Myocardial wall thinning predicts transmural substrate in patients with scar-related ventricular tachycardia

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BACKGROUND Scar-related ventricular tachycardia (VT) arises from specific substrate according to etiology.

OBJECTIVE The purpose of this study was to evaluate the relationship between wall thinning (WT) on multidetector computed tomography (MDCT) and local abnormal ventricular activity (LAVA) in patients with ischemic cardiomyopathy (ICM), postmyocarditis (PMC), and dilated cardiomyopathy (DCM).

METHODS Forty-two patients (40 male, age 58 ± 13 years, 22 ICM, 11 PMC, 9 DCM) underwent MDCT before a combined endo-/epicardial VT ablation procedure. WT (<5 mm) and severe wall thinning (SWT) (<2 mm) area on MDCT were compared to the prevalence of endo-/epicardial LAVA during sinus rhythm.

RESULTS WT and SWT were found on MDCT in 36 (86%) and 20 (48%) with 42 ± 37 cm^2 and 26 ± 24 cm^2, respectively. SWT was frequently detected in ICM (ICM 77% vs PMC 27% vs DCM 0%, P <.001). LAVA were frequently observed on the endocardium in ICM and on the epicardium in PMC. Endo-/epicardial facing LAVA were frequently found within SWT areas (91% in <2 mm, 9% in 2–5 mm, and 0% in >5 mm, P < .001). In SWT areas, the presence of endocardial LAVA in ICM and epicardial LAVA in PMC predicted opposite facing LAVA with sensitivity and specificity of 78% and 48% and 79% and 98%, respectively. SWT predicted epicardial LAVA in ICM and endocardial LAVA in PMC with sensitivity and specificity of 89% and 100%, and 100% and 100%, respectively.

CONCLUSION SWT is frequently found in ICM and PMC but is not common in DCM. SWT predicts LAVA on the opposite side of the wall (epicardial in ICM and endocardial in PMC), indicating transmural VT substrate. MDCT is useful for identifying VT substrate and helpful for understanding the mechanisms of the location of VT substrate domain.

KEYWORDS Ventricular tachycardia; Ablation; Imaging; Wall thinning; Multidetector computed tomography

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Introduction

Scar-related ventricular tachycardia (VT) can be treated by catheter ablation during VT and sinus rhythm (SR) with reduction of VT burden and mortality.[1–3] Elimination of substrate defined by local abnormal ventricular activity (LAVA) can improve clinical outcome,[3,4] which suggests that LAVA play an important causative role in scar-related VT.

The distribution of VT substrate depends on the etiology. Nonischemic cardiomyopathy (NICM), including dilated cardiomyopathy (DCM) and postmyocarditis (PMC), often demonstrates VT substrate on the epicardial aspect.[5,6] In contrast, VT substrate in ischemic cardiomyopathy (ICM) is often confined to the endocardium, with fewer cases needing an epicardial approach to eliminate VT.[4]

For identification of VT substrate, cardiac magnetic resonance imaging (CMR) using late gadolinium enhancement (LGE) is the emerging gold standard modality.[7,8] However, because many patients requiring ablation for VT have an implantable cardioverter–defibrillator, which either contraindicates CMR or degrades the quality of cardiac

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imaging, the utility of CMR in this patient group is limited. Wall thinning (WT) on multidetector computed tomography (MDCT) can also detect VT substrate.[8] Whether WT can predict the distribution of endo-/epicardial substrate in patients with ICM and NICM remains unknown. Therefore, we assessed the relationship between WT on MDCT and the distribution of electrical substrate in the form of LAVA over the endo-/epicardium in patients with ICM/NICM.

Methods

Patient population

Forty-two patients undergoing endo-/epicardial VT ablation underwent image integration from MDCT (22 ICM, 20 NICM [11 PMC, 9 DCM]). The diagnosis of PMC was based on previous clinical history, clinical examination, serology, CMR, and coronary angiographic findings[9] without routine endomyocardial biopsy. The diagnosis of DCM was based on the presence of left ventricular (LV) dilation and systolic dysfunction,[10] and exclusion of coronary artery disease, myocarditis, congenital heart disease, arrhythmogenic right ventricular cardiomyopathy, hypertrophic cardiomyopathy, primary valvular abnormalities, and alcoholic cardiomyopathy. All patients had monomorphic sustained VT demonstrated on 12-lead ECG or implantable cardioverter–defibrillator recordings despite receiving optimal medical treatment. The study was approved by the institutional ethics committee, and all patients provided informed consent.

MDCT acquisition, processing and registration

Imaging processing was performed using custom software (MUSIC, LIRYC-Université de Bordeaux/Inria-Sophia Antipolis, France) as described in our previous report (see Online Supplementary Material).[8] The segmented areas of moderate WT (<5 mm) and severe wall thinning (SWT) (<2 mm) segmented from the MDCT were imported a to 3-dimensional electroanatomic system. The "border zone" was defined as the area <1 cm from border of WT/SWT. Registration was refined using automatic surface registration with the CARTO-3 system (Biosense Webster, Diamond Bar, CA) with 3.8 ± 1.1 mm of registration error, and 41 ± 16 fusion points with the NavX platform (SJM, St. Paul, MN).

Electrophysiologic study and ablation

The preparation of the procedure is described in the Online Supplementary Material. Voltage map was acquired over the endo-/epicardium during SR with a high-density mapping catheter (PentaRay, Biosense Webster) and/or an open-irrigated 3.5-mm-tip catheter (NaviStar ThermoCool, Biosense Webster), which was also used for ablation. On areas demonstrating LAVA with far-field LV potentials, mapping annotation was manually performed to select near-field (LAVA) signal after acquisition of each point.[11,12]

For hemodynamically tolerated VT, ablation (25–50 W endocardial, 25–35 W epicardial) was targeted to critical isthmus sites demonstrating postpacing intervals <30 ms, concealed entrainment, and long S–QRS times (>40 ms).[13] If VT was noninducible or hemodynamically unstable, LAVA-based ablation was performed during SR. LAVA was defined as high-frequency, split, and/or fragmented ventricular potentials, with prolonged duration. Although distinct from the far-field ventricular electrogram, LAVA occur anytime during or after the far-field signal and demonstrate decremental properties when using extrastimulation from the right ventricular apex, due to abnormal conduction in surviving myocardial fibers. By using decrementation, the LAVA signal can be separated from the far-field electrogram.[3] Procedural endpoints were VT noninducibility and complete LAVA elimination. For LAVA detected over the epicardium, ablation was first attempted from the opposing endocardium, with epicardial ablation reserved for persistent LAVA. Radiofrequency (RF) energy was withheld <5 mm from the coronary arteries and left phrenic nerve (confirmed by capture with high-output pacing of 25 mA/2 ms).

Selection of facing LAVA points

"Facing LAVA" was defined as endo- and epicardial LAVA presenting on the opposite side <5-mm radius of each other on the 3-dimensional electroanatomic system (Figure 1). If multiple points were located in the range, the nearest point was selected. The amplitude and Q–LAVA duration (defined as the interval between onset of QRS and peak high-frequency LAVA signal component) on all facing LAVA points were measured.

Statistical analysis

Quantitative data are expressed as mean ± SD when normally distributed and as median (interquartile range) otherwise. To evaluate continuous variables among 3 groups, 1-way analysis of variance and Kruskal–Wallis tests based on parametric and nonparametric variables were used. Comparison between groups was analyzed using the unpaired Student t test or Wilcoxon rank-sum test based on the distribution of the values. The χ^2 test was used to analyze categorical variables, unless the expected values in any cells were <5, in which case the Fisher exact test was used. All tests were 2-tailed, and P <.05 was considered significant.

Results

Baseline characteristics and procedural data are summarized in Table 1. The delay since PMC diagnosis was 5 ± 4 years. Six PMC patients underwent CMR, and all demonstrated subepicardial LGE over the LV lateral wall. CMR was performed in 2 DCM patients and showed no LGE in 1 and nonspecific intramural LGE within the interventricular septum in the other.

Mapping and imaging characteristics

Endocardial LAVA were seen with highest frequency and number in ICM (Table 1). Conversely, epicardial LAVA were