Catheter ablation of ventricular tachycardia after left ventricular reconstructive surgery for ischemic cardiomyopathy

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BACKGROUND After surgical ventricular restoration (SVR) for ischemic cardiomyopathy, ventricular tachycardias (VTs) are an important reason for postoperative morbidity and mortality.

OBJECTIVE The purpose of this study was to elucidate the VT substrate, VT characteristics, and outcome of radiofrequency catheter ablation (RFCA) in patients with VT after SVR.

METHODS Twelve (3%) of 416 patients referred for RFCA for VT after myocardial infarction in three centers had undergone SVR. After induction of VT, left ventricular (LV) electroanatomical mapping was performed. Ablation target sites were identified by entrainment, substrate, and/or pace mapping.

RESULTS Four (33%) patients presented within the perioperative period with incessant VT, and eight (67%) presented with incessant or recurrent VT late after SVR (VT cycle length 453 ± 102 ms). The region of surgical scar was identified by electroanatomical mapping in 11 patients. Twenty-eight VTs (cycle length 384 ± 95 ms) were induced. The VT exit was bordering the surgical scar in 20 (71%) VTs, of which 15 were at the septal side. All VTs were abolished in five patients: in four only the clinical VTs were abolished, and in one reinducibility was not tested. In two patients, ablation failed after which surgical ablation was performed successfully. During follow-up, three (25%) patients died (nonarrhythmic deaths); all had presented early after SVR. Two (17%) experienced recurrent VT.

CONCLUSION VT after LV SVR seems to have a bimodal presentation; one-third presented with incessant VT in the acute postoperative phase and had a high mortality. Two-thirds presented late after SVR; in these patients RFCA is usually effective. Successful ablation sites are frequently located at the border of surgical scars and patch material.

KEYWORDS Surgical ventricular restoration; Ventricular tachycardia; Catheter ablation; Catheter mapping; Outcome

ABBREVATIONS **CL** = cycle length; **CT** = computed tomography; **EA** = electroanatomical; **EPS** = electrical programmed stimulation; **EUS** = electrically unexcitable scar; **LBBB** = left bundle branch block; **LV** = left ventricle; **RBBB** = right bundle branch block; **RF** = radiofrequency; **RFCA** = radiofrequency catheter ablation; **RV** = right ventricular; **SVR** = surgical ventricular restoration; **VT** = ventricular tachycardia

Introduction

Surgical ventricular restoration (SVR) of the left ventricle (LV) is a potential treatment option for patients with extensive LV scar due to ischemic cardiomyopathy.[1] Commonly used techniques are endoventricular repair with or without the use of a patch and aneurysmectomy with linear repair.[1–3] In the early and late postoperative period, ventricular arrhythmias are the second most important cause of death and an important reason for morbidity in these patients.[1,4] It has been demonstrated that surgical scars and patch material influence occurrence and characteristics of reentrant monomorphic ventricular tachycardias (VTs) in patients after repair of congenital heart disease and in patients after corrective valve surgery.[5,6] Despite the growing number of patients undergoing SVR, there are no systematic studies of the substrate and characteristics of sustained monomorphic VTs contributing to postoperative morbidity and mortality.

This study aimed to elucidate the VT substrate, VT characteristics, and outcome of catheter ablation in patients with recurrent VT after SVR.

Methods

Patients

The clinical records of 416 patients referred for radiofrequency catheter ablation (RFCA) for symptomatic VT late after myocardial infarction during 4.0 ± 2.6 (range 2–7) consecutive years in three centers (Brigham and Women's Hospital, Leiden University Medical Center, Hamburg Uni-

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versity Hospital) were reviewed, and 12 patients (3%) were identified who had LV reconstructive surgery defined as SVR by endoventricular patch plasty or LV aneurysm resection. General contraindications for endocardial catheter ablation were absence of vascular access to the LV, echocardiographic evidence of mobile LV thrombus, combined aortic and mitral valve replacement, and unstable angina. All patients who underwent catheter ablation after SVR were included in the study. Data regarding patient characteristics, clinical history, and acute outcome were obtained from the medical record.

Electrophysiological study and mapping

All patients underwent electrical programmed stimulation (EPS) and LV electroanatomical (EA) mapping. The EPS protocol consisted of three drive-cycle lengths (600, 500, and 400 ms) and up to three ventricular extrastimuli from two right ventricular (RV) sites and burst pacing. The positive endpoint of EPS was the induction of VT lasting >30 seconds or requiring termination for hemodynamical instability. Bipolar endocardial EA voltage mapping of the RV and/or LV was performed in all patients, facilitated by a three-dimensional EA mapping system (Carto XP EP System, Biosense Webster Inc., Diamond Bar, CA) during sinus or paced rhythm. A 4- or 3.5-mm-tip, irrigated quadripolar mapping catheter (NaviStar or Navistar ThermoCool, Biosense Webster Inc.) was used inserted via a transvenous or retrograde aortic approach. EA scar was defined by voltage criteria. Electrogram amplitudes ≤0.5 mV were defined as dense scar, and voltages >0.5 mV and ≤1.5 mV[7,8] as scar border zone. At low-amplitude sites, pacing was performed with 10 mA at 2-ms pulse width. Sites with a pacing threshold >10 mA were tagged as electrically unexcitable scar (EUS).[9]

Identification and ablation of VT circuits

Hemodynamically tolerated VTs were mapped and ablated during VT. Target sites for ablation were sites with an isolated mid-diastolic potential where pacing entrained the VT with concealed fusion and a postpacing interval within 30 ms of the VT cycle length (CL) and a stimulus-to-QRS interval <70% of the VT CL. For fast and hemodynamically nontolerated VT, exit sites were identified and targeted on the basis of pace mapping during sinus rhythm with a paced QRS morphology similar to the VT QRS morphology, indicating a potential reentry circuit isthmus site. Pacing was performed at the EA scar border zone and at sites where electrograms with low-amplitude fragmented components, double potentials, and/or isolated late potentials could be recorded during sinus rhythm. Substrate modification by linear ablation lines along the scar border extending lesions at exit sites or through identified isthmus was performed at the discretion of the operator. Complete procedural success was defined as the absence of any inducible monomorphic VT at the end of the ablation procedure. Partial success was defined as successful ablation of the clinical or presumed clinical tachycardia and ablation failure if the clinical or presumed clinical VT could not be successfully ablated. The definitions and ablation strategy used were alike in the three participating centers.

Follow-up

As a rule, all antiarrhythmic drugs, with the exception of intravenous drugs, were continued after the ablation procedure and at discharge from the hospital. Data regarding survival were collected (from the Social Security Death Index for the US patients and from long-term follow-up visits for the Dutch and German patients). The date of last contact, heart transplantation, or death was considered the date of last follow-up.

Results

The characteristics of the 12 patients (all male, age 64 ± 8 years) after myocardial infarction and LV reconstructive surgery included in the study are summarized in Table 1. Spontaneous VT had occurred before surgery in three patients. The indication for surgery had been congestive heart failure in 11 patients and inferior wall rupture due to acute inferior infarction in one patient. SVR reshaping the LV with an endoventricular patch was performed in eight, and aneurysm resection in four patients. Concomitant procedures were mitral/tricuspid valve repair in five, coronary artery bypass grafting in nine, and endocardial encircling cryoablation for VT in one patient. The LV ejection fraction at referral was 25% ± 9%.

VT had been refractory to class III (amiodarone in 10) or to a combination of class I and III antiarrhythmic drugs in all patients. In three patients, a previous RFCA attempt in another center had failed. Four (33%) patients presented early, within the perioperative period (<30 days) after surgery with an incessant monomorphic VT. The other eight (67%) patients presented with incessant monomorphic VT (n = 2), electrical storm (n = 1), or recurrent VT (n = 5) 3.8 ± 1.4 years after LV reconstructive surgery. Before ablation, 17 different VT morphologies with a CL of 453 ± 102 ms were documented. The mean VT CL of patients presenting in the early postoperative phase was 496 ± 87 ms and in those presenting late after SVR, 430 ± 106 ms. VT was hemodynamically tolerated and was relatively slow (CL >400 ms) in eight patients. At referral, nine (75%) patients had an implantable cardioverter-defibrillator (ICD).

Electrophysiological evaluation and mapping

A total of 28 monomorphic VTs (CL 384 ± 95 ms) were induced (2.3 ± 1.2 per patient). VT characteristics are provided in Table 2. Sustained polymorphic VT was not induced in any of the patients. EA mapping of the LV was performed in 11 patients. In one patient (patient 2), only RV mapping was performed since the patient presented with left bundle branch block (LBBB) like VT and the LV was not safely accessible owing to coexisting mitral valve endocarditis. In the 11 patients with LV mapping, an area of EUS or very low voltage was present in the region of reconstructive surgery. In all patients in whom patch material was used at