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Amiodarone Discontinuation or Dose Reduction Following Catheter Ablation for Ventricular Tachycardia in Structural Heart Disease

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ABSTRACT

OBJECTIVES This study sought to examine long-term outcomes in patients with structural heart disease in whom amiodarone was reduced/discontinued after ventricular tachycardia (VT) ablation.

BACKGROUND VT in patients with structural heart disease increases morbidity and mortality. Amiodarone can decrease VT burden, but long-term use may result in organ toxicities and possibly increased mortality. Catheter ablation can also decrease VT burden. Whether amiodarone can be safely reduced/discontinued following ablation remains unknown.

METHODS We studied consecutive patients undergoing VT ablation from 2008 to 2011, typically followed by noninvasive programmed stimulation several days later. Patients were divided into 3 groups by amiodarone use: group A—amiodarone reduced/discontinued following ablation; group B—amiodarone not reduced; group C—not on amiodarone at time of ablation. Baseline characteristics and outcomes were compared between groups.

RESULTS Overall, 231 patients (90% male; mean age: 63.4 ± 12.9 years; 53.7% ischemic cardiomyopathy) were included (group A: 99; group B: 29; group C: 103). Group B patients were older with more advanced heart failure. Group A patients less frequently had inducible VT at the end of ablation or noninvasive programmed stimulation. In follow-up, 1-year VT-free survival was similar between groups ($p = 0.10$). Mortality was highest in group B ($p < 0.001$). Higher amiodarone dose after ablation (hazard ratio: 1.23; 95% confidence interval: 1.03 to 1.47; $p = 0.02$) was independently associated with shorter time to death.

CONCLUSIONS After successful VT ablation, as confirmed by noninducibility at the end of ablation and noninvasive programmed stimulation, amiodarone may be safely reduced/discontinued without an unacceptable increase in VT recurrence. Reduction/discontinuation of amiodarone should be considered an important goal of VT ablation. (J Am Coll Cardiol EP 2017;■:■-■) © 2017 by the American College of Cardiology Foundation.

Ventricular tachycardia (VT) frequently occurs in patients with ischemic and non-ischemic cardiomyopathies. Whereas implantable cardioverter-defibrillators (ICD) prolong life in appropriately selected patients, ICD shocks themselves may be associated with increased morbidity and mortality (1,2). Antiarrhythmic drugs (AAD) are frequently prescribed to suppress VT in patients with structural heart disease. Amiodarone is the most effective AAD (3), but it can cause several

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ABBREVIATIONS AND ACRONYMS

AAD = antiarrhythmic drug(s)

ICD = implantable cardioverter-defibrillator

LVEF = left ventricular ejection fraction

NIPS = noninvasive programmed stimulation

NYHA = New York Heart Association

VT = ventricular tachycardia

organ toxicities with long-term use, which is particularly concerning in younger patients (4-7). Catheter ablation is a potentially curative treatment option for VT.

Management of AAD following VT ablation has not been well studied. Whereas the 2009 Consensus Statement on Catheter Ablation of Ventricular Arrhythmias states that amiodarone dose reduction or discontinuation may be considered following apparently successful ablation, this recommendation is based on expert consensus rather than clinical evidence (8). To our knowledge, outcomes following amiodarone reduction or discontinuation after VT ablation in patients with structural heart disease have not been previously reported.

We hypothesized that amiodarone could be reduced or discontinued following apparently successful VT ablation without an unacceptable increase in risk of VT recurrence. We tested this hypothesis retrospectively in our large, single center experience.

METHODS

STUDY POPULATION. The study cohort consisted of consecutive patients with sustained VT and structural heart disease who were referred to the Hospital of the University of Pennsylvania for VT ablation between January 1, 2008 and June 30, 2011. Patients with idiopathic VT were excluded. Whenever possible, AAD were discontinued at least 5 half-lives prior to the procedure; amiodarone was discontinued 2 weeks prior to ablation when possible. Per institutional guidelines, all patients provided written informed consent both for the ablation procedure and for their anonymized medical information to be included in research studies.

CATHETER ABLATION. Conscious sedation was used whenever possible. General anesthesia was used when necessary for ventilation, oxygenation, or patient comfort. In addition, general anesthesia was routinely initiated prior to obtaining epicardial access.

Electroanatomic mapping (CARTO, Biosense Webster, Inc., Diamond Bar, California) was performed during sinus or paced rhythm to define areas of low voltage and abnormal electrograms, consistent with scar (9,10). Programmed stimulation was performed, and induced VT were compared to those occurring spontaneously. Clinical VT was identified by matching the QRS morphology with either a 12-lead electrocardiogram (when available) or the stored ICD cycle length, as well as near-field and far-field electrogram morphology. All spontaneously occurring VT was considered clinical; thus, a single patient could have multiple clinical VTs. Special attention was paid to elimination of clinical VT. Additionally, all mappable VT and all VTs with cycle length >250 ms were considered relevant and targeted for ablation whenever possible.

When hemodynamically tolerated, entrainment mapping was used to define critical components of the VT circuit. If VT was not mappable, substrate modification was performed with linear and/or cluster lesions targeting sites identified by pace mapping and late potentials. Ablation was typically performed using an irrigated ablation catheter (Thermocool, Biosense Webster, Inc.) using powers up to 50 W with a goal of 12- to 15-Ω impedance drop. Epicardial mapping and ablation were performed when 12-lead electrocardiogram of VT suggested an epicardial exit and/or endocardial ablation failed to eliminate targeted VT (11). At the end of the ablation procedure, programmed stimulation was repeated in patients who were medically stable, with up to 3 ventricular extrastimuli delivered to refractoriness from up to 2 sites at 2 pacing cycle lengths.

NONINVASIVE PROGRAMMED STIMULATION. In the absence of clinical VT being inducible at the end of ablation or spontaneous VT recurrence, noninvasive programmed stimulation (NIPS) was typically performed within several days of ablation, before hospital discharge, as previously described (12). AAD were not restarted following ablation until after NIPS, if at all. In the fasting state, with intermittent boluses of propofol titrated to deep sedation, single, double, and then triple ventricular extrastimuli were delivered to refractoriness at drive trains of 600 and 400 ms, via the right ventricular ICD lead. In patients without ICDs, programmed stimulation was performed via a quadripolar catheter advanced through the femoral vein to the right ventricle. Response to NIPS was categorized as “clinical VT inducible” if any sustained, monomorphic VT was induced matching any spontaneous VT. Response was categorized as “nonclinical VT inducible” if only sustained monomorphic VT not matching any of the clinical VT was induced. Finally, if no sustained monomorphic VT could be induced, the response was categorized as “no VT inducible.”

Patients with inducible nonsustained monomorphic VT, polymorphic VT, or ventricular fibrillation only were included in the “no VT inducible” group. The NIPS results were used for prognostic purposes, to guide AAD prescription, and to optimize ICD programming. The detection rate was