EDITORIAL COMMENT

Ablation of the Epicardial Substrate in Ventricular Tachycardia Associated With Structural Heart Disease

Outside In or Inside Out?*

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Percutaneous epicardial access, introduced by Sosa et al. (1) in the mid-1990s, provided an important diagnostic and therapeutic approach to the recognition and management of scar-related epicardial ventricular tachycardia (VT). Since that time, the need for epicardial ablation has been reported in 10% to 15% of patients with ischemic cardiomyopathy (ICM) (2–4), 25% to 50% of patients with nonischemic cardiomyopathy (NICM) (4–8), and 30% to 40% of patients with arrhythmogenic right ventricular cardiomyopathy (ARVC) (4,9–12). Controversy persists with regard to the timing of epicardial intervention (initial procedure or only after unsuccessful endocardial approaches) and the extent of epicardial ablation required (focused regional ablation targeting specific VT morphologies or elimination of all epicardial late or abnormal potentials [13,14]). These con-

troversies persist for several reasons. Percutaneous epicardial interventions are associated with a 4% to 7% risk of major complications (4,15), including subdiaphragmatic or intrapericardial bleeding, coronary artery injury, and phrenic nerve damage. Percutaneous epicardial access can be technically challenging, particularly in patients with prior cardiac surgery or pericarditis. Finally, the presence of fat more than a few millimeters thick reduces the depth and volume of ablation lesions, thereby reducing the effectiveness of epicardial ablation (16).

The study of Komatsu et al. (17), published in this issue of the *Journal*, provides important new information and sheds additional light on these issues. The goal of the study was to determine the impact of endocardial ablation alone on the epicardial substrate for VT. In 51 patients with scar-related VT, high-density endocardial and epicardial mapping was performed with a 3-dimensional navigation system. In 46 patients, epicardial local abnormal ventricular activities (LAVA) were identified and comprised the subjects for this report (18 ICM, 13 NICM, 15 ARVC). Only a minority of patients (9%) had undergone a previous ablation procedure. Radiofrequency (RF) ablation was performed with an open irrigated catheter. The primary endpoint was elimination of all LAVA. By study design, the authors attempted to eliminate all epicardial LAVA by endocardial ablation first, with monitoring of the overlying epicardial surface by a multielectrode array.

The most important finding of the study was the relative effectiveness of endocardial ablation in eliminating epicardial LAVA and terminating epicardial VT. At 173 sites, endocardial ablation of epicardial LAVA was attempted (46% of sites had coexisting endocardial LAVA). Elimination of the appositional epicardial LAVA was achieved in 28%. Elimination of epicardial LAVA was more likely at endocardial sites with lower bipolar and unipolar voltage and where endocardial LAVA were also present. The type of underlying structural heart disease played an important role; successful endocardial ablation of epicardial LAVA occurred at 40% of sites in patients with ARVC, 28% with ICM, and 8% with NICM. From a global perspective, complete elimination of epicardial LAVA by endocardial ablation alone occurred in 13% of patients with ARVC, 22% with ICM, and none with NICM. At least partial elimination of epicardial LAVA occurred in 73% of patients with ARVC, 83% with ICM, and 13% with NICM. The outcome of conventional activation and entrainment mapping during stable VT revealed a similar pattern. In 23 of 39 mapped VTs, the critical isthmus was found to be on the epicardium. Of these epicardial VTs, 5 (22%) terminated during initial endocardial ablation (33% ARVC, 28% ICM, and 0 NICM).

The authors provide further insight into the potential mechanism for successful endocardial ablation of the epicardial substrate by examining data from multi-detector computed tomography scans obtained pre-procedurally in 21 patients (12 ICM, 9 IDCM). The scans were subsequently extracted and integrated into the electroanatomical map after the procedure. A region of wall thinning (<5 mm thickness) was present in all patients except 1 with NICM. Endocardial ablation was successful in eliminating epicardial LAVA at 32% of sites associated with wall thinning but at none of the sites without thinning. Of importance, epicardial LAVA were more commonly found at sites with wall thinning in ICM (88%) compared with NICM (46%). In the absence of CT data, the strongest predictor of patient response to endocardial ablation was the presence of all

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epicardial LAVA within the endocardial unipolar low-voltage zone. The less frequent association of epicardial substrate with wall thinning in patients with NICM might help account for the very poor response of NICM compared with ICM to endocardial ablation alone. Together with other indirect evidence (larger endocardial and epicardial scar areas), the study indicates that endocardial ablation of epicardial substrate (LAVA, critical isthmuses) is most likely to occur in areas of large, thinned transmural scars.

Production of transmural lesions in normal ventricular myocardium by endocardial ablation, even with irrigated RF, is rare (18). Thus, it is not surprising that scar-associated wall thinning enhances the probability that subepicardial and intramural arrhythmogenic substrate will be incorporated into lesion. In this study, RF energy was applied at a median power of 35 W for a median duration of approximately 1 min. However, lesion depth and volume are greater with higher powers or longer durations than those employed in this study, although the risk of steam pops and thrombus formation also increases (18,19). Nonetheless, it is possible that higher power or longer duration applications might have further increased the effectiveness of endocardial RF at selected sites. Endocardial ablation might also eliminate epicardial LAVA without transmural lesions when the isolated epicardial fibers producing LAVA are predominantly activated transmurally rather than from the surrounding subepicardium.

How do we best incorporate the lessons from this study into our current practice of scar-related VT ablation? In patients where concomitant endocardial and epicardial mapping is undertaken, the message is clear. Initial endo-cardial ablation might eliminate some epicardial LAVA and critical VT circuit sites, reducing the extent and in some cases the necessity of epicardial ablation. This approach might be particularly useful where effective epicardial abla-tion is limited by the proximity to major coronary arteries, the phrenic nerve, or thick layers of epicardial fat. In the absence of integrated pre-procedural images at the time of ablation, sites where endocardial ablation is most effective can be predicted by low unipolar voltage and the presence of endocardial LAVA.

From a broader perspective, data from this study help to reconcile several disparate clinical observations with regard to epicardial VT ablation. Despite reports that epicardial LAVA are common in patients with ICM when they are specifically looked for (up to 90% [13,20]), the proportion of patients that require epicardial ablation for long-term clinical success seems considerably lower. Although the arrhythmo-genic substrate in ICM is more extensive subendocardially (20), elimination of the overlying epicardial substrate by endocardial ablation through thinned scar might also contribute to the less frequent need for epicardial ablation in this population. These observations, in conjunction with the challenges of epicardial access associated with prior surgical revascularization in a substantial proportion of these patients, provide a rationale for deferring epicardial intervention in patients with ICM to those with clinical recurrence of VT

after technically adequate initial endocardial ablation. By contrast, the arrhythmogenic substrate of NICM is more commonly subepicardial or intramural, often with little extension to the subendocardium (5,7,8,20). Furthermore, as Komatsu et al. (17) demonstrate, the extent of thinned transmural scar is considerably less in this population. Endocardial ablation targeting epicardial substrate (LAVA and/or critical isthmuses) was almost uniformly ineffective. These data indicate that a more aggressive strategy for initial epicardial access and intervention in NICM remains clinically appropriate.

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