

Coronary arterial injury during right ventricular outflow tract ablation: Know your neighbors

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Abstract

Left anterior descending (LAD) coronary arterial injury is an underappreciated and rare consequence of ablation in the right ventricular outflow tract (RVOT). The authors present five cases of acute or subacute LAD injury after RVOT ablation. Most patients had fairly extensive ablation and two had coincident cardiac perforation. The patients reported also had a strikingly similar ECG morphology of their spontaneous ventricular arrhythmias. The authors' report serves an important cautionary tale regarding ablation of intramural septal VAs.

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The right ventricular outflow tract (RVOT) is the most common location of idiopathic ventricular arrhythmias (VA). Due to low procedural risk and high success rates, catheter ablation of VA originating from the OT has been routinely performed for decades. From an attitudinal perspective, the RVOT is oriented anteriorly and leftward relative to the left ventricular (LV) OT, and is bounded superiorly by the pulmonic valve and inferiorly by the RV inflow region. The topography of the RVOT below the pulmonic valve is commonly conceptualized as opposing “septal” and “free wall” surfaces; this schema is useful both in predicting site of VA origin from 12-lead ECG and in performing catheter-based mapping. The junction of these two surfaces is the most superior and leftward aspect of the heart, and arrhythmias from this region typically have a left bundle branch block configuration, a rightward frontal plane axis, and tall inferior R waves on ECG.

The proximity of coronary arteries to the aortic cusps and LVOT is well understood, therefore imaging of the coronary arteries is frequently performed during catheter ablation in the LVOT to prevent collateral injury. However, the three-dimensional relationship between the coronary arteries and the RVOT, and consequently the risk of potential collateral damage to the coronary arteries when ablating in the RVOT, is often overlooked.¹

The study by Dilling-Boer et al² in this issue of the *Journal* illustrates a rare and underappreciated hazard associated with ablation in the RVOT. The authors report acute or subacute injury to the left anterior descending (LAD) coronary artery in five patients after ablation in the RVOT. All patients required coronary intervention, four with acute vessel occlusion. This manifested as acute ST segment elevation in the precordial leads during catheter ablation in three of the patients, and as nonspecific ST segment flattening and depression in one. Review of the coronary angiogram demonstrated occlusion of the LAD segment between the first and second diagonal branches in four patients and the LAD segment between the second and third diagonal branches in one patient. Follow-up ambulatory monitoring data is reported for three of the patients, all of whom had VA suppression at last follow-up.

The manuscript is a welcome addition to the existing literature concerning OT VA ablation and the authors are congratulated for their important effort. The manuscript figures combine multimodality imaging platforms to highlight the proximity of the RVOT to the intraseptal course of the LAD and the anterior interventricular vein. There are several important observations from the report. First, there is remarkable similarity in 12-lead ECG VA morphology between the five reported patients: left bundle branch block pattern with late (V4) precordial transition and a right inferior frontal plane axis. These VAs have a tall R wave amplitude in the inferior leads with lead III greater in magnitude compared to lead II. The authors' Figures 5B and C illustrate well the cranial and relatively leftward position of this region compared to the interventricular septum. Also note the lack of initial r wave in V1, coupled with a relatively slurred QRS onset in these patients. Many of these ECG features are reminiscent of arrhythmias successfully ablated in the anterior interventricular or intraseptal veins.^{3,4}

Consider next the extent of ablation performed in these patients. We have relatively complete ablation data from only three patients, and the total radiofrequency ablation duration was 40 and 26 minutes in patients 1 and 3 respectively. Patients 2 and 4 had VA suppression coincident with a steam pop and pericardial tamponade respectively, thus the relatively short total duration of ablation likely underestimates the extent of the lesions applied. Only one patient underwent mapping of the LV or coronary venous system, thus it is reasonable to speculate whether there may have been alternative safer or more proximate sites for ablation compared to the RVOT. Neither pacemapping nor activation mapping data are included in the report to inform the reader in this regard.

In considering the ECG morphologic data and the extent of ablation performed, it is reasonable to conclude that most of the patients reported in this manuscript had intramural origin for their VAs. It is well known that VAs from this region can be approached from the RVOT, LVOT, or coronary venous system. Extensive mapping is required to locate a site to ablate that provides the best balance between safety and efficacy. More extensive ablation is likely to create collateral damage, and the authors' report illustrates this concept well. In select cases, one might consider novel techniques such as coronary venous ethanol ablation rather than more aggressive endocardial strategies like simultaneous unipolar or bipolar ablation.⁵ It is also reasonable to conclude that LAD injury is quite rare in patients with idiopathic VAs originating from the endocardial aspect of the RVOT in whom early suppression during ablation is achieved. Nonetheless, the manuscript underscores a distinct ECG signature for VAs originating in proximity to the mid LAD, allowing the operator to optimize patient consent and procedural workflow. Specifically, the use of preoperative tomographic imaging or intraprocedural coronary angiography may be appropriate in certain cases, particularly those with underlying cardiomyopathic processes or failed prior ablation procedures. Such images can be imported and co-registered with the electroanatomic mapping system to provide greater anatomical context. When ablating in this region, the operator should also be mindful of any ST segment deviation that may suggest coronary injury. These changes can be more difficult to appreciate on electrophysiology recording systems

due to increased sweep speed. Particular vigilance is needed in patients under general anesthesia in whom the presence of symptoms cannot be assessed.

In summary, the study by Dilling-Boer et al highlights the anatomical relationship of the RVOT with the intraseptal course of the LAD with attendant risk of vessel occlusion during ablation. A thorough understanding of spatial anatomy of this region and ablation biophysics will help the practicing electrophysiologist both to anticipate possible complications specific to this region and to tailor ablation strategies to mitigate procedural risk.

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