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EDITOR'S FORUM

Ablation of apparently idiopathic ventricular arrhythmias from atypical epicardial sites of origin: Take a look outside

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Catheter ablation is an effective treatment option for patients with ventricular arrhythmias (VAs) including premature ventricular complexes (PVCs) and ventricular tachycardia (VT). By definition, idiopathic VAs occur in patients with structurally normal hearts. The majority of idiopathic VAs tend to arise from a few typical sites (left and right ventricular outflow tracts, paraHisian region, mitral and tricuspid annulus, and papillary muscles) and can usually be successfully eliminated with endocardial ablation. However, idiopathic VAs may also originate from the epicardial aspect of the heart, usually involving the left ventricular (LV) summit and cardiac crux.

In this issue of Journal of Interventional Cardiac Electrophysiology, Ju et al. report a series of 9 patients with idiopathic VAs originating from uncommon epicardial sites of origin distinct from the LV summit and crux (5 posterolateral LV, 2 lateral LV, 1 anterior LV, 1 apical) which were eliminated with direct epicardial ablation [1]. Structural heart disease was excluded with echocardiography as well as endocardial and epicardial bipolar voltage mapping in all patients. Mean PVC burden at baseline was 37 ± 10% and 6 patients also had non-sustained VT. Four patients had prior failed endocardial PVC ablation. All patients first underwent endocardial mapping and the coronary venous system (CVS) was mapped in 7 patients prior to epicardial access. The earliest endocardial activation preceded the QRS onset by – 11 ± 4 ms and rS pattern on unipolar electrogram at the earliest endocardial sites was observed in all cases. Ablation from the endocardial side failed to eliminate PVCs; thus, percutaneous epicardial access was achieved for epicardial mapping and ablation. Epicardial mapping revealed the earliest activation time of – 25 ± 8 ms and QS pattern on unipolar electrogram at the earliest sites. Epicardial ablation successfully abolished PVCs in all patients with no recurrences over a median follow-up of 11 months.

The authors also compared patients with 9 demographically matched patients who underwent successful VA ablation at the adjacent endocardial sites (2 posterior LV, 2 posteromedial papillary muscle, 2 apical RV, 1 anterior RV, 3 lateral mitral annulus). The earliest endocardial activation time in the study group was significantly less early than in the control group (11 ± 4 ms VS, – 29 ± 8 ms, $p < 0.001$), whereas no significant difference was found between the earliest epicardial activation time of the study group and the earliest endocardial activation time in the control group (– 25 ± 8 ms vs – 29 ± 8 ms, $p = 0.389$).

Certain electrocardiographic patterns indicative of epicardial VAs have been previously established and validated, including maximum deflection index (MDI) > or = 0.55 [2], pseudo-delta wave > or = 34 ms, intrinsicoid deflection time > or = 85 ms, shortest RS complex > or = 121 ms [3]. The presence of Q-waves in the lateral (lead I) and inferior (II, III, and aVF) leads suggests epicardial origin from the epicardial aspect of the lateral and inferior LV, respectively [4]. The morphologies of PVCs in the epicardial ablation group in this series had "QS" pattern in leads II, III, and aVF for the PVCs originating from the inferior wall; in precordial leads for the PVCs originating from the anterior wall or apex; and in leads I and aVL for the PVCs originating from the lateral wall. Meanwhile, none of the VAs in the control group had a "QS" pattern on ECG. In this small series, the presence of pseudo-delta waves and MDI did not differ significantly between the study and control group.

Epicardial access for epicardial mapping and ablation is only rarely necessary in patients with idiopathic VA. The two most common sites of origin of idiopathic epicardial VAs are the left ventricular (LV) summit and cardiac crux and VAs from these sites can often be successfully targeted

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either from adjacent endocardial structures or through the CVS [5]. However, in some instances when ablation from the endocardium and CVS are ineffective, direct epicardial ablation may be necessary, especially in patients with significant symptoms or PVC-induced cardiomyopathy refractory to medical therapies.

Since percutaneous epicardial access, mapping, and ablation are associated with increased procedural risk, procedure duration, and hospital duration, our approach is to first attempt to eliminate VAs with endocardial and CVS ablation, and only proceed with percutaneous access for direct epicardial ablation if necessary. We have found the CVS to be very helpful to target epicardial VAs in many cases. While the anatomy of the ventricular branches of the CVS varies widely between patients, it can be helpful to determine whether the VA site of origin is likely to be epicardial or intramural. In patients with a suspected intramural or epicardial site of origin, occlusive coronary venography of the coronary sinus can delineate the course of the coronary veins and their branches. Coronary ventricular veins and their branches can then be cannulated using multielectrode microcatheters such as the EPStar (Baylis Medical; Toronto, CA) or Map-iT (Access Point Technologies EP; Rogers, MN) for activation, entrainment, and pace-mapping. Even smaller branches can be accessed with mapping wires (Vision Wire, Biotronik Inc., Germany). After identification of the true site of origin of VA, ablation can be performed either directly from within the coronary veins or via an anatomic approach from adjacent chambers guided by proximity to the site of origin.

Importantly, when atypical epicardial sites of origin are identified in patients with apparently idiopathic VAs, a more detailed workup should be considered to exclude the presence of occult nonischemic cardiomyopathy (NICM). One limitation of the study, as acknowledged by the authors, was that cardiac magnetic resonance imaging (MRI) was performed in only 3 patients prior to ablation. While endocardial and epicardial bipolar voltage mapping suggested no obvious abnormalities in any patients, cardiac MRI can be considered to exclude the presence of patchy mid-myocardial or layered delayed enhancement which could suggest early NICM or prior myocarditis. Furthermore, in younger patients with apparently idiopathic VAs originating from atypical sites, genetic testing should also be considered to exclude the presence of an underlying genetic cardiomyopathy (i.e., LMNA, PKP2, DSP, TTN) which might have not yet fully manifest with structural changes but may continue to progress over time. While implantable cardioverter-defibrillator was not indicated in any of the patients in this series since they were noninducible for sustained VA with programmed stimulation prior to ablation, recognition of the presence of underlying scar or genetic mutations may be helpful to identify patients who may benefit from continued long-term screening for the development of cardiomyopathy and sustained VAs.

We applaud the authors for sharing their interesting experience describing epicardial mapping and ablation in a series of patients with uncommon epicardial VA sites of origin. Detailed mapping of the CVS is very helpful and should be performed in all patients with suspected VA epicardial site origin. Further studies examining the value of cardiac MRI and genetic testing to identify occult NICM or genetic mutations in similar patients with apparently idiopathic VAs originating from atypical epicardial locations would be helpful.

Declarations

Conflict of interest The authors declare no competing interests.

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