Ventricular Tachycardia in Nonischemic Dilated Cardiomyopathy

Electrocardiographic and Intracardiac Electrogram Correlation

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KEYWORDS

• Nonischemic cardiomyopathy • Ventricular tachycardia • Catheter ablation
• Electroanatomic mapping • Epicardial

KEY POINTS

- The substrate for ventricular tachycardia (VT) in nonischemic cardiomyopathy (NICMP) is frequently basal scar that is epicardial and midmyocardial.
- VTs with an epicardial exit take longer to engage the His-Purkinje system, with greater initial slurring of the QRS and time to maximal deflection seen on electrocardiography.
- Anisotropic conduction through scar in NICMP results in late potentials and local abnormal ventricular activity that can be targeted for ablation.
- Unipolar electrograms with a wider field of view are useful for identifying epicardial and intramural substrate endocardially.

INTRODUCTION

Ventricular tachycardia (VT) is part of the clinical sequelae of nonischemic cardiomyopathy (NICMP) with dysfunction of the left ventricle (LV) in the absence of occlusive coronary artery disease or congenital heart disease. The prevalence of NICMP, also commonly referred to as idiopathic cardiomyopathy and dilated cardiomyopathy, is difficult to estimate, because of the heterogeneity in definitions and diagnostic criteria, selection bias, and geographic variation. In addition, there are clear differences in population characteristics between community-based studies versus analyses of populations from referral centers.[1] The estimated prevalence of NICMP is 36 to 40/100,000.[2] Its incidence discovered at autopsy is estimated to be 4.5 cases per 100,000 population per year, whereas the clinical incidence is 2.45 cases per 100,000 population per year.[3]

There is an estimated annual mortality of 7%, despite optimal medical therapy in patients with NICMP.[4] Sudden unexplained death accounts for 50%, and although primary bradyarrhythmias and asystole are contributory, VT/ventricular fibrillation accounts for many of these sudden cardiac deaths.[5] Consistent with this finding, a meta-analysis of implantable cardioverter-defibrillator

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(ICD) trials in patients with NICMP showed a 26% mortality reduction with ICD therapy,[6] which indicates that a significant proportion of deaths in patients with NICMP is tachyarrhythmia related. Accordingly, there has been an increase in the proportion of patients with NICMP and VT compared with ischemic cardiomyopathy (ICMP) in recent years, which may reflect: (1) more aggressive reperfusion strategies for acute myocardial infarction, reducing the number of patients with ICMP who need VT ablation, (2) more patients with NICMP with VT surviving to require ablation as a result of advanced medical therapy, and (3) greater use of ICDs or changes in referral patterns.[7]

The acute success of catheter ablation in NICMP has varied from 21% to 79% and is generally lower than for ICMP.[8–11] Unlike ICMP, endocardial mapping and ablation alone are often insufficient to achieve noninducibility in NICMP, because circuits are frequently intramural or epicardial.[12,13] This situation partially accounts for the lower success rates of initial series of VT ablation in NICMP, before the more routine access of the epicardial space.

Recognizing the electrocardiographic (ECG) characteristics that localize the exit or focus of the VT and intracardiac electrogram (EGM) features that delineate the substrate of the arrhythmia that can be targeted for ablation is essential to the development of a successful ablation strategy. These ECG and EGM characteristics are also useful to predict success and durability of the procedure, which influences the management approach for the patient's arrhythmia. This article focuses on these ECG and EGM characteristics of VT in NICMP.

PATHOLOGY

The most common arrhythmia mechanisms in patients with NICMP include (1) scar-based reentry, (2) abnormal automaticity, and (3) Purkinje system–related arrhythmias.[9,14] Autopsy series of explanted hearts in patients with NICMP have shown interstitial and replacement fibrosis. A necropsy study of 152 patients with idiopathic dilated cardiomyopathy reported a high incidence of myocardial fibrosis (57%), despite a relative paucity of visible scar (14%). The ventricular myocardium is histologically characterized by variable degrees of myocyte hypertrophy and atrophy, with replacement by fibrosis, leading to multiple patchy areas of fibrosis and myofiber disarray.[15–18] Unlike in ICMP, these changes are not limited to the endocardium but also have a predilection to the midmyocardial and epicardial layers of the basilar and perivalvular regions of the LV.[19,20] In addition to the presence of fibrosis and scar, the electric conduction properties of the myocardium are also altered. The altered cellular processes and abnormal membrane potentials associated with myocyte hypertrophy may be arrhythmogenic. Altered automaticity from delayed after-depolarizations or early after-depolarizations resulting in focal VT has been reported in patients and animal models of dilated cardiomyopathy.[21–23]

Similar to ICMP, the most common mechanism for monomorphic VT in NICMP is reentry.[10,14] The electrophysiologic mapping and histologic examination of hearts from transplant recipients with dilated cardiomyopathy have shown areas of fibrosis that form the substrate for VT by creating conduction barriers with fractionated EGMs and heterogeneous patterns of epicardial and endocardial activations with marked conduction disturbances that are the nidus for reentry.[22,24] The amount of fibrosis and myofiber disarray correlated with higher levels of nonuniform anisotropy and generation of reentrant wave fronts. The patients with greater degrees of electric abnormality were noted to have a greater burden of nonsustained ventricular arrhythmia on Holter monitoring.[24]

The conduction slowing in NICMP may occur within the specialized His-Purkinje system. Conduction delay in the His-Purkinje system can provide the requisite substrate for monomorphic ventricular arrhythmias from bundle branch reentry (BBR) or fascicular reentry; the average HV interval in patients with BBR VT is approximately 80 milliseconds.[25] BBR has been reported as the arrhythmia mechanism in 30% to 40% of patients with NICMP presenting with monomorphic VT.[19,26] The macro reentrant circuit of BBR typically initiates with a premature ventricular beat, which conducts slowly up the left bundle with retrograde block in the right bundle. The delay in left bundle conduction provides sufficient time for recovery of the right bundle and hence, subsequent antegrade conduction down the right bundle followed by transeptal intramyocardial conduction (**Fig. 1**). Less commonly, the reentrant circuit can be depolarized in the opposite direction, generating a QRS morphology with a right bundle branch block (RBBB) configuration. The same mechanism with difference in conduction velocity and refractory periods between the left fascicles can result in an interfascicular VT.[27]

ECG CHARACTERISTICS

For VT resulting from myocardial reentry in patients with NICMP, there are ECG characteristics