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STATE-OF-THE-ART REVIEW

VT Ablation in Nonischemic Cardiomyopathy

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ABSTRACT

Catheter ablation is being increasingly performed as adjunctive treatment to prevent recurrent implantable cardioverter-defibrillator therapies in patients with nonischemic cardiomyopathy and ventricular tachycardia (VT). In the context of VT ablation, nonischemic cardiomyopathy usually refers to dilated cardiomyopathy (DCM) as one morphological phenotype. Over the past decades, progress has been made to better characterize distinct subtypes and to differentiate between causes of DCM, which has important practical and prognostic implications. The goal of this review is to summarize available data on VT ablation in patients with DCM and, more specifically, review procedural and outcome data in specific etiologies and substrate location. It will focus on our current understanding of nonischemic scars, as well as the value of multimodal imaging, image integration, and electroanatomic mapping for substrate identification, procedural planning, and ablation. In addition, recent findings from whole human heart histology of patients with DCM and VT and their potential implications for imaging and mapping will be discussed. (J Am Coll Cardiol EP 2018;■:■–■) © 2018 by the American College of Cardiology Foundation.

Nonischemic cardiomyopathies (NICMs) have been classified according to morphological and functional phenotypes and include dilated (DCM), hypertrophic (HCM), restrictive, arrhythmogenic right ventricular (ARVC), and left ventricular noncompaction (LVNC) cardiomyopathies (1,2). In the context of catheter ablation of monomorphic ventricular tachycardias (MVTs), NICM often refers to the phenotype of a left dominant NICM or DCM, usually excluding patients with an HCM, restrictive cardiomyopathy, ARVC, or LVNC phenotype (3–5).

DCM has historically been defined by the presence of left ventricular (LV) dilatation and LV systolic dysfunction in the absence of abnormal loading conditions (hypertension, valve disease) or coronary artery disease (1). This DCM phenotype is in fact an umbrella term for different underlying etiologies that might be the consequence of genetic or acquired factors (or a combination of the two). Genetic testing has increasingly been integrated in clinical evaluation and provides important insights into early phases of a disease that does not meet the standard disease definition (6). Of importance, early phases of DCM can already be associated with life-threatening ventricular tachycardia (VTs) such as those typically observed in patients with mutations in the *LMNA* gene (7). Myocarditis as an acquired cause is another challenging diagnosis because of the heterogeneity of the clinical presentation and multiple underlying pathogens. The acute course can be asymptomatic but can range from mild symptoms with only transient ST-T-wave changes to cardiogenic shock. It can heal with small subepicardial scars or progress to a dilated cardiomyopathy with poor prognosis (8,9).

Advanced image modalities and comprehensive diagnostic workup, including high-resolution 3-dimensional (3D) late gadolinium enhancement

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ABBREVIATIONS AND ACRONYMS

3D = 3-dimensional

ARVC = arrhythmogenic right ventricular cardiomyopathies

AUC = area under the curve

BV = bipolar voltage

CS = cardiac sarcoidosis

DCM = dilated cardiomyopathies

EAVM = electroanatomic voltage mapping

ECG = electrocardiogram

EF = ejection fraction

EGM = electrogram

EMB = endomyocardial biopsy

HCM = hypertrophic cardiomyopathies

ICM = ischemic cardiomyopathy

ICD = implantable cardioverter-defibrillator

ILS = inferolateral scar

LBBB = left bundle branch block

LGE-CMR = late gadolinium enhancement cardiac magnetic resonance imaging

LP = late potentials

LV = left ventricular

LVNC = left ventricular noncompaction cardiomyopathies

MDCT = multidetector computed tomography

MSVT = monomorphic sustained ventricular tachycardia

MVT = monomorphic ventricular tachycardia

NICM = nonischemic cardiomyopathy

PN = phrenic nerve

RBBB = right bundle branch block

RFCA = radiofrequency catheter ablation

RV = right ventricle

SI = signal intensity

UV = unipolar voltage

VT = ventricular tachycardia

WT = wall thickness

cardiac magnetic resonance imaging (LGE-CMR), nuclear imaging, endomyocardial biopsy (EMB), and biomarkers allow (early) tissue-based recognition of specific nongenetic etiologies. As a consequence, diseases previously thought to be rare, such as isolated cardiac sarcoidosis (CS) or chagasic cardiomyopathy outside Latin America, might be much more frequently diagnosed as the underlying cause of DCM (10,11).

Despite the similarities in arrhythmogenic presentation and phenotypic overlap, different etiologies can have fundamentally different patterns of myocardial injury and fibrosis (12). The substrate can change over time, with disease progression dependent on the etiology (13,14).

Treatment of VT in DCM requires a comprehensive understanding of the substrate in an individual patient. Ablation outcome depends on identification and accessibility of the substrate, as well as the natural course of the disease (3,12,15–17).

This review will assess available data on VT ablation in mixed cohorts of patients with DCM and specifically in subgroups of patients with known etiologies and substrate location. Practical considerations and bailout strategies will be discussed. It will focus on our current understanding of nonischemic scars, their identification by imaging and electroanatomic mapping, and their differences according to etiologies. A comprehensive review of all known acquired and inherited causes of DCM is beyond the scope of this review but has been published recently (18).

MVT IN DCM

The first mapping studies in patients with DCM demonstrated that >80% of monomorphic sustained VT (MSVTs) are due to myocardial re-entry (19,20), and occasionally to triggered activity, both of which are associated with the presence of scar. VTs originating from the His-Purkinje system are less common but important to recognize. The relative contribution of bundle branch re-entry to inducible MSVT is higher in nonischemic etiologies (up to 40%) than in ischemic etiologies (up to 6%) (21). Bundle branch re-entry can occur in the presence of a preserved LV function (typical for myotonic dystrophy) and is susceptible to ablation, with high success rates because of the well-defined and easily approachable substrate (21,22).

Among DCM patients, the propensity for MSVTs that are amenable to radiofrequency catheter ablation (RFCA) is related to the etiology and amount and location of myocardial scar (23–25). LGE-CMR performed before implantable cardioverter-defibrillator (ICD) implantation demonstrated that LGE extent and, specifically, involvement of basal LV segments appeared to be the strongest and an independent predictor of MSVT (25). In unselected DCM patients who received ICDs for primary prevention of sudden cardiac death, MSVT responsive to antitachycardia pacing occurred in 17% of patients and appropriate ICD shock for ventricular fibrillation or rapid VT in 11.5% during a median follow-up of 68 months (26). Prophylactic ICD implantation was not associated with a lower rate of death compared with usual clinical care. In the control group not randomized to ICDs, sustained VT requiring medical intervention or electrical cardioversion or cardiac arrest occurred in only 2.5% and 2.5%, respectively. In contrast, >50% of patients with an *LMNA* mutation who received an ICD for primary prevention received appropriate ICD therapy during a median follow-up of 62 months (23). Of importance, LV ejection fraction (EF) was ≥45% in 82% of these LMNA patients at the time of the event. Similarly, the estimated incidence rate for appropriate ICD therapy in patients with CS was 15% per year, with an EF >35% in 41% of patients who had an event (24,27).

ABLATION OUTCOME IN MIXED GROUPS OF DCM

Data from the 2003 to 2014 National Inpatient Sample databases suggest an increasing trend in the use of VT ablation after the exclusion of patients with coronary artery disease (28). Current guidelines recommend RFCA as adjunctive treatment to prevent recurrent ICD therapies for MVT that cannot be controlled by amiodarone or sotalol independent of the underlying etiology (29). In tertiary referral centers, the proportion of patients with NICM among those referred for VT ablation has been reported at 15% to 25%, but this number is growing in specialized centers (5,30).

Short- and long-term outcome data after RFCA in mixed cohorts of DCM patients come from single-center observational studies (Online Table 1). Reported procedural success rates, often defined as noninducibility of any VT, range from 38% to 74%, with VT recurrence rates between 29% and 58% during a median follow-up of 9 to 22 months (3,5,19,20,31,32). Although this procedure is