Coronary artery vasospasm during catheter cryoablation of left ventricular summit nonsustained ventricular tachycardia

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Introduction

The left ventricular summit (LVS) is a major source of idiopathic ventricular arrhythmias (VAs).¹,² McAlpine described this portion of the epicardium of the left ventricular outflow tract as a triangular region bounded by the left anterior descending coronary artery and left circumflex coronary artery (Cx), which lies superior to the aortic portion of the left ventricle (LV) ostium.³ The LVS is bisected by the great cardiac vein (GCV) into an area lateral to this structure, which is accessible to epicardial catheter ablation, and a superior region that is inaccessible to catheter ablation because of the close proximity of the coronary arteries and the thick layer of epicardial fat that overlies the proximal portion of these vessels (Figure 1).

Epicardial catheter ablation of LV summit VAs has been associated with poor results, mainly owing to a high prevalence of VAs originating at the inaccessible area.⁴ We present the case of a nonsustained ventricular tachycardia originating at the inaccessible area of the LVS, which was successfully ablated through the coronary sinus (CS) using focal cryoablation. The Cx suffered vasospasm during cryoenergy delivery, without coronary artery injury. Cryoenergy delivery was continued, without ST segment alterations or chest angina, achieving complete elimination of the clinical VA.

Case report

A 58-year-old woman without medical history of structural heart disease was referred to our institution for frequent, symptomatic monomorphic premature ventricular contractions, with a Holter burden of 36% and nonsustained ventricular tachycardia runs. The clinical arrhythmia was refractory to multiple antiarrhythmic drug schemes: 200 mg/day flecainide, 450 mg/day propafenone, and 240 mg/day sotalol. Cardiac echocardiography showed normal diameters and preserved LV wall motion without any significant valve dysfunction, and the ejection fraction was calculated at 64% using the Simpson method. The patient underwent coronary angiography, which showed no significant coronary artery stenosis.

The arrhythmia exhibited an rS pattern in V₁, early R-wave transition at V₂, and QS pattern in lead I, with QRS duration of 175 ms. The QRS axis was inferiorly directed. The Q-wave ratio of aVL/aVR was 2. Pseudo-delta waves (PdW 78 ms) were observed in all electrocardiogram (ECG) leads. An initial q wave was present in lead I, q waves were absent in inferior leads, and the maximum deflection index was 0.6. All criteria were consistent with an epicardial origin.⁵

Catheter ablation was performed under conscious sedation. A perfect pacemap (24 points)⁶ was obtained while pacing from the distal aspect of the GCV, toward the anterior interventricular vein (AIV), where the earliest activation preceded the QRS for 46 ms (ventricular electrogram-QRS interval).

A coronary angiography revealed the close proximity of the ablation electrode placed at the earliest activation site and the Cx artery (Figure 2). This rendered radiofrequency (RF) ablation impossible, given the high risk of coronary artery injury. For this reason, RF energy was delivered at the endocardial aspect of the LV summit (Figure 3) using a 4-mm irrigated-tip ablation catheter. Endocardial ablation failed to eliminate the clinical arrhythmia.

A focal 6-mm-tip cryoablation catheter (Freezor Xtra, Medtronic, Inc, Minneapolis, MN) was introduced inside the CS, at the distal aspect of the GCV over the ablation site.

KEYWORDS Ventricular arrhythmias; Ventricular tachycardia; Catheter ablation; Cryoablation; Left ventricular summit

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Cryoenergy was delivered reaching \(-82^\circ\)C. Simultaneous left coronary angiography was performed every 20 seconds to ensure the location of the ablation catheter relative to the left coronary arteries and to minimize the risk of thermal injury. A vasospasm of the Cx artery was observed during cryoablation (Figure 3), which did not cause an ST-segment elevation or chest angina. Complete reversal of this phenomenon was observed with administration of nitroglycerine. For this reason a 240-second dose was completed and 2 freeze-thaw-freeze cycles were performed. The VA was abolished after ablation and remained noninducible. A coronary angiogram was repeated, showing no Cx artery stenosis or injury.

During the next 3 months of follow-up, Holter monitoring revealed no premature ventricular contractions (0% burden) and the patient remained asymptomatic, without antiarrhythmic drugs.

**Discussion**

The prevalence of LV summit VA is much higher within the GCV and AIV than on the epicardial surface of either side of these veins. LVS VAs with an origin lateral to the GCV are amenable to ablation using an intrapericardial approach. Sites of origin superior to this structure are located in the inaccessible area bounded by the left coronary arteries, GCV, and AIV. In this region, catheter ablation is unlikely to be successful because of a thick layer of epicardial fat overlying the proximal coronary arteries and may be potentially hazardous to these vessels.

Several ECG parameters may be helpful for predicting the site of origin. When LVS VAs exhibit a right bundle branch block pattern and transition zone earlier than lead V1, aVL/aVR amplitude ratio of 1.1, and S waves in V5 or V6, those VAs are likely to be cured by catheter ablation within the GCV, the AIV, or the accessible area. Adjacent sites, such as the endocardial left ventricular/right ventricular outflow tract, coronary cusp region, or coronary venous system, are successful in eliminating the arrhythmias. The accessible area of the LVS is located inferiorly and laterally to the GCV, where the anatomic separation between the left anterior descending coronary artery and Cx arteries is maximal and there is lower chance of having a thick layer of epicardial fat. Indeed, the 3 ECG features found to be more prevalent in successful vs unsuccessful cases (Q-wave ratio aVL/aVR \(> 1.85\), R/S ratio in V1 \(> 2\), and lack of Q wave in V1) and

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**Figure 1** Brocq-Mouchet triangle composed by the intersection of the great cardiac vein (GCV), the circumflex artery (LCX), and the left anterior descending coronary artery (LAD). These structures are located between conus arteriosus and left auricle, on the left side of anterior surface of the heart. AIV = anterior interventricular vein.

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**Figure 2** A: Endocardial activation map of the same anatomical region. B: Activation map of the coronary sinus, showing the earliest activation at the LV summit epicardium, where the great cardiac vein receives the anterior interventricular vein (AIV). C: Clinical premature ventricular contraction with the earliest activity recorded at the AIV with a 46 ms ventricular electrogram-QRS. D: Pacemapping showing a 12/12 match and 24-point score.
reflect a more lateral site of origin of the VAs (distant from the midline and the apex of the LVS triangle). Accordingly, more patients with successful ablations have a QS pattern in lead I, which again points to a more lateral (and epicardial) origin of the VAs.

When LVS VAs exhibit a III/II amplitude ratio of 1.25 and an aVL/aVR amplitude ratio of 1.75, those VAs are likely to require a pericardial approach for ablation.

Ventricular prepotentials are often recorded at the successful ablation sites. These ventricular prepotentials may be very helpful for guiding the site of successful ablation.

Successful ablation of VAs from the inaccessible LVS area can be achieved from the left coronary cusp in 56% of cases. Similarily, Yamada et al. first described the anatomy and electrophysiological characteristics of arrhythmias arising from the LVS in a series of 27 patients, reported successful ablation from the GCV–AIV region in >70% of cases.

Cryoablation has been reported as a safe and effective alternative for catheter ablation of ventricular arrhythmias. Cryothermal safety profile is attributed to the mechanism of tissue destruction. Histology of chronic lesions shows well-demarcated lesions with minimal tissue disruption and preserved underlying architecture. The use of RF energy in close proximity to coronary arteries can generate vasospasm and ST-segment elevation. In this case, the ablation site was in close proximity to the Cx artery. RF ablation was dismissed, given the elevated risk of coronary artery injury, and cryoenergy was undertaken. A mild Cx vasospasm occurred during energy delivery, but it did not cause ST-segment elevation or chest angina. This was regarded as an advantage over RF, which may have more likely caused a permanent vascular injury at that location. The Cx vasospasm observed during cryoenergy delivery was mild and affected the whole trajectory of the lateral branch rather than showing a focal mechanical

Figure 3  Fluoroscopic projections. Upper left image: Left anterior oblique (LAO) view of the coronary sinus phlebography showing the great cardiac vein (GCV) and anterior interventricular vein (AIV) distal to the inflated balloon. Upper right image: Right anterior oblique view of the 6-mm focal cryoablation catheter placed at the most distal aspect of the GCV, toward the AIV. Lower left image: LAO view of the 6-mm focal cryoablation catheter placed at the bifurcation of a lateral branch of the circumflex (Cx) artery, where the earliest activity was recorded. Lower right image: LAO cranial view (Spider). A vasospasm of the lateral branch of the Cx is observed while cryoenergy is being delivered at the effective lesion site, reaching –82 °C. A 4-mm irrigated-tip radiofrequency catheter was placed at the endocardial site (Kissing catheter). After cryoablation was completed and cryoenergy delivery discontinued (total dose of 240 seconds and 2 freeze-thaw-freeze cycles), a coronary angiogram was repeated. There was no vasospasm or coronary injury observed.
stenosis owing to “ice-ball” compression. The spasm was reversed with intra-coronary nitroglycerin, supporting this hypothesis.

To our knowledge, this is the first case reported in literature describing the use of cryoenergy through the coronary sinus system (CSS), for the treatment of LVS-related arrhythmias. Prior descriptions of cryoablation at the aortic cusps have been well correlated with significant vasospasm and ST-segment upstroke, while animal studies have shown that catheter-based cryoablation can produce lesions in the musculature of the adjacent ventricle when accessed from the CSS without significant injury to the CS or adjacent artery. Our case shows the development of a mild vasospasm, without any major clinical significance. The implications of this are the following: that cryoenergy delivery at the CSS can be safely performed, without the need of repeated coronary angiography.

Although coronary artery vasospasm occurred during cryoenergy delivery within the CS, it lacked clinical relevance, as it presented without symptoms and no ST-segment elevation, in contrast to those observed with RF energy. This rendered cryoablation as a safe and effective alternative for treating LVS VAs with high risk of coronary artery injury.

**Limitations**

Although coronary angiography showed no coronary artery stenosis after cryoablation, this was not repeated during follow-up. Nevertheless, stress testing was performed and no signs of myocardial ischemia were observed.

**Conclusion**

Catheter ablation of VAs arising from the LVS can be challenging. The risk of vascular injury is a major concern during ablation. Cryoenergy could be an effective and safe method to ablate LVS arrhythmias with sites of origin superior to the GCV, when RF catheter ablation is not recommended owing to potentially hazardous effects to the coronary vessels. This should be further investigated in prospective clinical trials.

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