Turk Kardiyol Dern Ars 2020;48(2):171-176 doi: 10.5543/tkda.2019.25342

CASE REPORT

Cryoablation of ventricular arrhythmia originating from the left coronary cusp in an adolescent with PVC-induced cardiomyopathy

Ventriküler ekstrasistol ilişkili kardiyomiyopatili bir adölesanda başarılı sol koroner yaprakçık kriyoablasyonu

Yakup Ergül, M.D., Senem Özgür, M.D., Hasan Candaş Kafalı, M.D.

Department of Pediatric Cardiology, University of Health Sciences İstanbul Mehmet Akif Ersoy Thoracic and Cardiovasculer Surgery Training and Research Hospital, İstanbul, Turkey

Summary– Ventricular arrhythmias arising from coronary cusps are not uncommon. However, the mapping and ablation of outflow tract ventricular arrhythmias originating from aortic cusps can be challenging. Radiofrequency ablation of this area can cause rare but serious complications. This was a report of a 17-year-old male patient with very frequent, nonsustained ventricular tachycardia attacks and premature ventricular contraction-induced cardiomyopathy. The origin of the ventricular arrhythmia was determined to be the left coronary cusp, and the patient was treated successfully with cryoablation. In high-risk areas, cryoablation is an effective and safe alternative method.

Özet– Koroner yaprakçıklardan kaynaklanan ventriküler aritmi nadir olmayan bir durumdur. Ancak aortik yaprakçıklardan kaynaklanan çıkım yolu ventriküler aritmilerinin haritalaması ve ablasyonu uğraştırıcı olabilir. Bu alanda radyofrekans ablasyon nadir ancak ciddi komplikasyonlara sebep olabilir. Bu yazıda, çok sık *nonsustained* ventriküler taşikardi atakları ve prematür ventriküler kontraksiyon ilişkili kardiyomiyopatisi olan 17 yaşındaki erkek hasta sunuldu. Hastanın ventriküler aritmisi, sol koroner yaprakçık kaynaklıydı. Hastaya başarılı kriyoablasyon işlemi uygulandı. Yüksek riskli bölgelerde, kriyoablasyon etkili ve güvenli bir alternatif yöntemdir.

Premature ventricular contractions (PVCs) are very common and usually do not require treatment.[1] However, when they cause symptoms such as PVC-induced cardiomyopathy or malignant arrhythmia, they should be treated.[2] Previously, a PVC burden of 24% was accepted as the threshold value for the development of cardiomyopathy;[3] however, it was later understood that there are variables other than PVC burden, like the origin of the PVCs, that can influence the development of cardiomyopathy.[2]

Outflow tract ventricular contractions can originate from either the right ventricular outflow tract (RVOT) or the left ventricular outflow tract (LVOT). The supravalvular region of the coronary cusp and the infravalvular endocardial region within the left ventricle (LV) are important sources on the left side. Ablation procedures in these areas have a high risk because of the proximity to vital structures, such as the coronary arteries. For this reason, the anatomy of this region should be well known before commencing an ablation procedure.

RF ablation in this region has been performed for a long time. However, it has been associated with life-threatening collateral damage.[4] Cryoablation should be considered as an alternative ablation energy method, as careful use appears to have fewer harmful effects on the coronaries.[5] Moreover, an atrioventricular (AV) block created with cryoablation is reversible. This is a case report of a patient who was treated successfully with coronary cusp cryoablation.

Abbreviations:

3D	*Three-dimensional*
AV	*Atrioventricular*
ECG	*Electrocardiogram*
EPS	*Electrophysiology study*
LCC	*Left coronary cusp*
LMCA	*Left main coronary artery*
LV	*Left ventricle*
LVOT	*Left ventricular outflow tract*
PVC	*Premature ventricular contraction*
RF	*Radiofrequency*
RVOT	*Right ventricular outflow tract*
VT	*Ventricular tachycardia*

Received: August 24, 2018 *Accepted:* August 05, 2019
Correspondence: Dr. Yakup Ergül. Mehmet Akif Ersoy Kalp ve Damar Cerrahisi Eğitim ve Araştırma Hastanesi, Çocuk Kardiyoloji Kliniği, İstanbul, Turkey.
Tel: +90 212 - 692 20 00 e-mail: yakupergul77@gmail.com

CASE REPORT

A 17-year-old male patient was referred to our center for PVC-related cardiomyopathy. The patient had been experiencing fatigue, abdominal pain, and stomach problems for 6 months. Frequent PVCs, ventricular triplets, and slow-rate ventricular tachycardia (VT) were detected on an electrocardiogram (ECG). To reduce the PVC load, a beta blocker agent was prescribed. A surface ECG showed frequent PVCs with a left bundle branch block, an inferior axis, a transition zone at V3, and a slow VT of 3-4 beats (Fig. 1). The intrinsicoid deflection time and the maximum deflection index of the PVCs were calculated as 120 milliseconds and 67%, respectively. There was no pseudodelta wave. Echocardiography images showed left ventricular (LV) dilatation. The ejection fraction was measured as 40% and the fractional shortening as 20%. About 70% of the Holter recordings indicated uniform PVC and non-sustained VT attacks at a speed of 140/minute. Cardiac magnetic resonance revealed LV dilatation and a decrease in LV function, but no findings supported arrhythmogenic right ventricular dysplasia. The patient underwent an exercise stress test, and even though the PVCs were suppressed at the maximal heart rate, there were very frequent PVCs and non-sustained VT attacks in the beginning and recovery phases. To completely—or at least largely—eliminate the PVCs, a decision was made to conduct an electrophysiology study (EPS) and ablation.

At the start of the procedure, local anesthesia was administered to avoid suppression of the PVCs. A three-dimensional (3D) navigation system (EnSite NavX, St. Jude Medical, Inc., St. Paul, MN, USA) and fluoroscopy were used. The diagnostic EPS catheters, guided by EnSite Velocity 3D electroanatomical mapping (St. Jude Medical, Inc., St. Paul, MN, USA), were placed within the right atrium, coronary sinus, and right ventricle. After basic measurements were taken, right atrial and right ventricular mapping was performed with a 6-F diagnostic catheter. In the posterior of the RVOT, the point of earliest activation was 33 milliseconds before the surface QRS complex and was marked using activation mapping (Fig. 2). At this point, a 90%–93% match was achieved with pace mapping. Three RF ablation lesions were delivered at 50 watts and 60°. However, the PVCs could not be completely eradicated during the RF procedure despite the burn response. A left-sided source was suspected due to the dominant negativity at DI and aVL and the early PVC transition ratio (V3). A 7-F, 4-mm tip RF Marin-R catheter (Medtronic, Inc., Min-

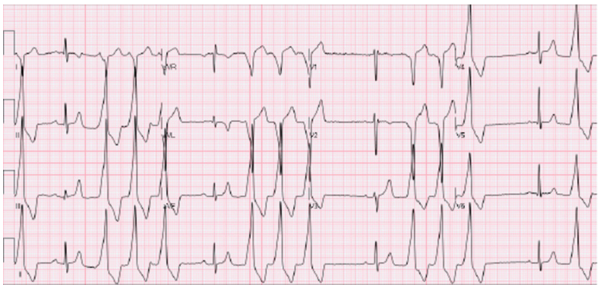

Figure 1. Frequent, nonsustained ventricular tachycardia attacks are seen on a surface electrocardiogram. The premature ventricular contractions (PVCs) have a left bundle branch block-inferior axis pattern. The PVCs transition zone was V3, which is before the sinus transition zone (V4).

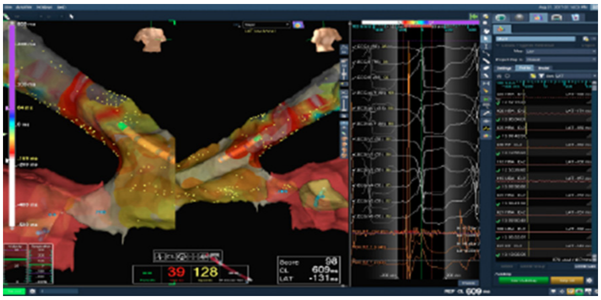

Figure 2. This illustration shows the initial ablation zone in the right ventricular outflow tract posteroseptal region. This unipolar recording is in the QS pattern. In unipolar recordings, the QS pattern is noteworthy.

Figure 3. There was a potential in the left coronary cusp. During premature ventricular contractions, the coronary sinus pattern was eccentric. This was in favor of the left ventricle source.

neapolis, MN, USA) was advanced to the aortic valve in a retrograde direction. The potential and fractional signals in the left coronary cusp (LCC) were obtained (Fig. 3). Moreover, for the first time, the earliest local ventricular activation was sufficient to precede the surface QRS complex by 56 milliseconds. Since the target was close to the left main coronary artery (LMCA), injections were given at the 45° left anterior oblique, 30° right anterior oblique, and 45° left anterior oblique/45° caudal positions before starting the procedure (Fig. 4). As it was not possible to deliver RF energy further in the area close to the LMCA, it was decided to continue the procedure with cryoablation, and the RF catheter was replaced with a cryoablation catheter. A separate focus located 6 mm from the LMCA was labeled using the EnSite Velocity 3D electroanatomical mapping system (Fig. 5). The position of the catheter was also confirmed fluoroscopically (Fig. 6).

First, a lesion on the marked region was created with an application lasting 360 seconds. Immediately after reaching a temperature of -80°C, the PVCs were completely eliminated during the application of the first lesion. Five more lesion applications of 240 seconds each were created in the vicinity of the target area. No arrhythmias were induced with or without orciprenaline and dobutamine. The procedure lasted 3 hours and 25 minutes, with a low fluoroscopy time of 8.7 minutes. During and after ablation, injections into the LMCA were repeated, and the coronary artery was

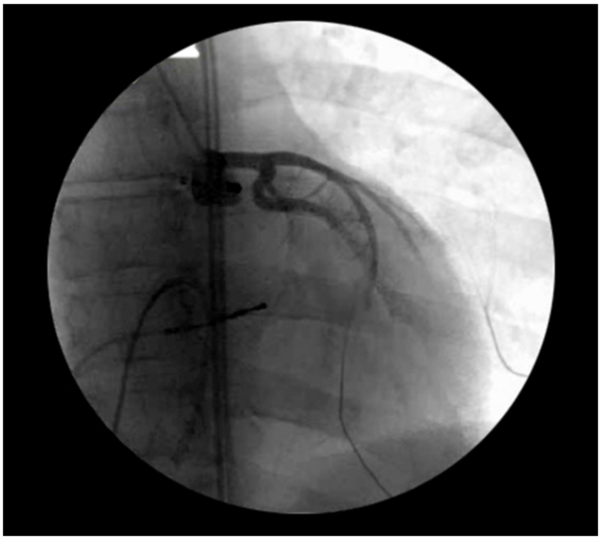

Figure 4. Left coronary artery patency was checked with recurrent angiograms The left Judkins catheter was kept in the left coronary cusp during all of the ablation. However, injection from the left main coronary artery orifice was performed only at the beginning and end of the procedure.

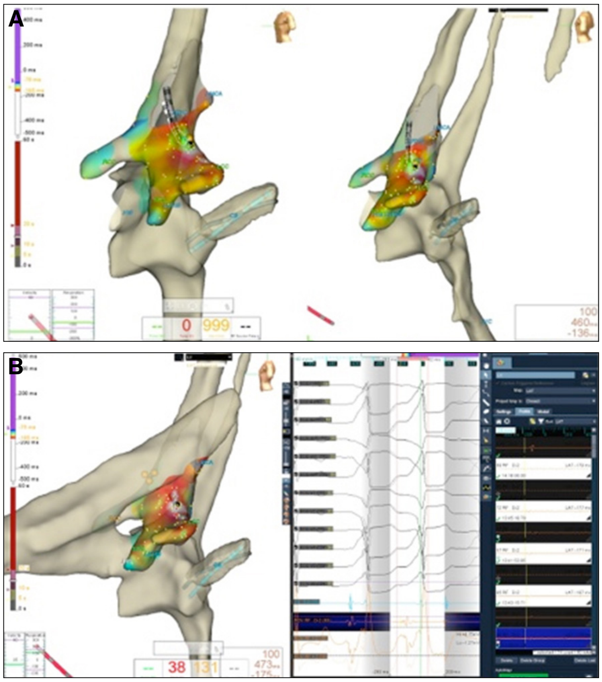

Figure 5. **(A)** Image of cryoablation catheter on a three-dimensional system. The distance from the marked point to the left main coronary artery is sufficient for safe ablation; **(B)** The distance between the first ablation zone and the successful point in the right ventricular outflow tract (RVOT) can be seen (The first ablation zones in the RVOT are indicated with yellow dots).

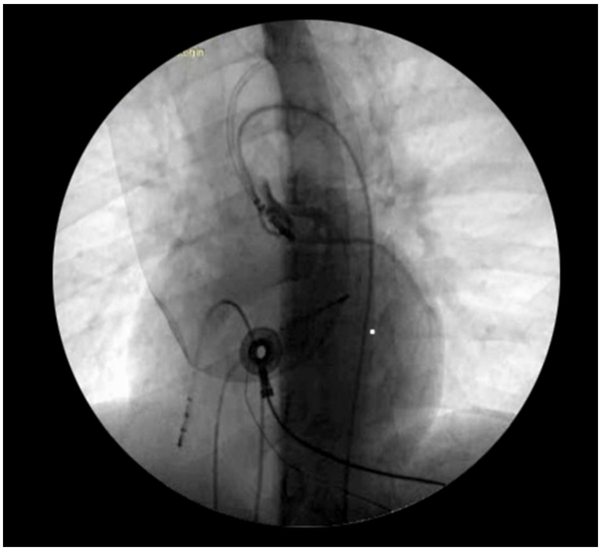

Figure 6. The location of the cryoablation catheter was confirmed with angiography.

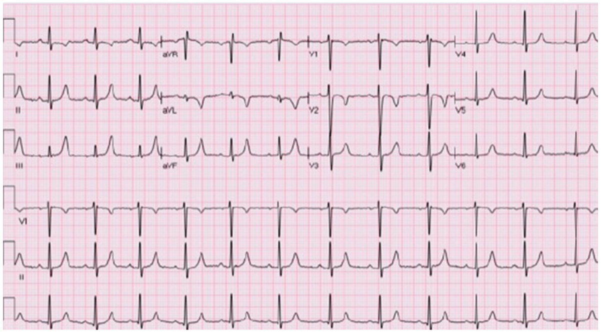

Figure 7. The patient's electrocardiogram results 1 day after the operation.

observed to be patent. The left Judkins catheter was not kept in the LMCA orifice during the entire procedure, and no ST-T changes were seen on the ECG. The procedure was terminated without complications. Heparin was administered to maintain a target activated clotting time of approximately 250–300 seconds. The patient was discharged after 1 night. Holter monitoring and another ECG did not demonstrate any PVCs after the procedure (Fig. 7). There was no damage to, or failure of, the aortic valve. Acetylsalicylic acid therapy was recommended for 3 months. Three months after the procedure, the patient did not have any symptoms, and the LV dilatation was reversed on echocardiography; however, full functional and anatomical recovery took a year. There were no indications of PVCs on a Holter recording 1 year after the procedure.

DISCUSSION

In the absence of structural heart disease, PVCs are almost always considered benign.[1,6] However, frequent PVCs can lead to depressed LV function, namely, PVC-induced cardiomyopathy.[2,7] In addition to the PVC burden, there are several risk factors associated with cardiomyopathy development. The long-term presence of PVCs, the absence of symptoms, a PVC with an epicardial origin, and a longer PVC QRS duration are linked to cardiomyopathy.[2,7–9] Due to the 75% PVC rate in our patient, the cardiomyopathy was largely associated with PVC frequency.

Outflow tract VT or frequent PVCs often occur in the absence of structural heart disease and account for 10% of all VT.[1,10] They may present in the form of isolated or incessant PVCs, or as tachycardia.[2,11] An important portion of outflow tract PVCs originate from the LVOT, including the cusps of the aortic valve.[12,13]

Due to their spatial proximity, it can be difficult to distinguish between PVCs originating from the RVOT and the LVOT.[14,15] In 3D, the aorta is located between the RVOT, the tricuspid annulus, and the mitral annulus. The RVOT has a slightly leftward and anterior position compared with the aorta. The right coronary cusp is located just behind the posterior portion of the RVOT, while the left coronary cusp is located in front of the mitral anterior leaflet and forms the aorto–mitral continuity.[15,16] The noncoronary cusp does not usually come into direct contact with the ventricular myocardium. PVCs may also originate from the AV valve annulus.[14,15] The anteroseptal and para-Hisian regions on the side of the tricuspid valve and the anterolateral and posteroseptal regions on the side of the mitral valve are the most common sources of PVCs.[14–16] PVCs can also originate from the epicardial regions. The crux where the AV groove and the posterior interventricular groove meet, and the summit, which is the superior portion of the LVOT, are the most important sites for epicardial PVC origin.[14–16]

The extension of the myocardium into the aorta makes the aortic cusp one of the most important targets of VT ablation. Ablation of VT and PVCs originating from the aortic cusps is indicated in patients with sustained arrhythmias, persistent symptoms that cannot be controlled with medical treatment, and arrhythmia-induced cardiomyopathies.[10,11,13]

RF ablation is still widely used in the treatment of outflow tract VT. However, it may be associated with life-threatening coronary collateral injury and irreversible AV conduction damage in certain locations.[4,5] In such regions, it is necessary to limit the amount of energy and not get too close to vital structures. Both can lead to a decrease in RF ablation efficiency.

In an animal study conducted by d'Avila et al.,[17] it was determined that the diameter and depth of lesions created by cryoablation were not more superficial or smaller than those produced by RF.[7] It has even been reported that, in some cases and case series, permanent cures were provided with cryoablation at some points where RF was not effective. Cryoablation is significantly safer than other methods in regions close to vital structures. Histological evaluation has demonstrated that cryolesions cause less endothelial disruption. Additionally, they provide a more homogeneous border zone with preservation of extracellular collagen than RF.[17,18] The latter is of particular interest when ablating near critical structures, including the His bundle and coronary vessels, which are encased in a thin layer of connective tissue. Thus, both the AV node damage and coronary spasm that are created by cryoablation are reversible.

Moreover, catheter adherence appears to be another important advantage of cryoablation versus other techniques. Since a large quantity of blood flow occurs in the ascending aorta, catheter stability is of primary concern during ablation. Cryoablation appears to dramatically reduce serious side effects without sacrificing either lesion size or depth.[17–19]

The biggest drawback of cryoablation is the relative catheter stiffness. However, weight-appropriate catheter selection and careful catheter manipulation can overcome this problem. Another concern is recurrence. Nonetheless, consistent with case series in the literature, there was no recurrence in our case.

In conclusion, cryoablation may be an effective and safe alternative method for the elimination of ventricular arrhythmias above the aortic valve in selected patients. Although we observed acute and short-term success in our patient, other studies are required to assess the likelihood of long-term recurrence and complications.[20]

Peer-review: Externally peer-reviewed.

Conflict-of-interest: None.

Informed Consent: Written informed consent was obtained from the patient for the publication of the case report and the accompanying images.

Authorship contributions: Concept: Y.E., S.Ö., H.C.K.; Design: Y.E., S.Ö., H.C.K.; Supervision: Y.E., S.Ö., H.C.K.; Materials: Y.E., S.Ö., H.C.K.; Data collection: Y.E., S.Ö., H.C.K.; Literature search: Y.E., S.Ö., H.C.K.; Writing: Y.E., S.Ö., H.C.K.

REFERENCES

1. Callans DJ. Premature Ventricular Contraction-induced Cardiomyopathy. Arrhythm Electrophysiol Rev 2017;6:153–5.
2. Panizo JG, Barra S, Mellor G, Heck P, Agarwal S. Premature Ventricular Complex-induced Cardiomyopathy. Arrhythm Electrophysiol Rev 2018;7:128–34. [CrossRef]
3. Baman TS, Lange DC, Ilg KJ, Gupta SK, Liu TY, Alguire C, et al. Relationship between burden of premature ventricular complexes and left ventricular function. Heart Rhythm 2010;7:865–9. [CrossRef]
4. Roberts-Thomson KC1, Steven D, Seiler J, Inada K, Koplan BA, Tedrow UB, et al. Coronary Artery Injury Due to Catheter Ablation in Adults. Circulation 2009;120:1465–73. [CrossRef]
5. McDonnell K, Rhee E, Srivathsan K, Su W. Novel utility of cryoablation for ventricular arrhythmias arising from the left aortic cusp near the left main coronary artery: a case series. Heart Rhythm 2014;11:34–8. [CrossRef]
6. Aras D, Topaloglu S, Ozeke O, Cay S, Ozcan F, Baser K. Left coronary cusp cryoablation guided by electroanatomic mapping for outflow ventricular arrhythmias. Int J Cardiol 2016;211:137–9. [CrossRef]
7. Yalin K, Gölcük E. Frequent Premature Ventricular Contractions and Cardiomyopathy, Chicken and Egg situation. Atr Fibrillation 2017;10:1674. [CrossRef]
8. Deyell MW, Park KM, Han Y, Frankel DS, Dixit S, Cooper JM, et al. Predictors of recovery of left ventricular dysfunction after ablation of frequent ventricular premature depolarizations. Heart Rhythm 2012;9:1465–72. [CrossRef]
9. Sadron Blaye-Felice M, Hamon D, Sacher F, Pascale P, Rollin A, Duparc A, et al. Premature ventricular contraction-induced cardiomyopathy: Related clinical and electrophysiologic parameters. Heart Rhythm 2016;13:103–10. [CrossRef]
10. Aliot EM1, Stevenson WG, Almendral-Garrote JM, Bogun F, Calkins CH, Delacretaz E, et al. EHRA/HRS Expert consensus on catheter ablation of ventricular arrhythmias. Europace 2009;11:771–817. [CrossRef]
11. Badran H, Samir R, Amin M. Outflow tract ventricular premature beats ablation in the presence or absence of structural heart disease: Technical considerations and clinical outcomes. Egypt Heart J 2017;69:273–80. [CrossRef]
12. Lerman BB, Stein KM, Markowitz SM Mittal S, Iwai S. Ventricular tachycardia in patients with structurally normal hearts. In: Zipes DP, Jaife J, editors. Cardiac Electrophysiology: from Cell to Bedside. 4th ed. Philadelphia, PA: WB Saunders; 2004. p. 668. [CrossRef]
13. Jamil-Copley S, Bokan R, Kojodjojo P, Qureshi N, Koa-Wing M, Hayat S, et al. Noninvasive electrocardiographic mapping to guide ablation of outflow tract ventricular arrhythmias. Heart Rhythm 2014;11:587–94. [CrossRef]
14. Yamada T, Kay GN. Anatomical Consideration in Catheter Ablation of Idiopathic Ventricular Arrhythmias. Arrhythm Electrophysiol Rev 2016;5:203–9. [CrossRef]
15. Heeger CH, Hayashi K, Kuck KH, Ouyang F. Catheter Ablation of Idiopathic Ventricular Arrhythmias Arising From the Cardiac Outflow Tracts – Recent Insights and Techniques for the Successful Treatment of Common and Challenging Cases. Circ J 2016;80:1073–86. [CrossRef]
16. Yamada T, Litovsky SH, Kay GN. The left ventricular ostium: an anatomic concept relevant to idiopathic ventricular arrhythmias. Circ Arrhythm Electrophysiol 2008;1:396–404.

17. d'Avila A, Thiagalingam A, Holmvang G, Houghtaling C, Ruskin JN, Reddy VY. What is the most appropriate energy source for aortic cusp ablation? A comparison of standard RF, cooled-tip RF and cryothermal ablation. J Interv Card Electrophysiol 2006;16:31–8. [CrossRef]
18. Uppu SC, Tuzcu V. Cryoablation of ventricular tachycardia arising from the left-coronary sinus cusp. Pediatr Cardiol 2013;34:725–8. [CrossRef]
19. Chen J, Lenarczyk R, Boveda S, Richard Tilz R, Hernandez-Madrid A, Ptaszynski P, et al. Cryoablation for treatment of cardiac arrhythmias: results of the European Heart Rhythm Association survey. Europace 2017;19:303–7. [CrossRef]
20. Wackel PL, McCrary AW, Idriss SF, Asirvatham SJ, Cannon BC. Radiofrequency Ablation in the Sinus of Valsalva for Ventricular Arrhythmia in Pediatric Patients. Pediatr Cardiol 2016;37:1534–8. [CrossRef]

Keywords: Coronary cusp; cryoablation; premature ventricular contraction-induced cardiomyopathy.

Anahtar sözcükler: Koroner yaprakçık; kriyoablasyon; ventriküler ekstrasistolün tetiklediği kardiyomiyopati.