Catheter Ablation of Supraventricular Tachycardia in the Transplanted Heart: A Case Series and Literature Review

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MAGNANO, A.R., ET AL.: Catheter Ablation of Supraventricular Tachycardia in the Transplanted Heart: A Case Series and Literature Review. *Clinically important supraventricular arrhythmias are occasionally encountered in patients following cardiac transplantation and the use of catheter ablation as a treatment has been reported. The following three cases are described: (1) atrial flutter, including electroanatomic mapping of the donor and recipient components of the right atrium, (2) a mid-septal accessory pathway, and (3) atrioventricular nodal reentrant tachycardia (AVNRT). A Medline database search was performed and articles addressing catheter ablation following cardiac transplantation were reviewed. The efficacy of RFA for treating various arrhythmia mechanisms was evaluated based on a summary of published case reports. (PACE 2003; 26:1878–1886)*

catheter ablation, cardiac transplantation, supraventricular tachycardia

Introduction

Cardiac arrhythmias are common in the transplanted heart. While isolated atrial or ventricular ectopy accounts for most of these arrhythmias, sustained arrhythmias are also encountered.[1,2] There have been several case reports of clinically important supraventricular tachycardia (SVT) treated with radiofrequency ablation (RFA).[3–6] Three cardiac transplant patients, each with a different arrhythmia mechanism, were treated successfully with catheter ablation. These three case reports are accompanied by a review of the literature addressing SVTs following cardiac transplantation and the use of RFA in their treatment.

Methods

During the 12-month period between July 2001 and June 2002, three catheter ablations were performed at the electrophysiological laboratory of the New York Presbyterian Hospital-Columbia University in patients following orthotopic cardiac transplantation. These case histories and the findings from the cardiac electrophysiological studies were reviewed.

The literature review was performed by a Medline database search of years 1966–2002, crossing all permutations of "ablation," "radiofrequency ablation," or "cardiac arrhythmias" with "cardiac" or "heart" and "transplant" or "transplantation." Based on the titles and abstracts of these publications, 47 were selected for review. References cited by these articles were also reviewed and included when relevant.

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Results

Case Summaries

Case 1

A 66-year-old man presented to the electrophysiological laboratory with a 4-month history of atrial flutter 8 years following orthotopic heart transplantation for ischemic cardiomyopathy. The atrial flutter began during an episode of grade 3A rejection and became symptomatic and persistent despite effective treatment of rejection and amiodarone therapy (200 mg daily). Other significant conditions included end-stage renal disease treated with a cadaveric renal transplant and endocarditis resulting in severe mitral regurgitation. Of note, the cardiac transplantation procedure was performed with a biatrial (standard) anastomosis.

Right atrial activation was mapped during the arrhythmia using the CARTO electroanatomic mapping system (version 4.2, Biosense Webster, Diamond Bar, CA, USA). The donor atrium heart rhythm was typical counter-clockwise atrial flutter (Figs. 1 and 2). The flutter had an atrial cycle length of 290 ms with 2 : 1 atrioventricular (AV) conduction. A large remnant of the recipient atrium (Fig. 2B) was identified and found to be electrically dissociated from the donor heart. Sinus bradycardia (Fig. 1, ABLp and ABLd) was present throughout this segment of native atrium. Application of radiofrequency (RF) energy between the tricuspid valve annulus and the donor-recipient atrial suture line resulted in termination of the tachycardia (Fig. 2C). Following ablation, halo catheter activation and electroanatomic mapping demonstrated bidirectional block. There has been no recurrence of atrial flutter during the 4-month follow-up.

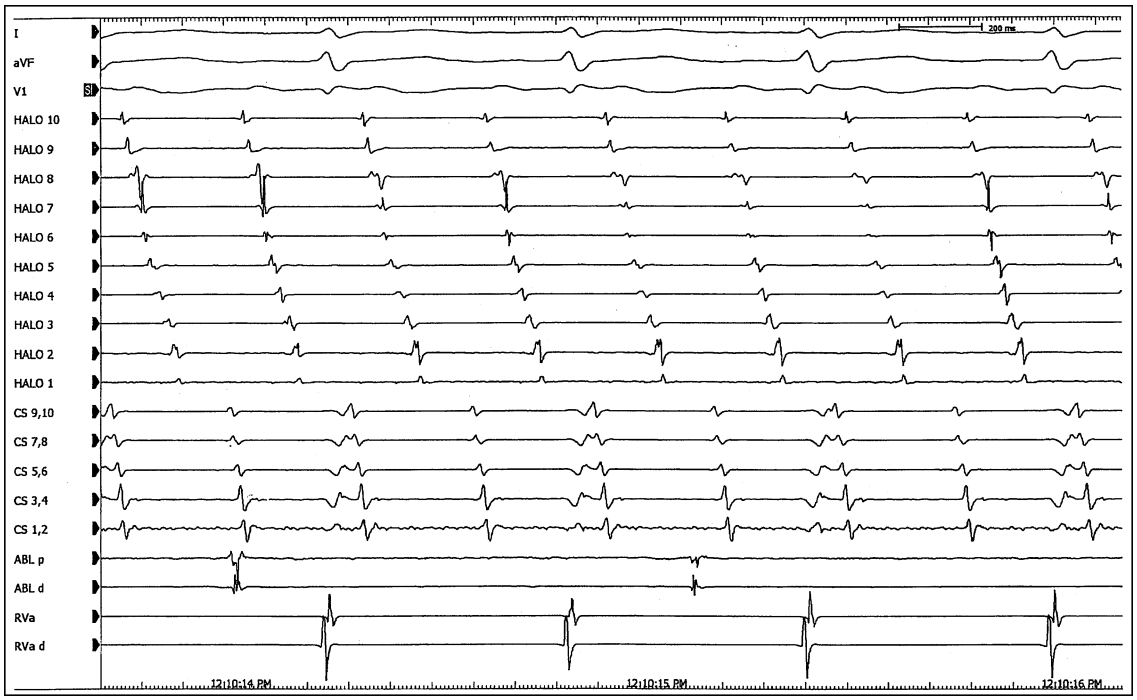

Figure 1. *Intracardiac electrogram recordings for case 1. The donor and recipient atria were dissociated, with atrial flutter in the donor atrium ("HALO" and "CS" catheters), but sinus bradycardia in the recipient atrium ("ABL"). The HALO catheter was positioned with the distal tip (HALO 1) at the posterolateral aspect of the tricuspid valve, extending proximally in a clockwise manner around the tricuspid valve annulus to HALO 10 (proximal pole). CS1,2 represents the distal aspect of the coronary sinus (CS) catheter while CS 9,10 represents the proximal CS catheter. The mapping catheter (ABL) was being used to record potentials from the recipient atrial remnant. Radiofrequency ablation was not carried out in the recipient tissue. I, aVF, and V_1 represent surface electrocardiogram leads while RVa represents the right ventricular apex.*

Case 2

A 59-year-old man underwent orthotopic heart transplantation for an end-stage ischemic cardiomyopathy. The donor was a 52-year-old man with intracranial hemorrhage and no history of arrhythmias. An electrocardiogram (ECG) done immediately prior to cardiac explantation revealed normal sinus rhythm at 86 beats/min with an early precordial R wave transition but no delta wave. An ECG recorded immediately following the surgical procedure showed sinus tachycardia at 106 beats/min with new evidence of ventricular preexcitation. On postoperative day 3, the patient developed prolonged episodes of preexcited tachycardia at rates between 150 and 160 beats/min. P wave morphology and analysis of irregularities in rate suggested a diagnosis of atrial tachycardia with ventricular preexcitation. The patient also had further episodes of SVT with rates up to 250 beats/min. Procainamide was administered, which resulted in loss of preexcitation and suppression of SVT. The patient was referred for a cardiac electrophysiological study.

The cardiac electrophysiological study was performed on postoperative day 10 following withdrawal of procainamide. The ECG in sinus rhythm showed ventricular preexcitation (Fig. 3A). Atrial tachycardia at 115–120 beats/min with ventricular preexcitation was induced during catheter placement that terminated spontaneously but could not be reproduced. A right posterolateral accessory pathway (AP) was anticipated from the ECG pattern, but detailed intracardiac mapping revealed a right mid-septal AP with anterograde and retrograde conduction. Because of rapid conduction over the AP during clinically documented atrial tachyarrhythmias, RFA of the AP was performed, eliminating preexcitation (Fig. 3B). Because the atrial tachycardia was not reproducible, catheter mapping and ablation of atrial tachycardia were not performed. During a 10-month follow-up, there has been no recurrence of atrial tachycardia, other SVTs, or ventricular preexcitation.

Case 3

An orthotopic heart transplantation was performed on a 71-year-old man with an ischemic

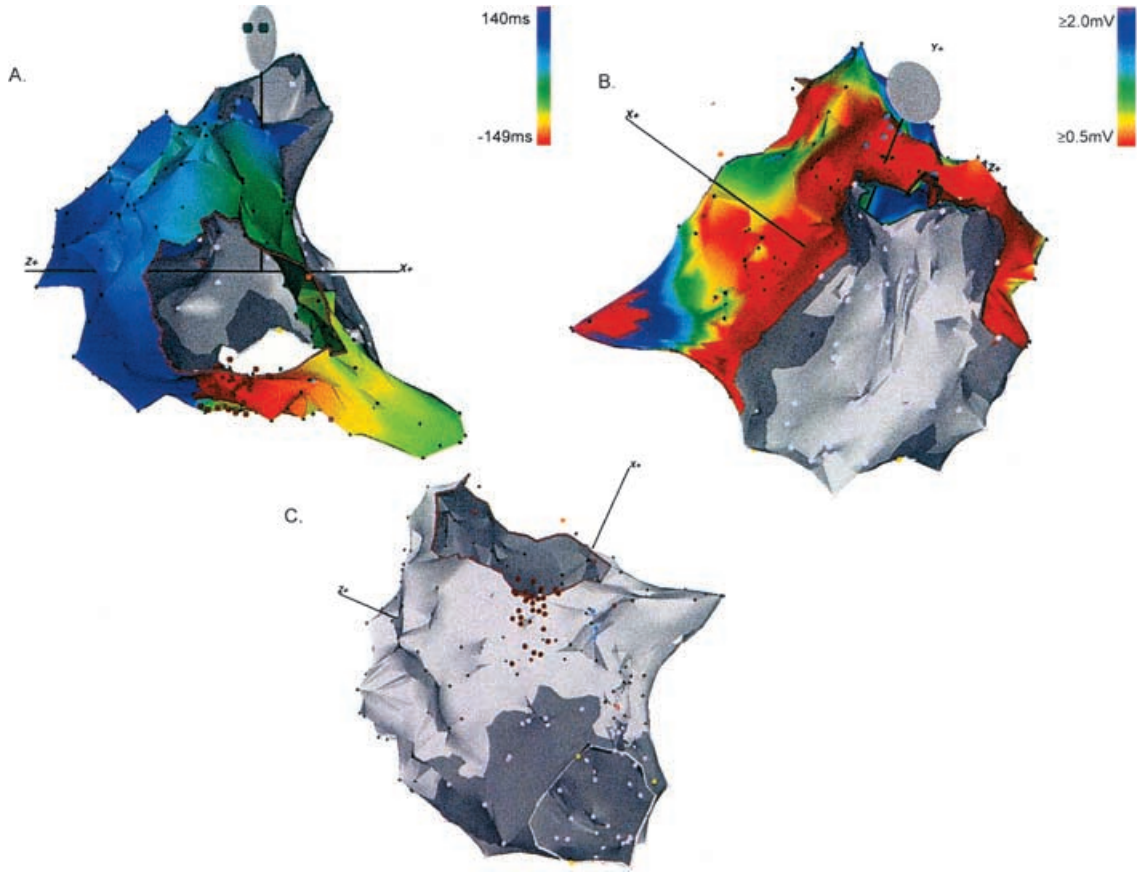

Figure 2. *Electroanatomic map of the right atrium of case 1. (A) Right atrial electroanatomic activation map during typical counter-clockwise atrial flutter. Regions of light gray represent sinus rhythm in the recipient atrial remnant. The dark gray perimeter delineates the border zone between the donor and recipient atria; there was absence of electrical potentials (scar) or simultaneous sinus rhythm and atrial flutter recorded when the mapping catheter was placed along this boundary. The color represents the atrial flutter circuit; with earliest activation (relative to coronary sinus reference) in red and latest activation in purple (see color scale). (B) A voltage map of the donor atrium. Red represents the lowest voltage while purple represents the highest voltage (color scale at upper right of caption). Note the low voltage signals along the border zone between the donor and recipient atria. (C) The isthmus between the tricuspid annulus (top) and inferior vena cava (bottom) is shown with the recipient atrium in dark gray and the donor atrium in light gray. Red markers indicate sites of radiofrequency ablation applications. Because the suture line between the donor and recipient atrium crosses the isthmus, there is no need to ablate tissue within the recipient's native atrium. The ablation line needs only to extend from the tricuspid valve annulus to the anastomosis line.*

cardiomyopathy. The donor had no known history of cardiac arrhythmias. The recipient's comorbidities included chronic obstructive pulmonary disease and mild renal insufficiency. An ECG recorded immediately postoperatively showed normal sinus rhythm. Several days following transplantation he developed palpitations and an ECG revealed a narrow complex tachycardia. He was treated with β-blockers and calcium channel blockers. Two months posttransplantation, he presented to the emergency department with incessant SVT in the context of a fasciitis of the left lower extremity. Neither β-adrenergic blockers nor calcium channel blockers were effective, but intravenous procainamide terminated the arrhythmia and restored sinus rhythm. On eventual withdrawal of procainamide, the patient had recurrent symptomatic SVT and asymptomatic episodes of nonsustained ventricular tachycardia.

The patient underwent a cardiac electrophysiological study 6 months following transplantation. Atrial pacing with single extrastimuli repeatedly induced a narrow complex tachycardia at a cycle length of 460 ms (Fig. 4).The retrograde atrial activation sequence during tachycardia was concentric. The high right atrium, coronary sinus, and right ventricular apex electrograms could be dissociated from the tachycardia circuit without

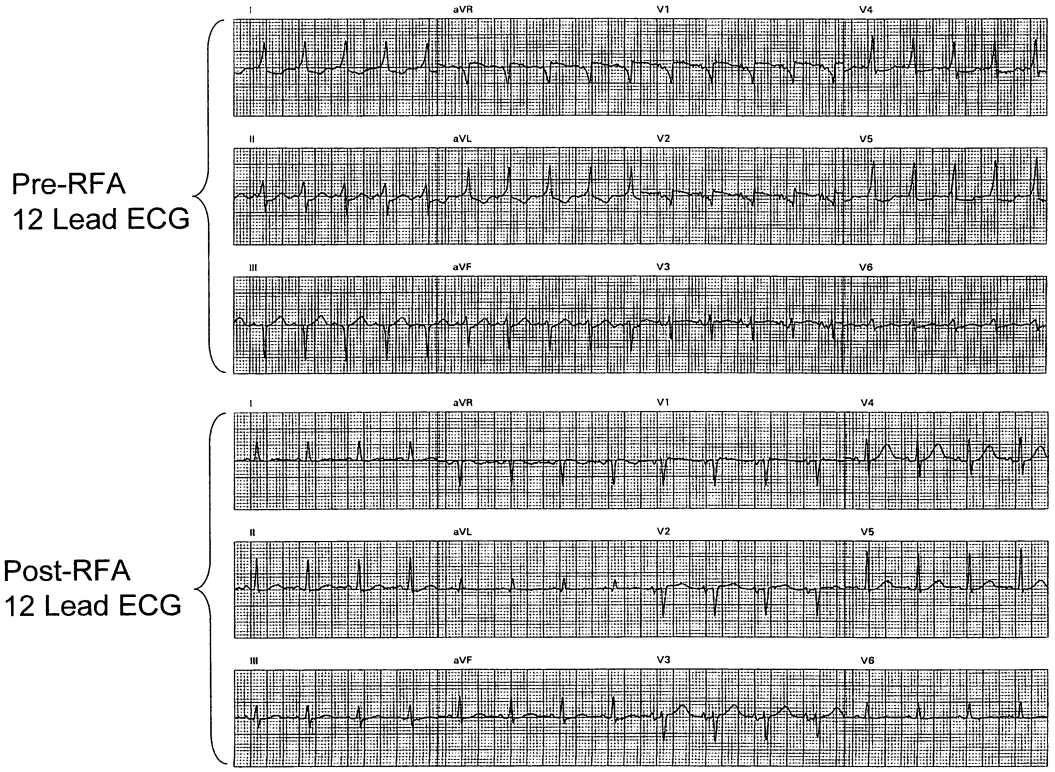

Figure 3. *Twelve-lead electrocardiograms from case 2 recorded before (A) and after (B) radiofrequency ablation of the mid-septal accessory pathway. Note the disappearance of delta waves.*

resetting the tachycardia. A diagnosis of atrioventricular nodal reentry tachycardia (AVNRT) was made. Application of RF energy at the posteroseptal tricuspid valve annulus resulted in slow pathway ablation, after which SVT was no longer inducible. There were no inducible ventricular arrhythmias with delivery of up to triple extrastimuli at the right ventricular apex. The patient remained free of recurrent SVT during the 12-month follow-up.

Literature Search

Including the three patients presented herein, 31 patients were found with prior cardiac transplantation who were studied in the electrophysiological laboratory for clinically significant supraventricular arrhythmias. Eleven patients were diagnosed with APs, 5 with atrial flutter, 12 with atrial tachycardia, and 3 with AVNRT. A total of 28 catheter ablation procedures (27 RF ablations and 1 direct current ablation [DCA]) were performed on these 31 patients (Tables I and II). In addition to these 28 catheter ablation procedures, 2 surgical "bench" ablations were performed for APs in the time interval between cardiac explantation and implantation, 1 of which was successful. Twenty-six of 27 catheter ablation procedures were successful, for an overall procedural success rate of 96.4%. During a mean follow-up of 9.3 months, only two patients with a successful ablation procedure had an arrhythmia recurrence. Although outside the scope of the present report, there has been one reported catheter ablation of ventricular tachycardia after cardiac transplantation, an idiopathic fascicular left ventricular tachycardia that was successfully treated with RF ablation.[7]

Discussion

Prevalence of Arrhythmias Following Cardiac Transplantation

Atrial arrhythmias are common during the index hospitalization for a cardiac transplantation procedure.[1,2] In a series of 33 patients, 15% were found to have atrial flutter, 12% atrial tachycardia, and 6% atrial fibrillation during hospitalization for the cardiac transplantation procedure. Prolonged donor heart ischemic time, elevated pulmonary artery pressure, and decreased ejection fraction were associated with occurrence of arrhythmias.[1] A second study of 25 patients reported a similar prevalence of 44% experiencing

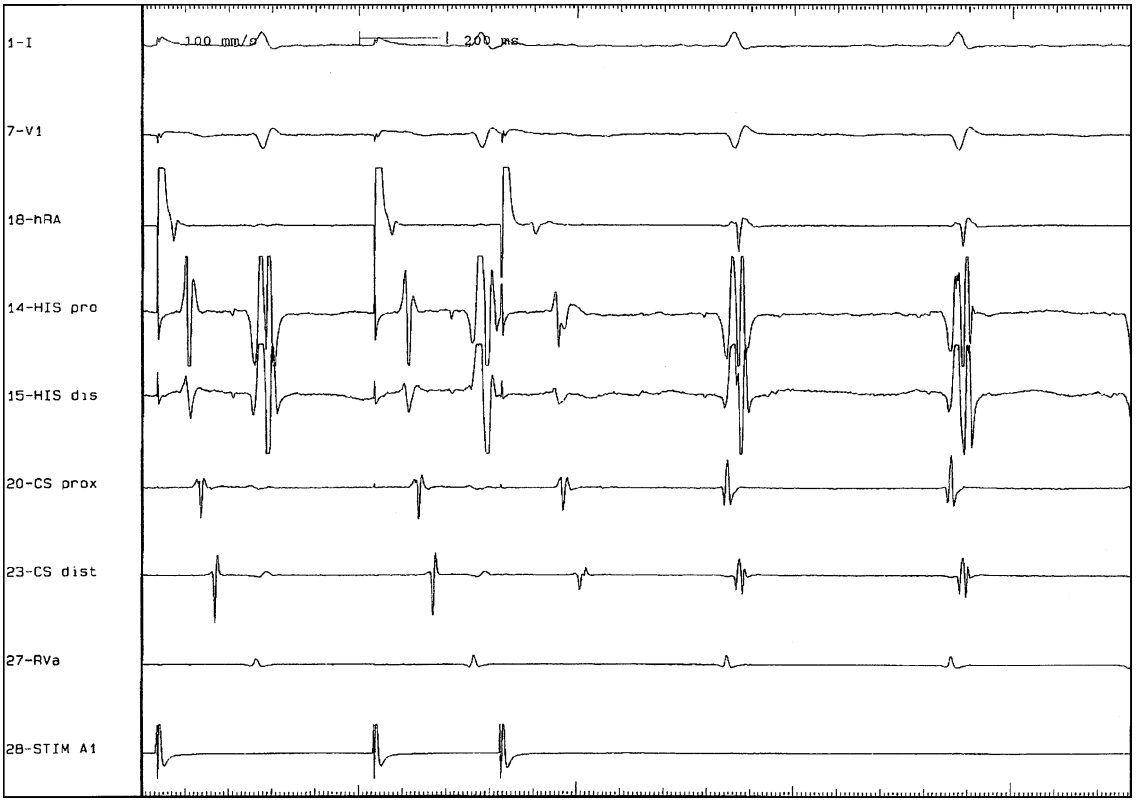

Figure 4. *Intracardiac electrogram recordings from case 3 during induction of atrioventricular nodal reentry tachycardia (AVNRT). Most inductions were achieved with a single atrial extrastimulus resulting in block in the fast pathway and conduction via the slow pathway. I and V1 = surface electrocardiographic leads; HRA = high right atrium; HIS = the proximal and distal bundle of His; CS = coronary sinus; RVA = right ventricular apex.*

Table I.

Summary of Catheter Ablation Procedures for SVT Following Cardiac Transplantation

Arrhythmia	Procedures	Donor Arrhythmia	Success	Recurrence	Comments
Accessory pathways: RFA	8 (in 7 patients)	3	8	1	6 left lateral (1 concealed), 1 mid-septal, 8 RFA, 1 DCA
Atrial flutter	4	0	4	0	TA-IVC or TA-Suture line isthmus ablated
Donor heart atrial tachycardia*	1	0	1	1	Ablation within reentrant circuit
Native atrium AT	11	0	11	0	RFA: 8 suture line, 2 native focus, 1 both
AVNRT	3	0	3	0	RFA slow pathway of AV node
His bundle*	1	0	1	0	Following failed RFA of AT (included above)
Total catheter ablations	28	3 (10.7%)	27 (96.4%)	2 (7.1%)	

*These cases represent the same patient, in whom a His-bundle ablation was performed following recurrence of previously ablated AT.
AT = atrial tachycadia; AV = atrioventricular; AVNRT = AV nodal reentrant tachycardia; DCA = direct current ablation; RFA = radiofrequency ablation; SVT = supraventricular tachycardia; TA-IVC = tricuspid annulus-inferior vena cava isthmus; TA = tricuspid annulus.

Table II.

SVT Cases Reported in the Literature Following Orthotopic Cardiac Transplantation

Lead Author	Year	Donor ECG	Donor Arrhythmia	Recipient Arrhythmia	Treatment	Rx Time	Success	Recurrence
Accessory pathways								
Goy[26]	1989	Normal	None known	Left lateral preexcitation/SVT	Flecainide	Appr. 1 m	Yes	None (2 m)
Thompson[21]	1989	Left lateral PE	None known	Left lateral preexcitation/no SVT	Surgical ablation	6 m	Yes	None (6 m)
Ollitraut[27]	1990	Normal	None known	Left lateral preexcitation/no SVT	None	3 m	NA	None (24 m)
Gallay[28]	1992	Normal	None known	Left lateral preexcitation/SVT	DCA	2 y 5 m	Yes	None (4 m)
Neuzner[3]	1994	normal	None known	Left lateral concealed pathway/AVRT	RFA	3 m	Yes	NR
Rothman[29]	1994	Left lateral PE	None known	Left lateral preexcitation/no SVT	RFA	7 d	Yes	None (12 m)
Blanche[22]	1995	Left lateral PE	SVT	Left posterolateral preexcitation/SVT	RFA - 2 attempts	15 d/ 6 m	No/Yes	None (6 m)
Guinvarc'h[30]	1995	Normal	Yes, unspec.	Left lateral preexcitation/no SVT	RFA	22 d	Yes	None (6 m)
Alexis[31]	1998	Normal	None known	Mid-septal/no SVT	None	N/A	N/A	None (14 m)
Sharma[32]	1999	Normal	None known	SVT via concealed left antero-lateral BPT	RFA	9 y	Yes	None (12 m)
Magnano	2003	Normal	None known	Posteroseptal preexcitation/ transient AT	RFA	10 d	Yes	None (9 m)
Atrial flutter								
Pitt[33]	1997	Not reported	NR	"Macroreentrant atrial flutter"	RFA near RA SL	NR	Yes	None (10 w)
Li[6]	1999	Normal	None known	Typical clockwise AFL	RFA TA-IVC isthmus	8 m	Yes	NR
Pinski[20]	1999	Not reported	NR	Typical counter-clockwise AFL	RFA TA-SL Isthmus	13 m	Yes	None (6 m)
Boveda[34]	2001	Not reported	NR	Typical counter-clockwise AFL	RFA TA-IVC isthmus	2 m	Yes	None (24 m)
Magnano	2003	Not reported	None known	Typical counter-clockwise AFL	RFA TA-SL isthmus	8 y	Yes	None (3 m)
His-bundle ablation								
Hoffman[9]	1999	Not reported	NR	Donor heart "macroeentrant AT"	RFA His bundle (rate control)	Appr. 3 m	Yes	None (6 m)
Atrial tachycardia								
Ott[4]	1995	Not reported	NR	Focal AT: SN region	RFA focal origin	2 y 6 m	Yes	None (4 m)
Rothman[35]	1995	ST/T wave abnormality	None known	AT with 1 : 1 - 2 : 1 R-D conduction	RFA RA SL	7 y 6 m	Yes	None (17 m)
Li[39]	1996	Not reported	NR	AT with 1 : 1 - 3 : 1 R-D conduction	RFA RA SL	10 y	Yes	None (3 m)
Lai[36]	1998	Not reported	NR	AT with 2 : 1 R-D conduction	RFA RA SL	8 y	Yes	None (6 m)
Lefroy[37]	1998	Not reported	NR	AT with 2 : 1 - 3 : 1 R-D conduction	RFA LA SL	7 y	Yes	None (8 m)
Saodi[38]	1998	Not reported	NR	AT	RFA RA SL	5 y	Yes	None (appr. 14 m)
Saodi[38]	1998	Not reported	NR	AT	RFA RA SL	5 y	Yes	None (appr. 14 m)
Saodi[38]	1998	Not reported	NR	AT	RFA native RA focus	5 y	Yes	None (appr. 14 m)
Gasparini[10]	1999	Not reported	NR	AT with 1 : 1, 2 : 1, & decr. R-D conduction	RFA RA SL	5 y	Yes	None (18 m)
Hoffman[9]	1999	Not reported	NR	Donor heart "macroreentrant AT"	RFA within reentrant circuit	3 m	Yes	Yes (3 m)
Birnie[40]	2000	Not reported	NR	AT w. 2 : 1 R-D conduction, prior AFL	RFA RA SL	8 y	Yes	None (10 m)
Strohmer[41]	2000	Not reported	NR	AT w. 1 : 1, 2 : 1 R-D conduction	RFA RA SL & native RA focus	9 m	Yes	None (5 m)
AV nodal reentrant tachycardia								
Padder[42]	1999	Not reported	NR	AVNRT (typical slow/fast)	RFA slow pathway	Appr. 2 w	Yes	None (6 m)
Zhu[5]	1998	Not reported	None known	AVNRT (typical slow/fast)	RFA slow pathway	10 y	Yes	None (9 m)
Magnano	2003	Normal	None known	AVNRT (typical slow/fast)	RFA slow pathway	6 m	Yes	None (12 m)

Abbreviations: AFL = atrial flutter; AT = atrial tachycardia; Appr. = approximate; AVNRT = atrioventricular nodal reentrant tachycardia; D = donor; decr = decremental; m = months; NR = not reported; PE = Preexcitation; SVT = supraventricular tachycardia; R = recipient; w = weeks; y = years; AV = atrioventricular; DCA = direct current ablation; ECG = electrocardiogram; RA = right atrial; RFA = radio frequency ablation; SVT = supraventricular tachycardia; TA-IVC = tricuspid annulus-interior vena cava isthmus; TA-SL = tricuspid annulus-suture line.

sustained atrial arrhythmias, which were predominantly atrial tachycardia and atrial flutter.[2]

Sustained atrial arrhythmias are also relatively common during the months following cardiac transplantation. Pavri et al.[8] evaluated all available telemetry and ECG tracings in a group of 88 consecutive heart transplant patients. Atrial flutter was the most common arrhythmia, observed in 21 patients with a mean onset time of 26 ± 10 days following transplantation. Ectopic atrial tachycardia was observed in 3 patients, atrial fibrillation in 23 patients, and SVT in 18 patients. Only atrial fibrillation was associated with adverse outcome, with a threefold subsequent risk of death.

Most arrhythmias following cardiac transplantation are nonsustained or self-limited and ultimately benign.[1,2] Some are more persistent, causing clinically important symptoms and, in some cases, even tachycardia mediated cardiomyopathy.[4,9–10] Atrial flutter has been found to be a relatively specific sign of humoral or cellular rejection, however its sensitivity is limited.[11,12]

Pathophysiology of Arrhythmias in the Transplanted Heart

The transplanted heart is exposed to a number of physiological and anatomic changes that may promote cardiac arrhythmias. Following explantation, the organ is subject to thermal and mechanical injury during the cold ischemic period.[13] During implantation, the suture lines may constitute electrical lines of conduction block that may facilitate incisional atrial arrhythmias. The presence of a remnant of native right atrium introduces the equivalent of a large region of scar in the donor atrium (Fig. 2B). The newer surgical technique of bicaval anastomosis minimizes the arrhythmogenic impact of this suturing.[14] Postoperatively, cellular architectural alteration may occur during episodes of acute rejection, later resulting in myocardial scarring and fibrosis that may provide a substrate for cardiac arrhythmias.[11,14–16] Cardiac denervation is another important consequence of heart transplantation with a number of important effects on cardiac electrophysiological properties, including enhanced sinus node automaticity, altered AV nodal conduction properties, and an altered heart rate response to exercise and recovery.[17,18]

Catheter Ablation of Arrhythmias Following Cardiac Transplantation

Atrial Flutter

Atrial flutter is a common arrhythmia following heart transplantation and is often an important sign of graft dysfunction. The correlation between atrial flutter and acute rejection strengthens with time following transplantation until, after 6 months, the vast majority of atrial flutter episodes are associated with severe cellular or humoral rejection or significant transplant related coronary disease.[8,12] These conditions should be considered and excluded when cardiac transplant patients present with atrial flutter.

The flutter circuits in transplant patients, including those with biatrial anastomoses, closely resemble those of other patients with atrial flutter (i.e., macroreentry around the tricuspid valve annulus).[19] Electroanatomic mapping in case 1 confirmed this mechanism. While the circuit is nearly identical to that in nontransplanted hearts, the atrial anastomosis line interrupts the corridor between the lateral tricuspid annulus and the crista terminalis; as a result, activation of the lateral wall is confined to a narrower region of longitudinal fibers close to the tricuspid annulus.[19] Brandt et al.[14] reported that 40% of all patients experienced atrial flutter and/or fibrillation following biatrial anastomoses, compared with only 4% following bicaval anastomoses.

Although atrial flutter is common following cardiac transplantation, only five cases, all with biatrial anastomoses, treated with RF ablation have been reported.[1,6,8,12,20] These procedures were successful, with no complications and no recurrence during follow-up. Case 1 is the fifth case, but the first that incorporated complete electroanatomic mapping of the arrhythmia (Fig. 2). This three-dimensional mapping technique was useful in defining the anatomic relationship of the donor and recipient atrium and confirmed that the atrial flutter circuit is similar to that in nontransplanted hearts. Of note, the presence of a biatrial anastomosis implies that a suture line will interrupt the isthmus between the tricuspid valve annulus and the inferior vena cava. It may be sufficient to continue ablation only between the tricuspid annulus and the suture line, if it can be clearly defined.[20] Bicaval anastomoses are most commonly used at present, thus the incidence of posttransplantation atrial flutter can be expected to decrease.[14]

APs

Including the current report, a total of 11 cardiac transplantation patients were found who received a heart with an AP (Table II); 9 were in a left lateral location (1 concealed) while 2 were in the mid or posterior septum. In three cases there was knowledge of preexcitation on the donor's ECG. However, in the remaining eight patients (including case 2), there was no history of ventricular preexcitation or clinical arrhythmias in the donor. This may reflect the intermittent bypass tract

conduction, the availability of few preoperative ECGs, the high prevalence of left lateral pathways in the sample (which may be associated with less prominent delta waves), and the possible exclusion of donor hearts with preexcitation for transplantation.

Nine patients underwent 11 ablation procedures (2 surgical ablations, 1 catheter DCA, and 8 RFAs), 1 was treated with flecainide and 2 were followed conservatively. Of the cases reported, there were no recurrent arrhythmias following treatment or the decision to follow conservatively. Of those treated with ablation, two were performed surgically as a "bench" procedure after explantation of the donor heart. This approach was successful in one of the two cases,[21] while the other required two postoperative RFA attempts for successful ablation.[22] Surgical bench ablation can be efficacious, but is associated with an increased graft ischemic time.[21,22] A total of eight catheter ablations were attempted in seven patients with APs, of which seven were successful. At least five of these procedures were performed in the first month following ablation, including case 2, who underwent RFA on postoperative day 10. There were no complications reported and the patients remained free from recurrent arrhythmias or preexcitation for a mean follow-up of 9.5 months. The feasibility of RFA of APs following cardiac transplantation coupled with the shortage of donor organs emphasizes the appropriateness of transplanting organs with known APs.

AVNRT

Including case 3, three catheter ablations of AVNRT following cardiac transplantation have been reported (Table II). Prior to treatment, the patient (case 3) experienced incessant AVNRT, although there was no known history of arrhythmias or symptoms of arrhythmias in the donor. With regards to AV nodal physiology following cardiac transplantation, conduction properties may be altered by denervation. For instance, the effective refractory period of the AV node normally increases at more rapid atrial drive rates, while it has been shown to decrease with faster rates following transplantation.[23–25] Only three relatively recent case reports have documented catheter ablation of this arrhythmia in a transplanted heart. This relatively small number of patients may reflect that AVNRT is a less common cause of SVT following orthotopic heart transplant. An alternative possibility is that publication bias has limited the number of case reports of AVNRT given its high prevalence in the nontransplant population. Of the reported cases with AVNRT treated with ablation, all were successful and without recurrence over a mean 9-month follow-up.

Atrial Tachycardia

Twelve cases of atrial tachycardia were found in the literature review (Table II), all of which were treated with RFA. All 12 patients had biatrial anastomoses. Eleven of these 12 cases were focal atrial tachycardias originating in the remnant of the recipient atrium with documentation of recipient to donor conduction across the atrial suture line. While this suture line is typically a line of conduction block, conduction across this line during atrial tachycardia has been documented, at times with variable conduction block or decremental conduction.[10] Ten of the 11 cases were right atrial in origin, while 1 was left atrial but also from a remnant of native atrial tissue. All cases were treated with RFA at 5–9 years following transplantation. Ablation was successful in all 11 cases with no recurrences during a mean follow-up of 9.7 months. The other case of atrial tachycardia was described as a macro-reentrant atrial tachycardia confined to the donor atrium and was associated with cardiomyopathy. Because of recurrence of atrial tachycardia 3 months following initial ablation, the patient underwent "ablate-and-pace" therapy with normalization of ventricular function.[9]

In total, three reported patients with atrial tachycardia following transplantation have shown signs of a tachycardia induced cardiomyopathy.[4,9,10] Ventricular function improved following catheter ablation in all three cases, underscoring the potential for tachycardia induced cardiomyopathy to complicate the course of the postcardiac transplantation patient and the ability to remedy this disorder with RFA.

Conclusion

Arrhythmias are common following cardiac transplantation in the early postoperative period and during later follow-up. Catheter ablation of supraventricular arrhythmias has been safely performed as early as 1 week and as late as 9 years following transplantation. Sufficient experience now exists to suggest that donor grafts demonstrating preexcitation are acceptable for transplantation, particularly in light of the limited donor pool and the feasibility of RFA. The biatrial anastomosis technique appears be a major factor in promoting incisional atrial tachycardia and atrial flutter. While RFA is highly efficacious in the treatment of these arrhythmias, the trend toward bicaval anastomoses should have a favorable impact on the incidence of posttransplantation supraventricular arrhythmias. Finally, in at least three cases, signs of tachycardia-mediated cardiomyopathy were reversed following ablation.

Since catheter ablation of arrhythmias following cardiac transplantation is not a common

occurrence, the entire literature is based on case reports. It is, therefore, important to recognize that publication bias may have resulted in the inclusion of only successful procedures. With that proviso, the authors conclude that catheter ablation appears to be a safe and effective therapeutic option applicable in a wide range of arrhythmias in the posttransplant population.

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