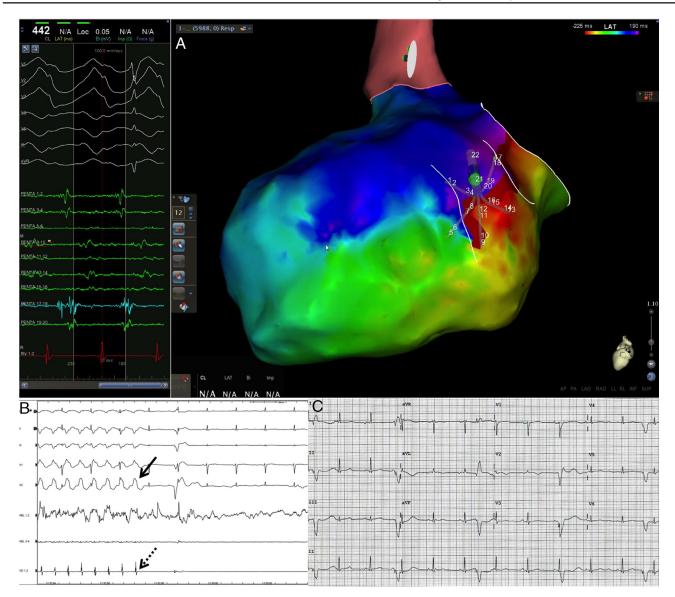


Figure 2 Mapping and ablation of left ventricular tachycardia (VT) in recipient heart. **A:** Posterolateral view of an activation map of the clinical VT. Redcolored areas correspond to the earliest points within the VT cycle length and purple areas to the latest points (see color scale in the upper right quadrant; LAT = location activation time). Presence of all colors in the recipient left ventricle suggests macroreentry. A slowly conducting laterobasal isthmus within the scar containing both purple and red is identified (flanked by 2 white lines). A 20-pole PentaRay mapping catheter is inserted in the recipient left ventricle via a retrograde approach. Local electrograms recorded by the PentaRay catheter positioned at the isthmus level (left quadrant) are low-voltage fragmented middiastolic potentials (particularly seen in PENTA 17-18 and 19-20). Other dipoles show signal covering the majority of VT cycle length. The green dot represents the position of the successful radiofrequency (RF) application targeting mid-diastolic potentials during VT. **B:** The clinical VT was terminated after 3 RF applications at 35 W. Solid arrow shows VT termination on surface electrocardiogram (ECG) followed by sinus rhythm of the donor heart. Dashed arrow shows asystole of the recipient heart after VT termination, recorded by a recipient right ventricular catheter. **C:** Twelve-lead ECG recorded the day after the procedure shows sinus rhythm of the donor heart at 70 beats per minute and a slow, wide QRS-complex activity corresponding to escape ventricular rhythm of the recipient heart owing to infrahisian third-degree atrioventricular block.

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Appendix Supplementary data

Supplementary data associated with this article can be found in the online version at http://dx.doi.org/10.1016/j.hrcr.2016. 09.011.

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