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Radiofrequency Ablation of Drug Refractory Ventricular Tachycardia Related to Cocaine Use: A Feasibility, Safety, and Efficacy Study

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Abstract

Background—Cocaine use is a known but rare cause of cardiac arrhythmias. Ventricular arrhythmias related to cocaine may not respond to antiarrhythmic drugs and may need treatment with radiofrequency ablation.

Objectives—We describe the clinical and electrophysiological characteristics of cocaine-related ventricular tachycardia (VT) from a multicenter registry.

Methods—Subjects presenting with VT related to cocaine use and being considered for radiofrequency ablation have been included in the study. Patients who were refractory to maximal medical therapy underwent radiofrequency ablation of the VT. Clinical, procedural variables, efficacy, and safety outcomes were assessed.

Results—A total of 14 subjects met study criteria (age 44 ± 13, range 18-to 68-year-old with 79% male, 71% Caucasian). MRI showed evidence of scar only in 43% of patients (6/14). The mechanism of VT was focal in 50% (n = 7) and scar related reentry in 50% (n = 7) based on 3D mapping. The mean VT cycle length was 429 ± 96 milliseconds. The site of origin was epicardial

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in 16% (3/18) of VTs. Most clinical VTs were hemodynamically stable (75%). Mean ejection fraction at the time of admission was 44 ± 14%. Duration of procedure was 289 ± 50 minutes. One subject developed pericardial tamponade requiring drainage. At 18 ± 11 months follow-up, freedom from arrhythmia was seen in 86% (1 case lost to follow-up and 2 died).

Conclusion—Radiofrequency ablation is not only feasible but also safe and effective in patients who have drug refractory VT related to chronic cocaine use.

Keywords

cocaine; focal mechanism; myocardial infarction; reentry; ventricular tachycardia

Introduction

Cocaine is a commonly abused drug in the United States. According to the 2008 National Survey on Drug Use and Health, an estimated 1.7 million Americans age 12 and higher had used cocaine in the preceding month.[1] Cocaine intake is associated with hypertensive crisis, acute myocardial ischemia/infarct, ventricular arrhythmias, and congestive heart failure. Some of these effects could be related to its ability to inhibit catecholamine reuptake at the presynaptic nerve endings causing exaggerated sympathetic surge or sodium channel inhibition. Cocaine is known to alter myocardial contractility, decreased rate of depolarization, diminished action potential amplitude and duration, and conduction. In most subjects with acute cocaine exposure, ventricular arrhythmias are self-limited and resolve with aggressive medical therapy due to its relatively short half-life (<10 hours).[2-4] However, cocaine can be detected in body fluids for more than a week and may sometimes have prolonged residual effects. Chronic cocaine abuse may lead to myocardial infarction with remodeling and resultant scar that forms the substrate for ventricular arrhythmias.

Over the last decade, radiofrequency ablation has emerged as a valuable therapy for patients who have drug refractory incessant ventricular tachycardia (VT). However, ventricular arrhythmias refractory to medical therapy in patients who abuse cocaine are not uncommon and, therefore, may benefit from ablation therapy. The role of ablation therapy in this subset of patients with refractory VT has not been reported previously to the best of our knowledge. We report a series of subjects with chronic cocaine use and refractory VT who were not responsive to medical management and underwent radiofrequency ablation.

Methods and Materials

This report is a retrospective, cohort, case series of 14 consecutive subjects known to be chronic cocaine abusers who presented with VT at 4 centers and underwent electro-physiology (EP) study, mapping, and ablation of the VT. Ablation was performed only after medical therapy with multiple antiarrhythmic drugs, including amiodarone, sotalol, β-blockers, calcium channel blockers, mexiletene, and lidocaine failed in controlling the arrhythmia. All of the subjects were in the hospital for at least 72 hours after hospital admission to allow cocaine washout before ablation was contemplated. All subjects underwent cardiac catheterization to rule out coronary artery disease during the index hospitalization before contemplating for EP study. The subjects also underwent cardiac

magnetic resonance imaging with Delayed Hyper Enhancement during this hospitalization before ablation to assess myocardial scar.

VT Ablation

VT ablation was performed using intracardiac echo (ICE), and 3D mapping using either retrograde aortic or transseptal approach.[5,6] All the subjects had recording catheters in the coronary sinus and right ventricular apex. ICE was utilized in all the subjects to guide catheter placement, transseptal access, and for monitoring ablation and the pericardial space. Additionally, epicardial mapping and ablation was done in a few subjects at the discretion of the operator, if deemed appropriate. Mapping and ablation was performed either manually (n = 10) or using remote magnetic guided navigation system (Stereotaxis^{QR} , St. Louis, MO, USA; n = 4). Following LV access, patients were treated with unfractionated heparin (UH), maintaining activated clotting time of 250–350 seconds.

Ventricular mapping was performed with 7F steerable catheters with a 3.5-mm electrode tip open irrigated catheter (Biosensor Webster Navistar, Thermocool or Thermocool RMT, Biosense Webster Inc., Diamond Bar, CA, USA). 3D mapping was performed using CARTO (Biosense Webster Inc.) or NAVX (St. Jude Medical, Minneapolis, MN, USA). In all the patients, a baseline scar map was created to assess the location and extent of the scar. Following mapping, VT was induced and, if stable, activation mapping and entrainment mapping were used to identify the exit site and critical isthmus. If the circuit could not be identified or when the VT was hemodynamically unstable, ablation was performed through the presumptive exit site based on voltage mapping combined with pace mapping. In patients without any scar on the voltage map, a focal source for the arrhythmia was sought from a combination of activation mapping and pace mapping (Fig. 1). Ablation lesions were created with radiofrequency current with a maximum power of 40 W. If nonclinical VTs were induced, an attempt was made to map and ablate those as well. Postablation VT induction was attempted with pacing maneuvers in all cases. In cases where the VT was deemed focal in origin, additional induction using isoproterenol (up to 5 mcg/min) was attempted. The procedure was considered complete when no VT was inducible or if the VT could not be ablated despite multiple attempts.[7,8] Postablation follow-up was obtained from the patients' office visits, hospitalization records and device interrogations for recurrence of VT, antitachycardia pacing and shocks.

Statistical Analysis

JMP 8.0 (SAS Institute Inc., Cary, NC, USA) was used for statistical analysis. Data are presented as mean ± SD. Comparison of continuous data was done using Student's *t*-test and chi-square or Fisher's exact test was used for discrete data. A P value less than 0.05 was considered significant.

Results

Baseline Characteristics

A total of 14 subjects were included in the study. Baseline characteristics of the subjects are listed in Tables 1 and 2. The mean age was 44 ± 13 years and 11 of them were males (79%).

Ejection fraction (EF) at the time of admission was 44 ± 14% (7 subjects had an EF less than 50% at the time of admission). Comorbidities included diabetes (21%), hypertension (71%), dyslipidemia (43%), renal insufficiency (21%), and coronary artery disease (36%). All patients had admitted to using cocaine within 4 weeks of admission to the hospital. Active cocaine metabolites were found in 12 and amphetamines in 3 subjects at the time of admission to the hospital (acute abuse). For those patients who had implantable cardioverter defibrillators (ICDs) and had frequent VT with ATP or shocks, outpatient β-blockers, amiodarone, sotalol, and/or mexiletene were used. During inpatient care a combination of β-blockers, calcium channel blockers, amiodarone, sotalol, lidocaine, and/or mexiletene were used for VT suppression. In all patients who were positive for cocaine metabolites, mean washout period of 6.5 ± 1.6 days was allowed and medical therapy was continued before radiofrequency (RF) ablation was contemplated. In 1 patient, without any cocaine metabolites at admission, VT ablation was performed after 3 days of hospitalization as he was a known cocaine abuser and had admitted to using cocaine within the prior 30 days.

All the subjects had cardiac catheterization and only 1 of them was found to have a significant 80% lesion in the LAD distribution and underwent PCI/stent placement. This patient continued to have further VT episodes after the intervention. MRI scans showed the presence of scar in 43% of patients, the distribution of which is also shown in Table 1. As suspected, the MRI scar location matched with that of the voltage map on the electroanatomic maps done intraprocedurally, except for 1 patient who had a scar on electroanatomic mapping but not on MRI (Fig. 2). The sites of VT ablation for the focal and reentrant VT's have been illustrated in Figure 3A, B.

Procedural and VT Characteristics

Fifteen EP studies (1 patient had repeat EP study 10 days after the first one) with VT ablation were performed. The VT mechanism was focal in 7 (50%) patients without any evidence of myocardial scarring either on MRI or voltage map. The mean induced VT cycle length was 443 ± 97 milliseconds. The VT cycle length was 517 ± 89 milliseconds in subjects without scar and 414 ± 80 milliseconds in subjects with scar related VT (P = 0.001). All but 3 VTs were hemodynamically stable. Tables 3 and 4 shows characteristics of the arrhythmia in the subjects with focal and recurrent VT, respectively. Epicardial mapping was done in 3 cases with successful ablation in two. In 1 patient we successfully eliminated the first VT but the second hemodynamically unstable VT could not be abolished. The mean procedure time was 289 ± 50 minutes; fluoroscopy time was 30 ± 7.5 minutes and RF time was 9.6 ± 5.4 minutes. One subject developed tamponade that was successfully treated with pericardiocentesis.

Outcomes

1. Feasibility—VT ablation was feasible in 100% of patients with this specific myocardial substrate.

2. Efficacy—The acute efficacy was 94% (17/18 of VTs induced) with short-term recurrence of 14% (2/14) and long-term recurrence of 14% patients. One patient had recurrence of VT with a different morphology at 3 months and was treated successfully with

amiodarone. A second patient had recurrence of VT requiring repeat ablation in 10 days. This subject subsequently died 10 days later from central line sepsis. Another patient died 3 months postablation of intractable heart failure with hospice care initiated. Both patients who died had evidence of reduced EF with scar on MRI and 3D voltage maps. At 18 ± 11 months follow-up freedom to survival was 86% and 1 subject was lost to follow-up. Additional 4 different VTs could be induced during the repeat ablation in 1 patient and these were not included in the statistical analysis.

3. Safety—Acute complications were seen in 14% (2/14) patients (pericardial tamponade = 1/Groin hematoma = 1). There was significant improvement in the left ventricular ejection fraction (LVEF) (49 ± 15 vs. 44 ± 14, $P < 0.01$) on follow-up echo in the majority of the cases. Table 5 shows follow-up results on these subjects. Two patients (Patient no. R2 and R4) who had evidence of myocardial scarring on MRI and 3D voltage maps received implantable cardioverter defibrillators before discharge for further protection from sudden cardiac death. Patients (Patient no. F5 and R4) with unstable VT but without any myocardial scar did not receive ICD device following VT ablation. One patient declined ICD therapy and subsequently died of heart failure. Before discharge, all the patients were provided with help to enroll in drug rehabilitation programs.

Differences Between VT Related to Scar and No Scar

There was no significant difference in demographic characteristics in subjects who had myocardial scar (43%) or did not have scar (57%) on MRI. One patient had evidence of endocardial scar on 3D mapping without evidence of the same on the MRI. The presenting VT was faster in subjects with scar tissue. Patients with scar had 11 different VTs, while patients without scar had a single focal VT each. The procedure was also longer in subjects with scar related VT (Table 6). All the patients with a scar (7) on electroanatomic mapping had a reentrant VT, while all those without a scar (7) had focal VT ($P < 0.001$). The focal VTs had spontaneous onset and termination. All the focal VTs were also inducible by means of programmed electrical stimulation. Subjects with no scar tissue in myocardium had better long-term survival.

Follow-Up

All the patients received extensive counseling and help with drug rehabilitation before they were discharged from the hospital. Antiarrhythmic drugs were stopped in all of the patients at discharge. Two patients had recurrence of VT within the first 3 months and were initiated on antiarrhythmic drugs. One of these patients underwent repeat ablation of the VT and was being continued on antiarrhythmic drugs; this patient died from sepsis in the hospital. The other patient was initiated on amiodarone and did not have any recurrence of VT during the subsequent months. This patient was lost to follow-up after 1-year follow-up. All of the patients underwent repeat urine drug screen during the initial 3 months after the ablation and all of them tested negative for cocaine metabolites. There was 1 additional death during the 1-year follow-up.

Discussion

Main Findings

This is the first case series to demonstrate the feasibility, safety, and efficacy of radiofrequency ablation in subjects with cocaine-related VT after they failed antiarrhythmic drug therapy. In our series, chronic cocaine was associated with myocardial scarring in 50% of patients, probably related to small vessel disease and recurrent vasospasm. In patients with scar, VT appeared to be related to macro reentry. In these patients, ablation using traditional scar mapping techniques as well as entrainment was helpful. In 50% of patients, there was no evidence of significant scar and all these patients had VT that was focal in origin.

Pathophysiology of Cocaine Related VT

The pathophysiology of cocaine induced ventricular arrhythmias is complex. Triggered activity, early and delayed after depolarization, has been reported as the cause of arrhythmia related to cocaine use.[9,10] Myocardial scar secondary to myocardial infarct/ischemia, myocarditis, and microscopic changes in myofilament arrangement can create a substrate for reentrant arrhythmias.[11-16] Although the trigger for ventricular arrhythmia may fade after the systemic cocaine levels drop, the underlying structural changes in the myocardial substrate may not be reversible and could increase the long-term propensity to ventricular arrhythmias as myocardial remodeling continues to evolve.

VT Ablation in Patients Without Myocardial Scar

In our small series, half the patients did not have any evidence of scar either by MRI or 3D voltage mapping. Interestingly, VT in patients without scar was relatively slow and tended to demonstrate greater hemodynamic stability. In patients without scar, the VT behaved like a focal VT with a single earliest site of origin without reentrant characteristics. Also in this group voltage maps were normal. The focal VTs in these patients may be due to microvascular changes in the coronary circulation affecting the ventricular myocardium from chronic cocaine use. The micro scarring may be too small for the resolution of the 3D mapping systems and may not, therefore, present itself as a reentrant circuit. It is also possible that some of the focal VTs could have been unmasked by cocaine exposure or it could be a coincidence, like in the patient with right ventricular outflow tract VT. Alternatively, the focal VTs could be a manifestation of endocardial breakthrough of an epicardial VT circuit.[17] Thus, the best technique for ablation was to perform activation mapping in patients with focal VTs. RF ablation in these patients seems to be very effective with excellent acute, short- and long-term outcomes. Three patients required an epicardial approach with 1 of them having focal VT.

Previous studies have shown that chronic use of cocaine is associated with an increase in QRS voltage and ST elevation on the electrocardiogram as compared to the general population. Acute cocaine use increases the prevalence of sinus and supraventricular tachycardia, and prolongs the QT.[18] The ability of cocaine to generate ventricular tachyarrhythmia has been reported in the past. The role of ventricular arrhythmia as a cause of sudden cardiac death in cocaine abusers is well known. Based on animal studies, it is well

known that cocaine can decrease the ventricular fibrillation threshold.[19] Acute cocaine intoxication usually does not induce ventricular arrhythmia in the absence of myocardial ischemia.[20] In humans, life-threatening arrhythmias and sudden death due to cocaine use occur most often in patients with myocardial ischemia or infarction. We have previously shown in a swine study that cocaine in fact increases VT threshold for TASER induced ventricular arrhythmias.[21] However, long-term cocaine use is associated with systemic hypertension and increased left ventricular mass and wall thickness, which is known to be a risk factor for ventricular dysrhythmias and may provide the substrate for development of ventricular arrhythmias.[22] It is possible that cocaine use leads to myocardial disarray that is not visible on MRI and this could lead to microreentry potentially giving rise to focal VTs.[23]

Treatment of Cocaine Related VT

Several therapies have been suggested for the treatment of arrhythmia related to cocaine use. First line therapy includes β-blockers (usually combined with α-blockers to prevent spasm). In addition, the use of sodium bicarbonate[24,25] and lidocaine[26] have been suggested specially in subjects with prolonged QRS possibly secondary to sodium channel blockage by cocaine. The use of lidocaine carries an increased risk of seizures.[27] Seizures can also occur in subjects with acute cocaine toxicity. However, its use in subjects with cocaine induced cardiac ischemia appears to be safe without increased risk of seizure.[23] Potassium channel blockage by cocaine can manifest as QT prolongation and both early and delayed after depolarization, which can cause Torsades.[28-33] Correction of systemic hypokalemia and hypomagnesemia is important in these subjects who are oftentimes electrolyte depleted. Use of amiodarone with cocaine in an animal model did not show survival benefit.[34] Use of calcium channel blockers has shown reduction in arrhythmia[35,36] probably due to the coronary vasodilatory effects in subjects with cocaine induced arrhythmias.[37]

VT associated with acute cocaine toxicity usually terminates with supportive measures.[35] Data on the role of antiarrhythmic drug therapy in acute or chronic cocaine-associated VT are relatively sparse. For VT related to chronic cocaine use, antiarrhythmic drugs seem to be a logical choice of therapy. Data on the role of RF ablation for management of VT in subjects with cocaine induced ventricular arrhythmias are largely nonexistent. Patients who develop ischemic cardiomyopathy with evidence of underlying myocardial scarring, especially in the setting of VT, may benefit from an ICD. Clearly, more data are needed on the role of ICD therapy in this subgroup of patients who present several challenges from clinical, social, and ethical aspects. It is important to note that some patients had evidence of myocardial scar despite well preserved EF and continue to be at risk for recurrent VT (Patient no. 6).

Conclusion

Radiofrequency ablation is not only feasible but also safe and effective in patients who have drug refractory VT related to chronic cocaine use.

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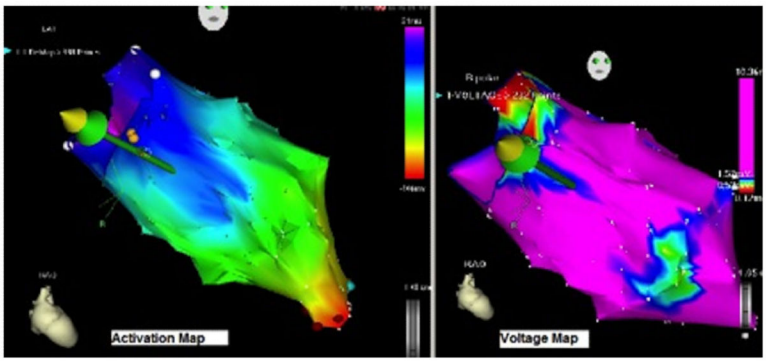

Figure 1.

Shows the activation and voltage maps of a focal VT localized to the apex of the LV. For a high quality, full color version of this figure, please see Journal of Cardiovascular Electrophysiology's website: www.wileyonlinelibrary.com/journal/jce

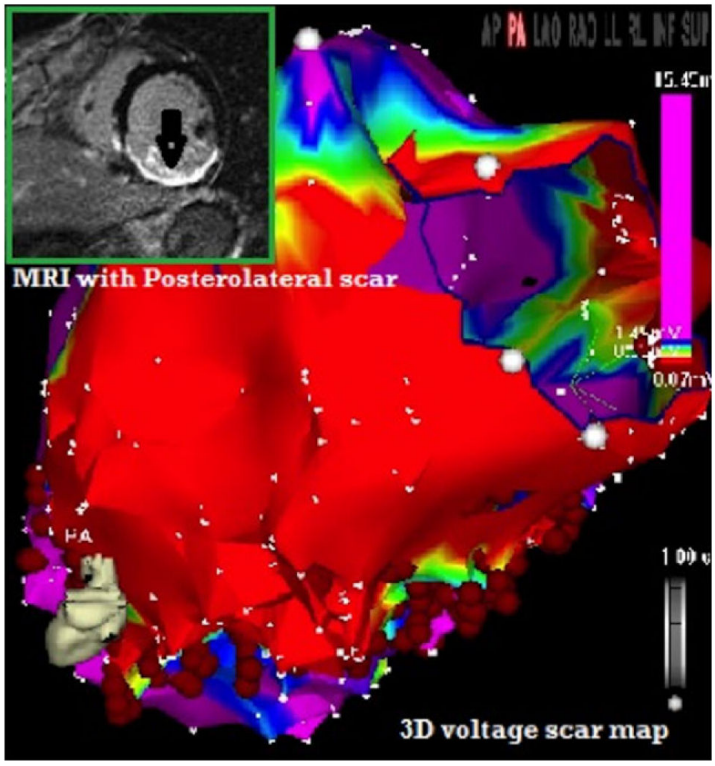

Figure 2.

Shows CARTO 3D map with extensive posterolateral scar corresponding to the posterolateral scar on MRI in the inset indicated by the black arrow. For a high quality, full color version of this figure, please see Journal of Cardiovascular Electrophysiology's website: www.wileyonlinelibrary.com/journal/jce

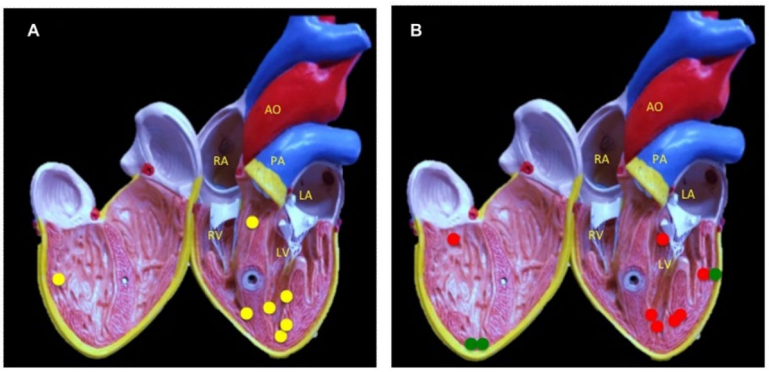

Figure 3.

A: Location of focal tachycardias. Yellow circle—location of the focal tachcardia. AO = *aorta; LA = left atrium; LV = left ventricle; PA = pulmonary artery; RA = right atrium; RV* = right ventricle. B. Location of reentrant ventricular tachycardia. Red circle—endocardial sites of ventricular tachycardia. Green circle—epicardial sites of ventricular tachycardia. AO = *aorta; LA = left atrium; LV = left ventricle; PA = pulmonary artery; RA = right atrium; RV* = right ventricle. For a high quality, full color version of this figure, please see Journal of Cardiovascular Electrophysiology's website: www.wileyonlinelibrary.com/journal/jce

TABLE 1

Baseline Characteristics of Patients with Focal Ventricular Tachycardia

Patient ID	F1	F2	F3	F4	F5	F6	F7
Age	45	49	34	38	47	45	53
Gender	M	M	M	M	M	M	M
Race	B	W	B	B	W	W	W
Diabetes mellitus	N	Y	N	Y	N	N	Y
Hypertension	Y	Y	Y	Y	Y	Y	Y
Hyperlipidemia	Y	Y	N	Y	N	Y	Y
CAD	Y	Y	N	N	N	N	Y
Cr > 1.5	N	N	Y	N	N	N	N
AF	N	N	N	N	N	N	N
Cocaine use within 7 days of admission	Y	Y	Y	Y	Y	Y	Y
β-Blocker	Y	Y	Y	Y	N	Y	Y
ACE inhibitor	Y	N	N	Y	Y	Y	Y
CCB	N	Y	Y	N	Y	Y	Y
Failed AAD (outpatient or inpatient)	Y	Y	Y	Y	Y	Y	Y
Type of AAD at the time of VT ablation	Sotalol	Sotalol	Sotalol + Lidocaine	Amiodarone + Lidocaine	Sotalol	Sotalol	Sotalol
LVEF (%)	53	45	58	52	55	50	55
Previously implanted ICD/CRTD	No	No	No	No	No	No	No
Clinical presentation	Stable VT	Stable VT	Stable VT	Stable VT	Recurrent VT with syncope	Recurrent VT with severe fatigue	Stable VT
Hospitalization days before VT ablation	7	8	5	6	7	10	7
UDS +ve for cocaine	Y	Y	N	Y	Y	Y	Y
Other drugs	Y	N	N	N	Y	N	Barbiturate
Amphetamine	Y	N	N	N	Y	Y	Y
Significant CAD	N	N	N	N	N	N	N
Evidence of scar in LV (MRI)	N	N	N	N	N	N	N

AAD = antiarrhythmic drug; AF = atrial fibrillation; B = black; CAD = coronary artery disease; CCB = calcium channel blockers; Cr = creatinine; F = female; HTN = hypertension; ICD = implantable cardioverter defibrillator; LV = left ventricle; M = male; N = no; W = white; Y = yes; LVEF = left ventricular ejection fraction.

TABLE 2

Baseline Characteristics of Patients with Reentrant Ventricular Tachycardia

Patient ID	R1	R2	R3	R4	R5	R6	R7†
Age	47	18	38	39	29	64	68
Gender	M	F	F	M	F	M	M
Race	W	W	W	W	B	W	W
Diabetes mellitus	N	N	N	N	N	N	N
HTN	N	N	N	Y	Y	Y	N
Hyperlipidemia	N	N	N	N	N	N	Y
CAD	Y	N	N	N	N	N	Y
Cr > 1.5	N	N	N	N	Y	Y	N
AF	N	N	N	N	N	N	Y
Cocaine use within 7 days of admission	Y	Y	N	Y	Y	Y	Y
β-Blocker	Y	N	Y	Y	Y	N	Y
ACE inhibitor	Y	N	Y	N	N	Y	Y
CCB	N	N	N	Y	Y	N	Y
Failed AAD either outpatient or inpatient	Y	Y	Y	Y	Y	Y	Y
Type of AAD at the time of VT ablation	Amiodarone	Amiodarone, Mexiletene	Amiodarone	Amiodarone + Mexiletene	Amiodarone	Amiodarone + Lidocaine	Amiodarone + Lidocaine
LVEF	15	45	30	30	60	48	22
Previously implanted ICD/CRTD	Yes	No	No	No	No	Yes	Yes
Clinical presentation	Recurrent ICD Rx & unstable VT	Recurrent stable VT	Stable VT	Unstable VT	Unstable VT with syncope	Recurrent stable VT & ICD Rx	Recurrent ICD Rx
Hospitalization days before VT ablation	6	5	3	6	7	6	8
UDS +ve for cocaine	Y	Y	N	Y	Y	Y	Y
Other drugs	N	Y	N	Marijuana	N	N	N
Amphetamine	N	Y	N	Y	Y	N	N
Significant CAD	Y	N	N	N	N	N	N
Evidence of scar in LV (MRI)	Y	Y	N	Y	Y	Y	Y
Location of myocardial scar	LV Apical anteroseptal	LV basal anterolateral		LV basal inferoseptal and mid lateral	Apical septum	LV basal inferior	LV inferoseptal and distal apical

[†]This patient had a repeat procedure 10 days later. VTs with CL of 575, 570, 455, and 414 milliseconds were induced. AAD = antiarrhythmic drug; AF = atrial fibrillation; B = black; CAD = coronary artery disease; CCB = calcium channel blockers; Cr = creatinine; F = female; HTN = hypertension; ICD = implantable cardioverter defibrillator; LV = left ventricle; M = male; N = no; W = white; Y = yes.

TABLE 3

Procedural Characteristics of Patients with Reentrant Ventricular Tachycardia

Patient ID	F1	F2	F3	F4	F5	F6	F7
Morphology of clinical VT	LBBB, Sup axis, negative precordial	LBBB, Sup axis	LBBB, Inf axis, trans V4	LBBB, Inf axis, trans V3	RBBB, Sup axis, trans V4	LBBB, Sup axis, negative in precordial	LBBB, Inf axis, trans V3, negative in Avl and I
CL (milliseconds)	665	290	390	520	566	462	400
Hemodynamic stability	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Response to BB/CCB/AAD	None	None	Partial to CCB	None	Partial to AAD	Slowed	Slowed
3D mapping	Carto	Carto	Carto	Carto	Carto	Carto	Carto
ICE	Yes	Yes	Yes	Yes	Yes	Yes	Yes
If scar on 3d map	No	No	No	No	No	No	No
Location of successful ablation	LV apex	RV apical septum	LV distal LAF	RVOT anteroseptal	LV distal posterolateral	LV apical	LV mid anterolateral
VT CL (milliseconds) during ablation	670	530	461	521	580	460	400

AAD = antiarrhythmic drugs; BB = β-blocker; CCB = calcium channel blocker; CL = cycle length; ICE = intracardiac echo; Inf = inferior; LAF = left anterior fascicle; LBBB = left bundle branch block; LV = left ventricle; RV = right ventricle; RVOT = right ventricular outflow tract; Sup = superior.

TABLE 4

Procedural Characteristics of Patients with Reentrant Ventricular Tachycardia

Patient ID	R1	R2	R3	R4	R5	R6	R7
Morphology of clinical VT	LBBB, Sup axis, negative precordial	RBBB, Sup axis, trans V3	LBBB, Sup axis, negative precordial	RBBB, Sup axis, postive precordial QRS	RBBB, Sup axis, R/S trans V5	RBBB, Inf axis, trans V1, negative in leads I And Avl	RBBB, M-shaped, Sup axis, V4 trans
CL (milliseconds)	400	300	320	375	469	390	330
Hemodynamic stability	Yes	Yes	Yes	No	Yes	No	No
Response to BB/CCB/AAD	Slowed	None	Slowed	None	None	None	None
3D mapping	Carto	Carto	Carto	Carto	Carto	Carto	Navx
ICE	Yes	Yes	Yes	Yes	Yes	Yes	Yes
If scar on 3D map	Yes	Yes	Yes	Yes	Yes	Yes	Yes
Location of successful ablation	Apex epicardial	Lateral endo/ epicardial	Apex epicardial	LV submitral annular posterior base/LV submitral annular anterolateral base	LV distal septum	LV Basal inferolateral	LV inferoseptal and basal inferior
VT CL (milliseconds) during ablation	400	300	320	375, 400	470	390	390, 360, 370, 330

AAD = antiarrhythmic drugs; BB = β-blocker; CCB = calcium channel blocker; CL = cycle length; ICE = intracardiac echo; Inf = inferior; LAF = left anterior fascicle; LBBB = left bundle branch block; LV = left ventricle; RV = right ventricle; RVOT = right ventricular outflow tract; Sup = superior.

TABLE 5

Follow-Up Characteristics in the Study Patients

Patient No.	R1	R2	R3	R4	R5	R6	R7	F1	F2	F3	F4	F5	F6	F7
ICD Implant before discharge	–	Yes	–	Declined	Yes	–	–	–	–	–	–	–	–	–
3 months recurrence	No	No	No	No	No	Yes	Yes†	No	No	No	No	No	No	No
6 months recurrence	No	No	No	No	No	No	No	No	No	Yes, different morphology	No	No	No	No
Mortality 1 year	Alive	Alive	Alive	Dead	Alive	?	Dead	Alive	Alive	Alive	Alive	Alive	Alive	Alive
Procedure time (minutes)	720	260	320	310	221	474	350	180	210	156	223	196	180	245
EF at last F/U	50	50	35	–	60	–	10	56	60	58	50	59	55	–
Complications	None	None	Tamponade	Groin hematoma	None	None	None on the first procedure	None	None	None	None	None	None	Mild pericardial effusion

†Recurrence of VT, underwent repeat ablation and died before discharge due to sepsis. ? = lost to follow-up.

TABLE 6

Differences in Clinical and VT Characteristics Between Patients Who Had Evidence of Scar Versus Those Who Did Not Have Scar on Electroanatomic Mapping

Clinical Characteristics	Myocardial Scar (N = 7)	No Myocardial Scar (N = 7)	P Value
Age	44 ± 20	44 ± 7	1
Diabetes	0	3	0.2
EF	37 ± 17	53 ± 4	0.4
Mean VT CL (milliseconds) at the time of presentation	368 ± 58	518 ± 87	0.004
Mean CL of induced VTs (milliseconds) during the procedure	414 ± 18	517 ± 89	0.001
Mean procedure time (minutes)	389 ± 184	199 ± 30	0.02
Mortality	29%	0%	0.001