CASE REPORT

Right ventricular outflow tract ablation associated with anomalous origin of coronary artery: A "double-edged sword"

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Introduction

Catheter ablation is a curative and effective therapeutic option for idiopathic and symptomatic right ventricular outflow tract (RVOT) ventricular tachycardia (VT).[1] However, cases of coronary artery injury during catheter ablation have been reported.[2–4] Anomalous aortic origin of the coronary artery (AAOCA) is a rare condition, usually associated with an abnormal trajectory of the involved vessels, being a "double-edged sword," depending on whether the coronary artery is distant or close to the site of origin of the VT. Therefore, in these cases it is essential to determine the relationship between these structures to avoid iatrogenic damage to the epicardial vessels. There is only one reported case of successful ablation of RVOT VT in a patient with an anomalous aortic origin of right coronary artery (R-AAOCA), in which the artery ran very close to the ablation site (risk factor for coronary artery injury). We present this exceptional case of L-AAOCA that resulted as a protective factor during successful radiofrequency cardiac ablation (RFCA) of symptomatic RVOT VT.

Case report

A 57-year-old woman with a history of obesity, hypertension, dyslipidemia and frequent ventricular extrasystoles, came to the outpatient clinic for repeated episodes of rapid palpitations, dizziness, and syncope on up to 2 occasions. The patient reported that the loss of consciousness was abrupt and transient, with rapid recovery of alertness occurring while she was engaged in low-intensity physical activity, and preceded by rapid palpitations. She denied chest pain during these episodes. On physical examination, vital signs were preserved; on cardiac auscultation, the heart sounds were of good intensity, irregular and a low-intensity diastolic murmur was perceived. The rest of the physical examination was normal. The electrocardiogram showed an episode of nonsustained ventricular tachycardia whose probable origin was from the septal aspect of the RVOT (Figure 1). A 24-hour Holter monitor showed the presence of a high burden (15%) of monomorphic ventricular extrasystoles with left bundle branch block morphology.

Based on cardiovascular risk factors, complementary examinations were requested, to rule out an ischemic origin of the referred symptoms and VT. Coronary angiography revealed an anomalous origin of the LMCA from the right coronary sinus and absence of significant atherosclerotic lesions (Figure 2A, Video 1). This coronary anomaly is associated with a risk of sudden cardiac death (SCD), especially if it has high-risk anatomical features (see Discussion) or is associated with myocardial ischemia. For this reason, computed tomography (CT) coronary angiogram, myocardial perfusion test and tissue characterization by cardiac magnetic resonance were performed, demonstrating the presence of low-risk anatomical features (Figure 2B–2D, Video 2–4), absence of ischemia (Figure 2E) and fibrotic foci (Figure 2F), which would justify an arrhythmogenic focus for VT. Likewise, cardiac magnetic resonance demonstrated discrete decrease in LV systolic function (LV ejection fraction 50%), mild aortic insufficiency and ruled out other causes of structural cardiomyopathy (Video 5). Ischemic and hypertensive etiology were ruled out as causes of reduced ejection fraction, based on coronary angiography (which showed no significant

KEYWORDS Ventricular tachycardia; Right ventricle outflow tract; AAOCA; Electroanatomic mapping; Radiofrequency ablation
(Heart Rhythm Case Reports 2024;■:1–5)

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https://doi.org/10.1016/j.hrcr.2024.07.021

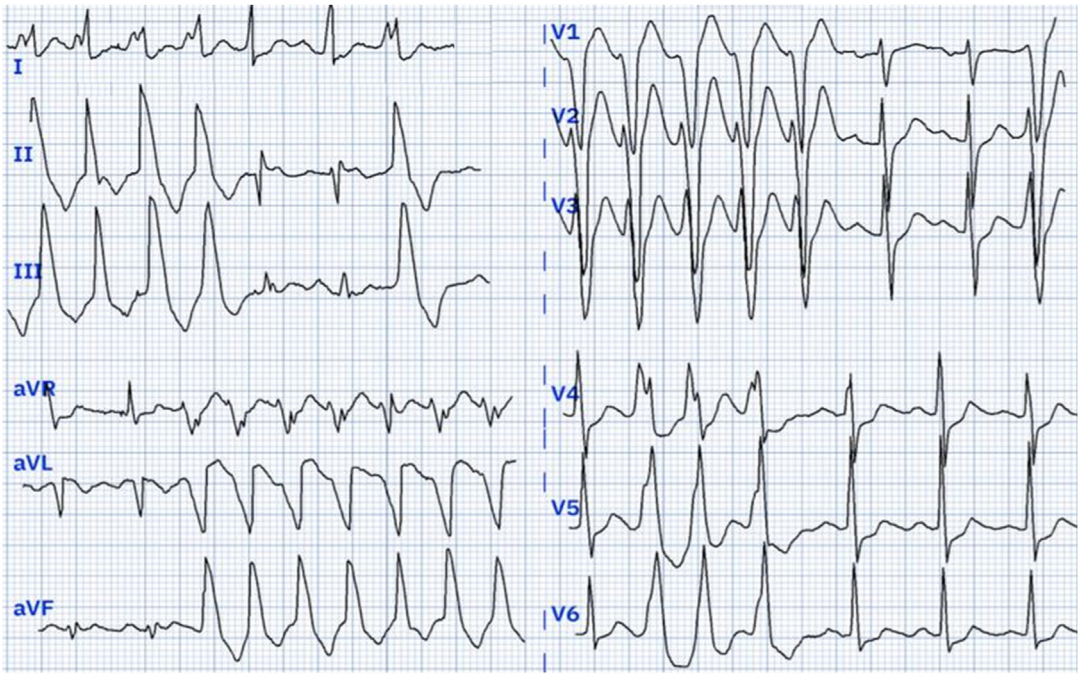

Figure 1 Septal RVOT-NSVT. QRS complexes with LBBB morphology, inferior axis, late transition (V_4), V_2 transition ratio 0.35, V_2S/V_3R 3.72. The absence of notching in inferior leads suggested an origin from the septal aspect of the RVOT. LBBB = left bundle branch block; NSVT = non-sustained ventricular tachycardia; RVOT = right ventricle outflow tract.

lesions), magnetic resonance imaging (MRI; absence of late gadolinium enhancement) and adequate ambulatory blood pressure control with antihypertensive treatment. Likewise, aortic regurgitation was mild, without left ventricular remodeling, which did not justify the reduction in ejection fraction. In the context of a high burden of ventricular extrasystoles, a presumptive diagnosis of cardiomyopathy associated with ventricular extrasystoles was made.

After clinical discussion, the HeartTeam at that time (year 2018) decided to implant a single-chamber implantable cardioverter defibrillator (ICD; Medtronic Evera MRI™ SureScan®) as secondary prevention in the context of syncope of unknown cause and systolic dysfunction (Class of recommendation IIa, level of evidence C).[5] The patient was discharged under treatment with amiodarone (200 mg daily), atorvastatin (40 mg daily), and enalapril (10 mg twice a day).

Subsequently, by the end of 2022, our patient discontinued amiodarone on her own. At the beginning of 2023, during a festive activity associated with alcohol consumption, she again presented with rapid palpitations and repeated ICD shock sensations. She was admitted hemodynamically stable to the emergency department, where ICD interrogation was performed, showing up to 20 episodes of sustained VT, many of them managed with anti-tachycardia pacing and appropriate shock discharges. The diagnosis of electrical storm was made, and deep sedation and administration of amiodarone (150 mg over first 10 minutes, followed by continuous infusion at 36 mg/h) were selected, which led to the cessation of VT and left the patient with frequent ventricular extrasystoles. Finally, after discussing the case, HeartTeam decided to perform an electrophysiological study plus VT ablation by 3-dimensional (3D) mapping. The patient was admitted to the electrophysiology laboratory in sinus rhythm with frequent ectopy from the RVOT. During the electrophysiological study, VT was induced with isoproterenol, starting at 1 μg/min and gradually increasing up to 3 μg/min, triggering VT that had identical morphology to that previously objectified in 2018 (Figure 3A, Video 6). Likewise, the 3D electroanatomic map identified the earliest activation zone at the level of the posteroseptal aspect of the RVOT (25 ms pre-QRS with a negative deflection [QS morphology] in the unipolar electrogram; Figure 3B, Video 7). Left ventricle outflow tract and sinuses of Valsalva were also mapped, finding a precocity of 11 ms with an rS morphology in the unipolar electrogram (Video 8), so it was decided to ablate the earliest activation zone of the RVOT. CT coronary angiogram images were integrated with a 3D electroanatomic map (Figure 3C and 3D; Video 6 and 7) which allowed a better understanding of the anatomic landmarks between the likely site of origin of VT and the coronary vessels. With the certainty that the LMCA and LAD were more than 5 mm apart, ablation points were applied at 30 W (ThermoCool SmartTouch D-F; Biosense Webster) (Figure 3E, Video 9), with an ablation index of 450 and subsequent disappearance of VT. Finally, the VT induction protocol was performed with isoproterenol, with 400/290/260/250 extrastimulus train, with no evidence of ventricular extrasystoles.

At 1 year of follow-up, the patient remains asymptomatic with NYHA class I. The latest ICD interrogation demonstrated absence of VT or VF episodes (supplementary material). Moreover, after successful ablation, there was

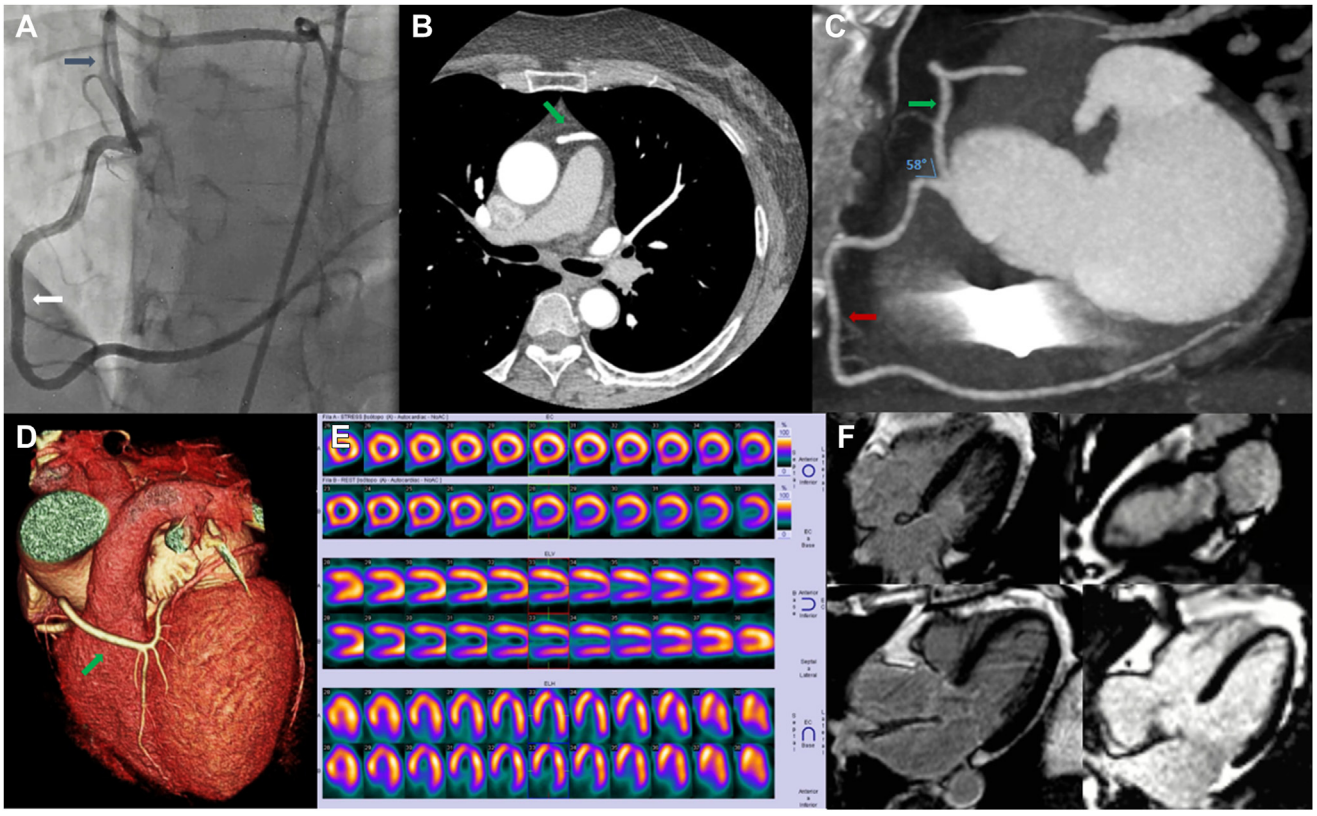

Figure 2 **A:** Coronary angiography demonstrated the common origin of the LMCA (blue arrow) and the RCA (white arrow) from the right coronary sinus. **B:** Contrasted chest CT. The prepulmonary course of the LMCA is seen (green arrow). **C:** CT coronary angiogram. Coronal view of the heart showing the anomalous origin of the LMCA (green arrow) from the right coronary sinus. The RCA (red arrow) also originates from the same coronary sinus. The 58° birth angle, the oval morphology of the ostium and the absence of an intramural course can be seen. **D:** Cardiac volumetric reconstruction. Demonstrates the absence of interarterial course (between the aorta and pulmonary artery) of the LMCA. **E:** Myocardial perfusion test negative for ischemia. **F:** Cardiac magnetic resonance imaging. Absence of late gadolinium enhancement in 4-, 2- , 3- and 5-chamber views. CT = computed tomography; LMCA = left main coronary artery; RCA = right coronary artery.

significant improvement in LVEF (50%–62%), confirming the diagnosis of cardiomyopathy associated with extrasystoles.

Discussion

Coronary arteries (CA) typically arise from the right and left sinuses of Valsalva, presenting a course and distribution of collateral branches with significant variability, such that variants of normality have been identified. On the other hand, coronary artery anomalies are those with a prevalence <1% of the general population[6] and can be classified as anomalies of origin, course, or termination. Within anomalies of origin, AAOCA is characterized by anomalous origin of the CA from the opposite sinus of Valsalva (R-AAOCA, if the right coronary artery arises from the left sinus of Valsalva; L-AAOCA, if the left coronary trunk arises from the right sinus of Valsalva). The estimated prevalence for R-AAOCA and L-AAOCA is 0.33% and 0.12%, respectively.[7] Historical reports point to an association between AAOCA and SCD, especially L-AAOCA (Figure 2), particularly in the presence of high-risk features (cleft ostium, acute birth angle (<45°), intramural and/or interarterial course).[8] Our patient had L-AAOCA with an oval ostium (7 × 6 mm), 58° birth angle, absence of intramural course and a pre-pulmonary course, all of which conferred low anatomic risk and resulted as a protective factor during definitive management of VT.

Several theories have been put forward regarding the mechanisms for SCD. One theory is the development of severe acute ischemia, especially during strenuous exercise, because of fixed and dynamic factors that would decrease coronary blood flow.[9] Another hypothesis is the presence of an arrhythmogenic substrate from myocardial scars, which would trigger malignant arrhythmias.[10] However, the functional tests performed in our patient demonstrated the absence of ischemia during pharmacological stress, and tissue characterization ruled out the presence of foci of myocardial fibrosis. For this reason, electrophysiological study and ablation of the VT was decided.

Idiopathic ventricular arrhythmias (VAs) include premature ventricular depolarizations, nonsustained ventricular tachycardia, and, rarely, sustained VT. Triggered activity or abnormal automaticity is usually the mechanism of a VA.[11] A VA tends to originate frequently from the ventricular outflow tract, either above or below the semilunar valves,

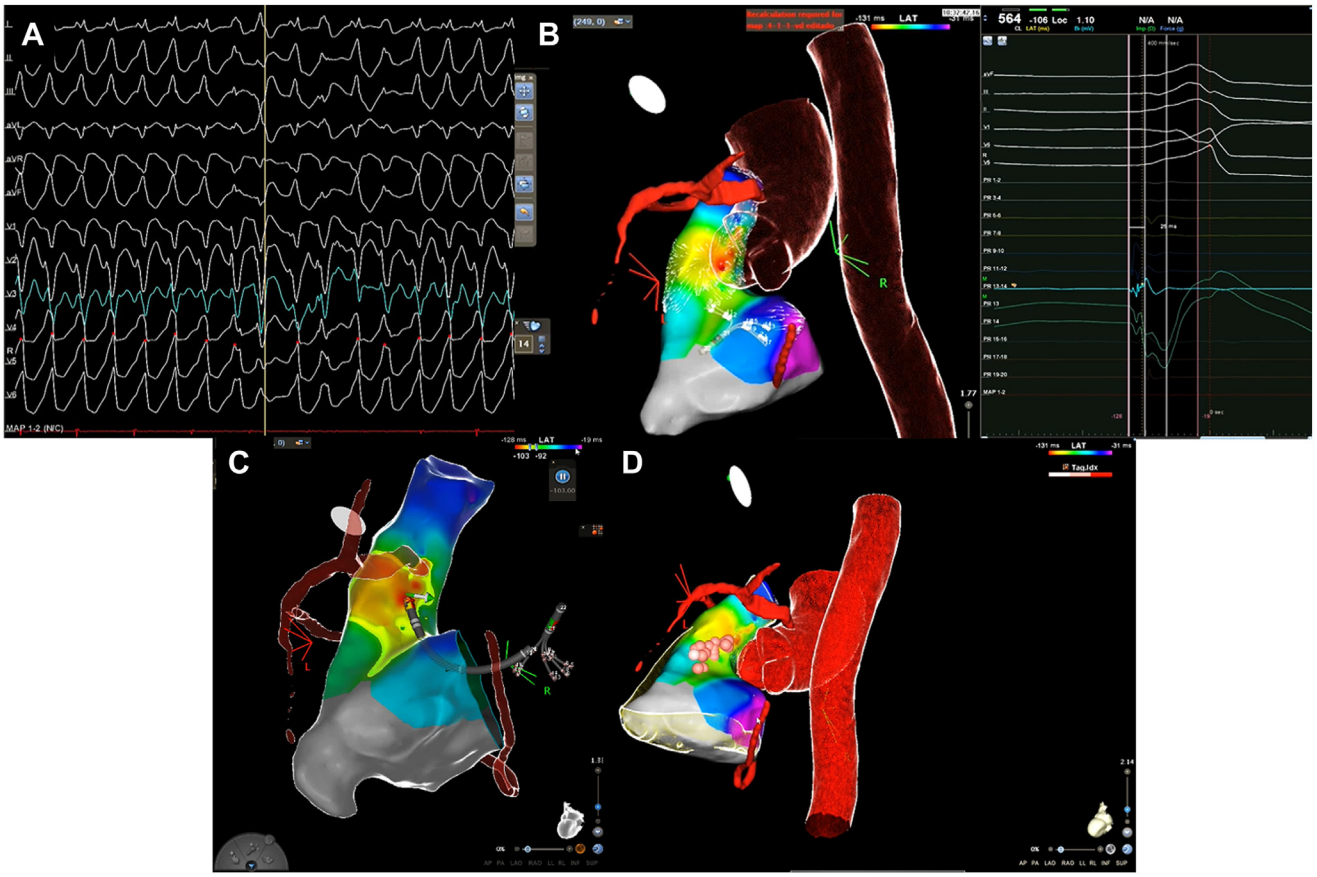

Figure 3 **A:** VT induction with morphology identical to that objectified in 2018. **B:** Activation map showing the earliest activation at the posteroseptal aspect of the RVOT (25 ms pre-QRS with a negative deflection [QS morphology] in the unipolar electrogram). **B, C:** 3D electroanatomic map integrated to the CT coronary angiogram. Notice the relationship between the ablation catheter and the coronary trajectory. **D:** Ablation points (30W, ablation index of 450) with subsequent disappearance of ventricular extrasystoles. 3D = 3-dimensional; CT = computed tomography; RVOT = right ventricle outflow tract; VT = ventricular tachycardia.

and may have a close anatomic relationship to the epicardial vessels. Meticulous analysis of VA morphology on the 12-lead electrocardiogram is a cornerstone in pre-procedural planning. Thus, our patient had a ventricular tachycardia whose probable origin was from the posteroseptal wall of the RV outflow tract (LBBB morphology, inferior axis, late precordial transition, V_2 precordial transition ratio < 0.6, V_2S/V_3R ratio > 1.5, rightward and positive QRS morphology in lead I).

In patients with usual coronary anatomy, the left anterior descending (LAD) artery usually runs in proximity to the septal region of the RV outflow tract, and there are several case reports regarding LAD injury during RFCA.[2–4] Thus, AAOCA may prove to be a "double-edged sword" because it may confer an increased or reduced risk at the time of RFCA VT RVOT,[12] by approaching or deviating, respectively, from the usual path of the epicardial vessels. For better visualization of the anatomic relationship of the L-AAOCA and the earliest site of activation, we integrated the CT coronary angiogram with the electroanatomic 3D map by using the CARTO Merge feature (Biosense Webster, Diamond Bar, California). This corroborated the abnormal trajectory of the left main coronary artery, running in front of the pulmonary artery (pre-pulmonary trajectory) and far away from the earliest activation site, which allowed a safer and more complete RFCA.

In addition, intracardiac echocardiography (ICE) is a major advance in cardiac imaging and is a useful tool in various interventional and electrophysiologic procedures. Among its multiple uses, it reduces procedure time, reduces radiation exposure time, prevents esophageal damage associated with transesophageal echocardiography, optimizes results, and reduces the rate of complications through better visualization of cardiac structures. Within the electrophysiologic procedures, ICE plays a leading role during the ablation of ventricular arrhythmias, allowing the identification and delimitation of an arrhythmogenic substrate (myocardial scars), the continuous assessment of the catheter-tissue contact and the incorporation of anatomic information to the electroanatomic map by means of the CARTOSOUND software. Likewise, ICE is particularly useful for mapping anatomic structures that are critical to preserve such as parahisian tissues and coronary arteries. Thanks to its greater depth of penetration, its greater maneuverability, and its ability to acquire Doppler flow and color images, phased-array ICE allows localization of the ostium and the proximal

course of the epicardial arteries, thus ensuring that the radiofrequency is applied at a safe distance (>5 mm) and without potentially fatal complications. Despite these advantages, its cost, the need for an operator with expertise in its use, and its limited availability in all hospital centers limit its routine use. ICE was not available in our center during the study period.

Although it is unusual to determine the coronary anatomy before electrophysiologic procedures, consideration of its origin, trajectory, and termination is important to obtain optimal results and reduce life-threatening complications.

Conclusion

In cases of symptomatic idiopathic VT, radiofrequency cardiac ablation is curative, safe, and effective. Therefore, the determination of the probable site of origin of VT, by means of the 12-lead electrocardiogram, is a cornerstone for planning the procedure. AAOCA is a rare condition and, in its presence, it is essential to determine the coronary pathway and its anatomic relationship with the ablation site. The integration of the CT coronary angiogram in the 3D electroanatomic map allows a better understanding of the anatomic repairs and thus avoids iatrogenic injury of the anomalous coronary vessels. Likewise, AAOCA is associated with SCD, especially if associated with high-risk anatomic features or ischemia. It is essential to rule out these conditions, as management changes significantly depending on the etiology of the ventricular arrhythmia. This is the first reported case of successful RVOT VT ablation in a patient with L-AAOCA, in which the trajectory of the anomalous left main coronary artery resulted as a protective factor for radiofrequency cardiac ablation.

Appendix

Supplementary Data

Supplementary data associated with this article can be found in the online version at https://doi.org/10.1016/j.hrcr.2024.07.021.05.005.

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