Catheter ablation for ventricular tachycardia (VT) in ischemic heart disease is considered standard therapy in patients with recurrent, sustained monomorphic ventricular tachycardia, usually after failed antiarrhythmic therapy. Many centers worldwide use pre-procedural imaging with cardiac magnetic resonance (CMR) to assess the “scar substrate” in patients with ischemic and non-ischemic cardiomyopathy. CMR’s exquisite delineation of myocardial scar location and extent allows for targeted mapping of areas with known fibrosis and informs whether endocardial versus epicardial access is warranted.

So far, studies have compared pre-procedural CMR with periprocedural variables of VT ablation such as scar size, substrate-related surrogate ablation targets (e.g., late potentials, local abnormal ventricular activities), or successful ablation sites (1–3). Similarly, CMR scar characteristics have been used to define long-term prognosis in ischemic patients not undergoing VT ablation (4,5).

In this issue of JACC: Clinical Electrophysiology, Avila et al. (6) present a study that attempted to prospectively combine those 2 approaches and determine CMR-derived predictors of long-term outcome in ischemic patients after VT ablation. Forty-six consecutive patients (87% males) scheduled to undergo de novo VT ablation (including substrate modification) underwent pre-procedural late gadolinium contrast-enhanced CMR, using a 1.5-T scanner, followed by insertion of an implantable cardioverter-defibrillator (n = 41). First, use of the well-validated full width half maximum (FWHM) and normalized standard deviation methods facilitated calculation of total myocardial scarring and heterogeneous zone burden. Second, signal intensity (SI) maps using FWHM were used to generate 3D shells of dense scar and heterogeneous tissue in endocardial and epicardial halves of the myocardial wall, which were projected onto the corresponding endocardial or epicardial surface. During subsequent VT ablation, endocardial bipolar voltage was used to define scar (0.5 to 1.5 mV) and dense scar (<0.5 mV) tissue. Electrogram signal characteristics (fractionation and late or isolated potentials) and pace-mapping techniques were used to identify conduction channels. Third and finally, endocardial substrate ablation was conducted, targeting complete elimination of electrograms with isolated components/late potentials and conduction channels or failure to capture locally at high output. In 17 patients (37%) ablation failed to achieve complete substrate elimination, and in 3 cases clinical VT remained inducible. VT recurrence was assessed on the basis of implantable cardioverter-defibrillator interrogation or documentation of sustained monomorphic VT (in 5 patients without implantable cardioverter-defibrillators). After a mean follow-up of 32 months, freedom from VT and/or VT therapy was 63%. A higher incidence of VT recurrence...
(primary endpoint) was observed in patients with larger scar masses (31 ± 14 g vs. 21 ± 10 g, respectively; p = 0.014) and scar and heterogeneous tissue areas in SI maps in both the endocardial and epicardial locations. However, in univariate analysis, only the more process-intensive SI map parameters predicted arrhythmia-free survival. Consequently, in multivariate analysis, SI map-based endocardial extension of scar mass by CMR was the only independent predictor of VT recurrence (hazard ratio [HR]: 1.31 per 10 cm² of scar tissue, with a 95% confidence interval [CI]: 1.05 to 1.63; p = 0.034). Overall, freedom from VT recurrence was higher in patients with smaller endocardial scars by CMR (<65 cm² (85%) vs. >65 cm² (20%); log-rank p = 0.018). Nine patients with recurrent VT underwent repeated ablation, including epicardial substrate modification in 5 patients who showed extensive scarring on SI maps.

Despite some limitations such as small sample size and limited use of epicardial mapping, this well-designed study has significant importance. Avila et al. (6) demonstrated the potential role of preprocedural CMR to predict freedom from VT following ablation and expanded the use of CMR beyond that of periprocedural guidance. Findings are consistent with those of previous studies using noninvasive 12-lead electrocardiography or invasive electrophysiology (EP) mapping criteria, which have shown correlation of scar size with VT recurrence and/or mortality following ablation (7,8).

The hypothesis that larger scar areas predict VT recurrence is plausible, as the likelihood of putative VT circuits should be higher in areas of increased ventricular fibrosis and anisotropic electrical conduction. This concept was the underlying rationale for exploring a primary prevention indication based on ischemic scar burden in the DETERMINE (Defibrillators to Reduce Risk by Magnetic Resonance Imaging Evaluation) trial (>10% left ventricle scarring) and demonstrated its value even in non-coronary artery disease patient populations (where hypertrophic cardiomyopathy showed ≥15% left ventricle scarring) (5,9). Interestingly, in Avila et al. (6), the total scar burden or HZ (which can be quantified without further imaging processing) determined using FWHM and normalized standard deviation methods did not predict VT recurrence in univariate or multivariate analysis. However, applying the same FWHM algorithm to the endocardial or epicardial half of the myocardial wall and projecting the labeled voxels onto the 2 surfaces (labeled “SI map” and which requires additional imaging processing), the total amounts of such endo- and epicardial scarring/frequency endpoints were predictive of VT recurrence. This finding is somewhat surprising as the same FWHM algorithm was applied in both of the analyses. A possible explanation may be that, in the first variable analysis, each scar voxel was being weighted identically, like a continuous variable, whereas the second analysis (SI map) included some categorical characteristics, as a wall segment of 100% endocardial scar transmurality was weighted equally as a segment of only 10% endocardial scar transmurality. This increased the analytical weight of lower scar transmural segments. However, it is not intuitively clear why this would present a better predictor for VT recurrence as VT channels are commonly located in areas of low bipolar voltage (representing a more transmural scar region). Previous studies have demonstrated that successful VT ablation sites are commonly localized in areas of rapidly decreasing scar transmurality, presenting a possible explanation for these findings (2,2).

Another interesting observation in this study and in previously published data is the modest correlation between the scar mass assessed by voltage and that by late gadolinium-enhanced CMR. Previous studies (1) showed significant mismatches between voltage- and late gadolinium enhancement-derived scar tissue in 1 of 3 of VT patients. Some of those discrepancies could be explained by intramyocardial scar and lack of catheter-to-tissue contact. However, it raises the question whether CMR- and EP voltage-defined scars are characterizing different aspects of underlying pathophysiological processes which are closely related but not identical. Such differential information could be potentially exploited for an improved substrate characterization and therapeutic decision making.

Although the study by Avila et al. (6) opens new fields of outcome-based research, it is too early to apply the results for clinical decision making. The freedom of VT recurrence of nearly 3 years, even in the patient group with large SI map-based endocardial scar mass, would be a clinically acceptable result in most patients presenting with recurrent VT. Nevertheless, this is an important contribution, extending for the first time the current use of preprocedural CMR from periprocedural guidance toward prediction of long-term outcomes in ischemic patients requiring VT ablation. This will undoubtedly open the door for further noninvasive image-guided outcome research and may ultimately improve patient selection and clinical care.

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