



Scar modification for polymorphic ventricular tachycardia and ventricular fibrillation: hope beyond trigger ablation?

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Polymorphic ventricular tachycardia (PMVT) and ventricular fibrillation (VF) are a common cause of sudden cardiac death in patients with structural heart disease (SHD) and occur less frequently in patients without any identifiable structural or metabolic disease which is defined as idiopathic PMVT/VF. Even though implantable cardiac defibrillator (ICD) therapy is highly effective for aborting sudden cardiac death, recurrent ventricular tachycardia (VT)/VF with subsequent ICD shocks are associated with decreased quality of life, hospital readmissions, and increased mortality.

Polymorphic VT and VF are traditionally viewed as reversible arrhythmias that frequently occur in the setting of myocardial ischemia and efforts to optimize reperfusion with revascularization are paramount. Beta-blockers and antiarrhythmics, including quinidine (1) have been shown to be effective therapies for PMVT. Catheter ablation has been shown to be effective to eliminate monomorphic VT, but there is a paucity of evidence that ablation directed toward arrhythmogenic substrate can decrease PMVT recurrence, as the mechanism of unstable reentry with continuous meandering eludes traditional mapping strategies.

Catheter ablation can be directed at triggers for PMVT/VF, akin to ablation of pulmonary veins for atrial fibrillation (2). Haïssaguerre *et al.* (3) first described ablation of idiopathic VF by mapping and targeting premature ventricular contraction (PVC) with short coupling interval

initiating the VF. The triggering PVCs were localized and eliminated at the right ventricular outflow tract (RVOT) in 4 of 16 patients and at the peripheral Purkinje system in the remaining 12 patients. This strategy of ablation of PMVT/VF with and without SHD has been increasingly reported with a high success rate ever since, and the triggering PVCs were identified in the Purkinje network, RVOT, the papillary muscle, right ventricular free wall and moderator band (4-9). So catheter ablation is recommended for recurrent episodes of VF when there is a triggering PVC with a consistent QRS morphology (10). Different from trigger ablation strategy, Nademanee *et al.* (11) mapped the substrate of right and left ventricles instead of the PVC triggers of VT/VF in 9 patients with Brugada syndrome. They detected unique abnormal low voltage, prolonged duration and fractionated late potentials exclusively localized in a cluster over the epicardium of the anterior aspect of the RVOT. Catheter ablation targeting the abnormal substrate area achieved 78% noninducibility of VT/VF and 89% freedom from recurrent VT/VF with normalization of Brugada ECG pattern during long-term follow-up. Modification of the Purkinje has been shown effective in suppression of VF without elimination of triggering PVCs suggesting Purkinje network may be critical substrate in some idiopathic VF cases (12,13). Zhang *et al.* (14) reported PVC initiation sites were associated with areas of J-waves and steep repolarization gradients where may be the substrate of early repolarization syndrome.

However, the relationship between myocardial scar and PMVT/VF is not well understood in patients with other forms SHD. In theory, if unstable forms of reentry responsible for PMVT originate within or anchor to regions of fibrosis, substrate modification strategies that are typically performed for monomorphic VT may be effective to reduce PMVT. In this context, Nakamura *et al.* reviewed 32 consecutive patients (13 idiopathic VF, 19 SHD) undergoing first radiofrequency catheter ablation for recurrent episodes of PMVT/VF to characterize the electrophysiological findings and ablation outcomes for patients with SHD other than Brugada syndrome compared to those with idiopathic VF (15).

Electroanatomic mapping was performed using the Carto system (Biosense Webster). Triggering PVCs were targeted with activation mapping or pacemapping. Sustained monomorphic ventricular tachycardia (SMVT) was also targeted for ablation if inducible by programmed electrical stimulation. A low-voltage area of myocardial scar was detected in 15 of 19 patients with SHD. SMVT associated with the scar was inducible and targeted in 8. Triggering PVCs were identified and ablated in 11 patients, among which endocardial scar was detected and also targeted in 7 (combination group) and only PVC ablation was performed in 4 (trigger group). Scar modification alone was performed in 8 patients who did not have triggering PVCs (substrate group). All idiopathic VF patients underwent PVC ablation only (idiopathic VF group). During a median of 540 days, 74% of SHD patients and 77% of idiopathic VF patients were free of recurrence, including 75% of trigger group, 86% of combination group, and 63% of substrate group.

This retrospective study provides hypothesis-generating insights and potentially implicates scar substrate with PMVT mechanisms. The major findings of this analysis were that: (I) low voltage scar was found commonly in recurrent PMVT/VF patients with SHD, with a prevalence of 79% in the study cohort; (II) SMVT can coexist with PMVT/VF in SHD which can be induced by programmed stimulation and was associated with low voltage scar, which indicates that they may share the same substrate; (III) triggering PVCs to guide ablation was not identifiable in about one-fourth of the cases. In this circumstance, substrate ablation targeting scar appeared to be a reasonable option with a similar success rate with trigger ablation strategy.

As the authors noted, since it was a nonrandomized, observational study and the sample size was small, further study of the substrate ablation for PMVT/VF should be

carried out. The study leaves some questions to us: (I) Are the prevalence and characteristics of the substrate similar across ischemic and nonischemic SHD? (II) What is the comparative efficacy of substrate modification *vs.* noninvasive medication therapy for recurrent PMVT/VF? (III) Given success rates in combination group was relatively higher than other groups in present study, is there any role combining substrate and trigger ablations? (IV) What are the optimal ablation strategies and lesions sets to target scar for PMVT?

The authors should be congratulated for this novel analysis that provides indirect evidence of a causal relationship between low voltage regions and PMVT mechanism. Traditionally, catheter ablation is not recommended in patients with PMVT and the present study suggests a paradigm shift to justify further investigation. In the SMASH VT study (16), where prophylactic ablation was shown to be superior to continued observation with ICD alone, 20% of patients randomized met inclusion criteria with VF as the presenting rhythm. Larger prospective studies to compare trigger ablation with and without substrate modification are necessary in this difficult population at high risk for mortality and frequent ICD shocks.

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Footnote

Conflicts of Interest: The authors have no conflicts of interest to declare.

Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

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