Catheter ablation of ventricular arrhythmia originating in the tricuspid annulus in a patient with biventricular noncompaction: a case report

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Summary– It is rare for ventricular tachycardia arising from the right ventricle to originate in the tricuspid annulus, and the clinical presentation and cardiac abnormalities associated with this type of arrhythmia have not been clearly established. This report describes a case of biventricular noncompaction presenting with ventricular arrhythmia originating in the tricuspid annulus and successfully treated with radiofrequency ablation.

The tricuspid annulus is an uncommon site of origin for ventricular tachycardia (VT) originating from the right ventricle (RV).[1]

This report presents a case of biventricular noncompaction (NC) in which the tricuspid annulus was the site responsible for the ventricular arrhythmia.

CASE REPORT

A 20-year-old male presented to our clinic with palpitation of six months duration. No significant findings were noted during the physical examination. Baseline electrocardiography (ECG) showed sinus rhythm, and frequent premature ventricular contractions (PVCs) with a left bundle branch block (LBBB) pattern and superior axis (Figure 1). More than twenty thousand PVCs and 2 episodes of nonsustained VT were revealed by 24-hour Holter ECG monitoring. Transthoracic echocardiography showed normal left ventricular systolic function, but hypertrabeculation of the left ventricle (LV) apical and mid-antero-lateral segments and RV lateral segments (Figure 2a). The patient underwent magnetic resonance imaging (MRI) which showed hypertrabeculation in LV and RV (Figure 2b). Thus, biventricular NC was diagnosed. In the electrophysiological (EP) study, spontaneous PVCs were present, and RV mapping showed that the earliest endocardial activation site was in the midlateral portion of the tricuspid annulus. Radiofrequency ablation applied to this site resulted in resolution of the PVCs (Figure 2c, d). The patient was discharged on acetylsalicylic acid therapy, and during a 3-month follow-up was asymptomatic, with no PVCs revealed on 24-hour Holter ECG monitoring.

Abbreviations:
ECG Electrocardiography
EP Electrophysiological
ICD Intracardiac cardioverter defibrillator
LBBB Left bundle branch block
LV Left ventricle
MRI Magnetic resonance imaging
NC Noncompaction
PVCs Premature ventricular contractions
RF Radiofrequency
RV Right ventricle
VT Ventricular tachycardia
Discussion

Noncompaction of the ventricular myocardium is a rare cardiomyopathy resulting from abnormal embryogenesis of the endocardium and myocardium, leading to hypertrophic ventricular trabeculations.\(^1\) Echocardiography is frequently used in the diagnosis of myocardial NC and the presence of an end-systolic ratio of noncompacted to compacted layers >2 is accepted as diagnostic for NC.\(^1\) In our case, echocardiography was the initial method of diagnosis, but the apical region could not be properly viewed and MRI was used to confirm the diagnosis.

The main clinical manifestations of NC include congestive heart failure, arrhythmias and systemic thromboemboli.\(^2\) Our patient did not have heart failure or history of a systemic thromboembolic event. Frequent ventricular arrhythmias was the main complaint. Ventricular arrhythmias are the major cause of death in NC.\(^2\) The ECG morphology in this case resembled idiopathic ventricular arrhythmias originating from the tricuspid annulus. Idiopathic right ventricular arrhythmias are relatively benign and can be treated with drug therapy and radiofrequency (RF) ablation.\(^3,4\)

In our case, the RV myocardium was also abnormal, which may have been linked to the localization of the arrhythmia in the tricuspid annulus. In the liter-
ature, Honarbaksh et al. reported a case of NC complicated by VT originating in the tricuspid annulus. [5] In that case MRI showed normal RV function, but the authors mentioned that normal RV trabeculations can be difficult to differentiate from those seen in NC cases with biventricular disease. In our case, MRI showed RV NC, which supports the possible clinical importance of an abnormal RV myocardium in the precipitation of arrhythmias originating in the RV. Also, we have previously reported two different cases of NC with normal LV function, normal RV MRI findings and presenting with RV outflow tract tachycardia.[6,7] The electrophysiological basis for arrhythmias in patients with NC has not been clearly defined. Even though it may be thought that the presence of NC arrhythmias originating in the RV is coincidental, the possibility of the presence of a cellular abnormality affecting both RV and LV is logical. Microscopic changes or structural abnormalities undetected by imaging modalities may explain these unusual clinical presentations.[5–7]

Previous case series of idiopathic ventricular arrhythmias originating in the tricuspid anulus have shown that RF ablation is an effective treatment modality.[3,4] However, in patients with myocardial NC, the role of RF ablation has not been well defined and because of uncertainty about the etiology, we chose electrophysiology (EP) study and RF ablation in our patient. In the management of myocardial NC-related arrhythmias, intracardiac cardioverter defibrillator (ICD) implantation plays an important role and guidelines recommend ICD implantation in cases of spontaneous VT.[8] In our case, Holter monitoring in the follow-up period showed no ventricular arrhythmias, so an ICD was not implanted.

Prevention of thromboembolic complications is crucial in patients with NC. Some authors have recommended long-term prophylactic anticoagulation for all patients. Nonetheless, we think chronic antiplatelet therapy was sufficient for our patient, who had normal systolic function.[8]

Many studies have shown that ECG is useful in localizing the site of origin of PVCs before an ablation procedure. On 12-lead ECG, the presence of VT/PVCs showing an LBBB pattern with positive QRS polarity in leads I, V5 and V6 may indicate the tricuspid annulus as the origin of the arrhythmia. In the literature, the septal region has been reported as the most common site for VT/PVCs whose origin is the tricuspid annulus.[3]

The presence of notching of the QRS complexes in the inferior leads, rS pattern in lead V1, transition at V4 or later, and QRS duration ≥160 ms may indicate the free wall of the tricuspid annulus as the source of the arrhythmia.[3,4] In our case, all of these ECG findings were present and EP study confirmed the origin of the arrhythmia as the midlateral portion of the tricuspid annulus. Tada et al. demonstrated that success rates of RF catheter ablation for VT/PVCs originating from the lateral segment of the tricuspid annulus are higher than for VT/PVCs of septal origin, the rates being 90 and 57% respectively.[3]

Ablation of accessory pathways in the tricuspid annular region may be difficult secondary to problems related to anatomical abnormalities and insufficient stabilization of the ablation catheters. In our case, we completed an anchoring maneuver which involved excessive flexion of the catheter tip on the ventricular surface of the tricuspid annulus and pulling back the catheter until it rested beneath the junction of the tricuspid leaflet and annulus. If this maneuver is unsuccessful due to anatomical problems such as tricuspid regurgitation, annular dilation or dilation of the right heart chambers, a long sheath may increase stabilization of the catheter and allow the ablation procedure. For ablation of right-sided cardiac structures, catheters are usually positioned via a femoral vein, but in some cases this may be very difficult, and many authors recommend the transjugular approach with a long sheath to counter the disadvantages of the superior approach.[9]

In cases where it may be difficult to localize the precise origin of a PVC with conventional activation mapping, a halo catheter and a recording of a bipolar electrogram polarity reversal may be used.[10] Previous case reports also recommend the use of a 3-D mapping system (CARTO biosense Webster, NavX St. Jude) to precisely locate ventricular arrhythmias originating in the tricuspid annulus.[11]

Noncompaction involving both ventricles may manifest as ventricular arrhythmias originating in the tricuspid annulus. Catheter ablation is an effective treatment modality for this type of arrhythmia.

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REFERENCES


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Anahtar sözcükler: Ablasyon, kateter; ekokardiyografi; kalp yetersizliği/etyoloji; süngerimsi miyokart; triküspit kapak.