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CASE REPORT

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Idiopathic Ventricular Tachycardia: Good Prognosis but Debilitating Symptoms

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Abstract

Ventricular arrhythmias may occur in patients without anatomical heart abnormalities, a condition that is known as idiopathic ventricular arrhythmias. The most common form originates at the level of the outflow tracts. It can manifest as PVCs, non-sustained ventricular tachycardia (VT), and sustained VT. A 52-year-old female patient was admitted for 2 episodes of syncope related to a high burden of premature ventricular contractions (PVCs) and several episodes of VT with the same QRS morphology as the PVCs. A diagnosis of sustained monomorphic VT was formulated in a patient with no structural heart disease and a normal ejection fraction. Antiarrhythmic drugs such as metoprolol, propafenone, and amiodarone failed to reduce the number of PVCs, hence catheter ablation was suggested to the patient. The patient consented to the treatment, and following catheter ablation, the patient no longer experienced syncope. The 24-hour Holter ECG monitoring revealed no PVCs or VTs. PVCs and VTs from the right ventricular outflow tract (RVOT) are typically benign with a good prognosis. Catheter ablation should be employed as the definitive treatment for RVOT-VT, as our case demonstrated only partial response to antiarrhythmic drugs. (**International Journal of Biomedicine. 2022;12(4):667-670.**).

Keywords: catheter ablation • ventricular tachycardia • premature ventricular contractions • syncope • RVOT

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Abbreviations

EAM, electroanatomical mapping; **LBBB**, left bundle branch block; **LVOT**, left ventricular outflow tract; **LVOT**, left ventricular outflow tract; **PVCs**, premature ventricular contractions; **RVOT**, right ventricular outflow tract; **RBBB**, right bundle branch block; **VT**, ventricular tachycardia.

Introduction

Ventricular tachycardia (VT) may occur in patients that have no structural impairment. Most often, in this case, the origin of tachycardia is at the outflow tract level. Arrhythmias that originate at the level of the outflow tract are represented by premature ventricular contractions (PVCs), non-sustained VT, or sustained VT. Outflow tract VTs are triggered by emotional stress, and exercise, and occur more often during the day.[1,2] ECG recognition of outflow tract VT is made by the morphology and axis in the 12 leads: broadly, RVOT-VT is suggested by an LBBB morphology with an inferior axis, while LVOT-VT is recognized by an RBBB morphology with the inferior axis.[3]

Long-term options for the treatment of outflow tract VT include antiarrhythmic drugs and catheter ablation.[3] We present the case of a young female patient with RVOT-VT and 2 syncopes with failure of antiarrhythmic drug treatment that necessitated catheter ablation for a complete cure of the arrhythmia.

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Case Presentation

A 52-year-old female patient was admitted to the Department of Cardiology with palpitations and dyspnea. Palpitations were characterized by a rapid irregular rhythm, variable duration, spontaneous remission, and occurrence during both exertion and rest. In 2005, she presented with palpitations and syncope, and echocardiography revealed a normal ejection fraction and absence of valvulopathy. Due to the presence of PVCs on the ECG (Figure 1), therapy with metoprolol 50 mg per day was initiated. The patient had normal carotid arteries, a normal neurological examination, and a normal cerebral computed tomography. No other cause of syncope could be identified. After treatment with beta-blockers, symptoms persisted, so propafenone 450 mg was administered. In 2012, after seven years of treatment, the patient had a second episode of syncope. In 2005, only PVCs were discovered; all other exams were normal. Additionally, coronarography was performed, which was normal. Despite treatment with propafenone and metoprolol, the Holter ECG revealed 21,000 PVCs/24 hours (Figure 2), accompanied by episodes of nonsustained VT. Accordingly, propafenone was discontinued, and amiodarone was substituted. Under antiarrhythmic medication with amiodarone and metoprolol, she did not exhibit syncope; nonetheless, repeated Holter ECGs revealed 18,000 to 20,000 PVCs/24 hours. Catheter ablation was proposed in 2017 following an arrhythmic evaluation.

During the present hospitalization, a physical examination revealed an obese patient with a BMI of 36 kg/m^2, normal blood pressure of 110/70 mmHg and heart rate of 75 bpm, and normal lung auscultation. Her ECG showed PVCs with an LBBB morphology, inferior axis, and precordial transition in V4. Echocardiography revealed a normal left ventricular ejection fraction, normal values of left ventricular dimensions, a normal left atrium, the absence of valvulopathy, and the absence of pericardial effusion. For the quantification of PVCs, a Holter ECG revealed 18,000 PVCs with frequent episodes of nonsustained VT of 3 to 10 PVCs. After informed consent, an electrophysiological study (Figure 3) with EAM was performed. After the femoral vein was punctured, three catheters were inserted at the level of the heart: a quadripolar catheter at the level of the right ventricular apex, a decapolar catheter at the level of the coronary sinus, and a Flexibility Saint Jude catheter at the level of the RVOT. During activation mapping, the ablation catheter electrogram demonstrated a 20-ms pre-QRS local potential (Figures 4A, 4B, 5). Catheter ablation caused the elimination of PVCs and VTs, and the inability to induce VTs at the termination of the procedure (Figure 6). After the ablation, amiodarone was discontinued, and a Holter EKG 30 days after discharge revealed no PVCs; thus, metoprolol was also discontinued 24, 48, and 72-month follow-up, the patient did not take any medications, and her Holter ECG showed no PVC or syncope.

Discussion and Conclusion

VT may occur in patients with underlying heart impairment, such as ischemic heart disease,[5] dilated cardiomyopathy,[6] hypertrophic cardiomyopathy,[7] arrhythmogenic right ventricular dysplasia,[8] or in patients without structural impairment.[9] The morphology of PVCs and VTs in 12-lead derivations can provide further information on the location of the arrhythmogenic focus. Because both RVOT and LVOT are superior structures, there will be a positive QRS complex in leads D2, D3, and aVF, which corresponds to an inferior electrical axis. V1 is an anterior lead on the right side of the thorax, RVOT is also an anterior structure, and LVOT is a posterior structure relative to RVOT. When PVCs originate in the RVOT, there will be a negative QS pattern in lead V1, and when they arise in the LVOT, there will be an rS pattern in lead V1. Despite its name, RVOT is located considerably leftwards compared to LVOT. Therefore, when RVOT-VT originates from the anterior or posterior RVOT wall, the QRS complex in lead D1 will be biphasic. If the origin is towards the pulmonic valve, the QRS complex morphology in lead D1 will be QS; however, if the origin is from the right margin of RVOT, the QRS complex morphology in lead D1 will be positive. The morphology of VT in our patient was LBBB with an inferior axis. The small r wave in lead V1 and biphasic rs complex in lead D1 are consistent with the RVOT anteroseptal region origin identified by EAM. RVOT-VT is often a benign arrhythmia characterized by palpitations. However, our patient presented two syncopes due to rapid heart rates; hence, aggressive treatment with catheter ablation was recommended for a complete recovery. Viskin et al.[10] reported three patients with RVOT-VT who developed severe forms that were accompanied by syncope or cardiac arrest as a result of polymorphic VT or ventricular fibrillation. Individuals with malignant RVOT-VT had longer coupling intervals of PVCs compared to patients with benign RVOT-VT. Moreover, Shimizu et al.[11] demonstrated the electrocardiographic characteristics of malignant RVOT-VT and a correlation between a shorter coupling interval of PVCs and the risk of VT. Noda et al.[12] similarly observed shorter coupling intervals in malignant RVOT-VT types.

Chronic management of outflow tract VTs includes antiarrhythmic drugs, implantable defibrillators, or catheter ablation. Medical options include beta-blockers, verapamil, and diltiazem, which have a success rate of 20% to 50%.[13] Other antiarrhythmic medications include class IC: propafenone, flecainide, and class C: sotalol, amiodarone[14] when beta-blockers or calcium blockers are ineffective. Unfortunately, pharmacological treatment was unsuccessful in our patient, with PVC recurrence under metoprolol, propafenone, and amiodarone; therefore, catheter ablation was proposed. A history of syncope is a strong indicator that radiofrequency ablation should be considered as a therapeutic option. EAM revealed that the origin of RVOT-VT in our patient was at the anteroseptal level, with the local potential occurring 20 ms before the onset of QRS. During a 24-hour Holter ECG, radiofrequency treatments at this level rendered tachycardia uninducible and eliminated all PVCs. The patient reported neither syncope nor palpitations following catheter ablation.

Informed written consent was obtained from the patient for the publication of this case report and any accompanying medical images.

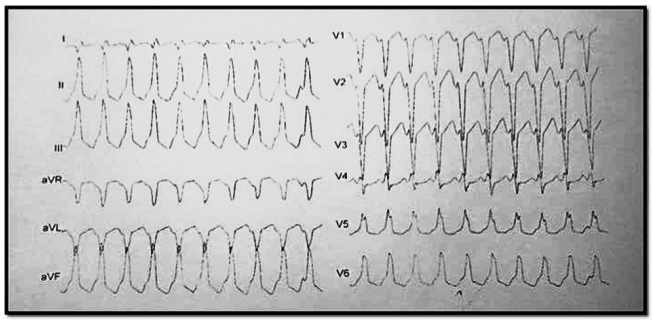


Fig. 1. *12-lead ECG during a sustained episode of ventricular tachycardia shows an LBBB morphology of the QRS with inferior axis. The biphasic pattern of QRS in lead D1 with negative QRS in leads avR and avL suggests an origin in the anterolateral RVOT.*

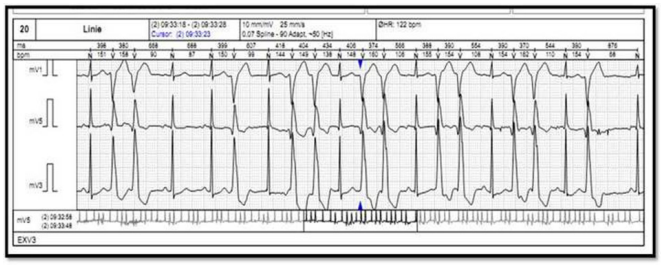
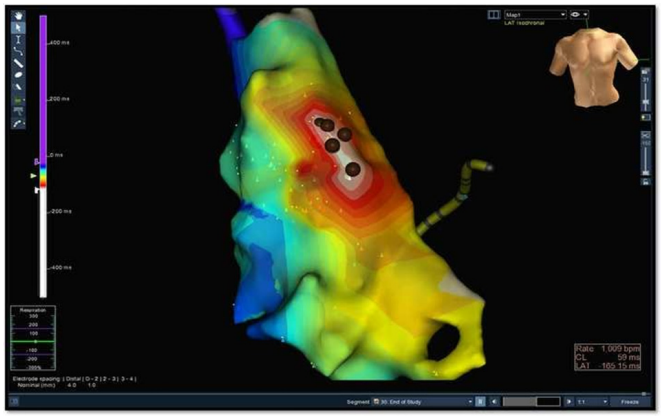
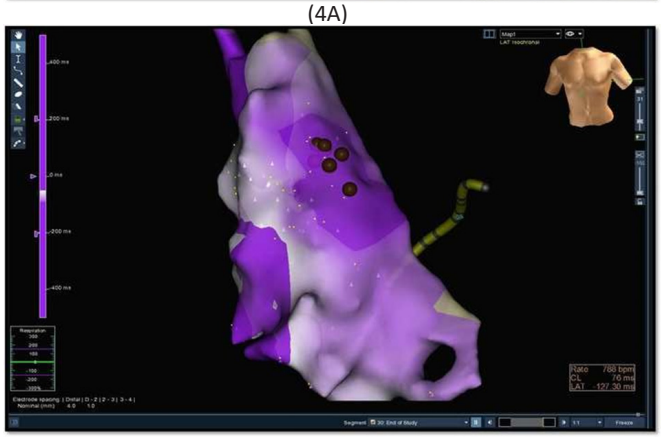


Fig. 2. *Holter ECG shows more than 21,000 PVCs/24 hours*



(4A)



(4B)

Fig. 4. *Activation map (4A) and propagation map (4B). Both maps show the origin of PVC at the level of RVOT. Activation map shows with white color the earliest RVOT zone activated by the PVC focus. The electrical activation passes from the white zone to red→yellow→green→blue, the latest area of the RVOT being activated is the posterior RVOT. Catheter ablation (5 brown dots) at the level of the earliest electrical signal (white zone) made PVCs disappear and VT uninducible. Propagation map (4B) shows the electrical activity propagating from the anterior region of the RVOT where RF dots were placed to the latest activated zone: posterior RVOT.*

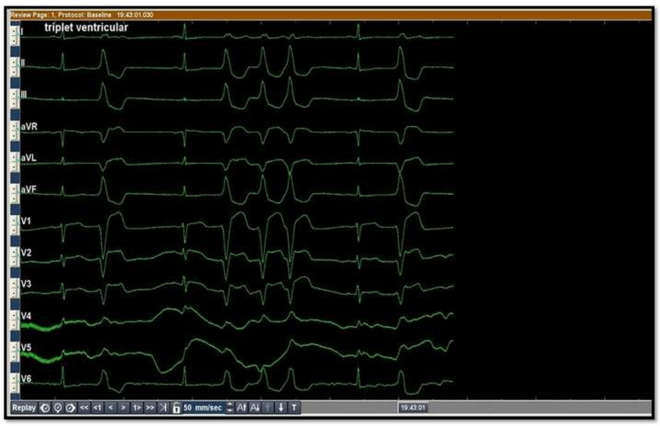


Fig. 3. *Beginning of the electrophysiological study. At the beginning of the study the patient had multiple PVCs with nonsustained episodes of VT. The morphology with LBBB pattern and inferior axis suggests origin in the RVOT.*

Fig. 5. *Local potential at the ablation target. Abl d= bipolar signal from the ablation catheter which precedes with 37 seconds the onset of QRS during PVC on 12 lead ECG. Abl uni – unipolar signal form the ablation catheter.*

Fig. 6. *12-lead ECG at the end of ablation procedure. Sinus rhythm can be seen without any PVC.*

Competing Interests

The authors declare that they have no competing interests.

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