Catheter Ablation of a Polymorphic Ventricular Tachycardia Inducing Monofocal Premature Ventricular Complex

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Abstract

Ventricular tachycardia originating from the right ventricular outflow tract (RVOT) is considered benign, but sometimes it causes polymorphic ventricular tachycardia and ventricular fibrillation, resulting in sudden cardiac death. A 58-year-old woman without structural heart disease was admitted for evaluation of recurrent episodes of syncope. Surface ECG showed frequent repetitive premature ventricular contraction (PVC) of RVOT origin. Polymorphic ventricular tachycardia triggered by the same PVC was documented by Holter ECG during an episode of syncope. Radiofrequency catheter ablation was performed to eradicate this PVC. No polymorphic ventricular tachycardia has developed after the procedure, and the patient has had no recurrence of syncope.

Key words: polymorphic ventricular tachycardia, right ventricular outflow tract, catheter ablation

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Introduction

Ventricular tachycardia in patients without evidence of structural heart disease that has QRS morphology with a left bundle branch block pattern and an inferior axis commonly originates from the right ventricular outflow tract (RVOT) (1). In general, RVOT tachycardia is believed to carry a favorable prognosis (2, 3) despite some reported cases of sudden death (4, 5). Recent studies reported that RVOT tachycardia sometimes causes polymorphic ventricular tachycardia and ventricular fibrillation that result in sudden cardiac death. It was also shown that ventricular fibrillation and/or polymorphic ventricular tachycardia caused by premature ventricular contractions originating from RVOT can be treated by radiofrequency catheter ablation (6, 7). We report a case of polymorphic ventricular tachycardia triggered by premature extrasystole from the RVOT that was successfully treated with radiofrequency catheter ablation.

Case Report

A 58-year-old woman was admitted to our hospital for evaluation of syncope. A diagnosis of idiopathic premature ventricular contraction (PVC) was made 13 years ago, but she had no symptoms and thus received no medical treatment. The patient experienced her first syncope while watching television and was transported by ambulance to our hospital. No structural heart disease was detected on physical examination or transthoracic echocardiography. A 12-lead ECG showed frequent repetitive PVC of RVOT origin, but there was no abnormal ST elevation or QT prolongation (Fig. 1). The signal-averaged electrocardiogram showed an absence of the late potential. The patient had no family history of sudden cardiac death. During hospitalization, the patient experienced syncope again. During this episode, polymorphic ventricular tachycardia triggered by PVC which presents the same morphology was documented by Holter ECG (Fig. 2). The QT interval preceding polymorphic ventricular tachycardia was normal, and the coupling
Figure 1. Premature ventricular contraction of right ventricular outflow tract origin recorded using a 12-lead electrocardiogram.

Figure 2. Initiation of polymorphic ventricular tachycardia following RVOT PVC as documented by Holter ECG recording.

interval of the initiating PVC was 400 msec. Afterwards, ventricular tachycardia did not appear on continuous ECG monitoring, but 7,600 beats of monofocal PVCs originating from the RVOT was documented in one day using Holter ECG. The polymorphic ventricular tachycardia was thought to be initiated by PVC. Thus, we decided to eradicate the culprit complexes by radiofrequency catheter ablation, targeting the focus in the right ventricular outflow tract. The ablation site was determined by activation mapping using a 20-pole LASSO catheter (Biosense Webster, Inc., Diamond Bar, CA, USA) and a 7Fr large-tip (4-mm-long) deflectable quadripolar electrode catheter (Biosense Webster, Inc.). The earliest activation site was observed at the anteroseptal right ventricular outflow tract just below the pulmonary valve with an earliest ventricular potential that was 32 msec earlier than the QRS onset of the PVC (Fig. 3). Radiofre-
Intracardiac electrograms (left panel) and fluoroscopic images (right upper and lower panels) are shown. Surface electrocardiograms as well as electrograms recorded by a LASSO catheter placed in the RVOT, the distal and proximal electrodes of the ablation catheter (ABLd and ABLp), and the right ventricular apex (RVd and RVp) are shown. The electrogram recorded by the distal electrodes of the ablation catheter at the onset of PVC preceded the onset of QRS morphology by 32 msec and a unipolar electrogram recorded by the distal electrode shows QS morphology. LAO: left anterior oblique, RAO: right anterior oblique

Figure 3. Intracardiac electrograms (left panel) and fluoroscopic images (right upper and lower panels) are shown. Surface electrocardiograms as well as electrograms recorded by a LASSO catheter placed in the RVOT, the distal and proximal electrodes of the ablation catheter (ABLd and ABLp), and the right ventricular apex (RVd and RVp) are shown. The electrogram recorded by the distal electrodes of the ablation catheter at the onset of PVC preceded the onset of QRS morphology by 32 msec and a unipolar electrogram recorded by the distal electrode shows QS morphology. LAO: left anterior oblique, RAO: right anterior oblique

quency energy was delivered to this earliest activation site with a temperature limit set at 55°C for 120 seconds. After the first application of radiofrequency energy, PVCs which were observed frequently prior to ablation had disappeared. After 5 additional applications of radiofrequency energy to this site, the induction of ventricular tachycardia was examined but could not be detected even with triple extrastimuli from the right ventricle. Holter ECG recorded one day after ablation showed that the number of PVCs had been reduced to 3 beats in a day, and their morphology was different from that recorded before radiofrequency catheter ablation. Over the subsequent 20 months, there was no recurrence of syncope. Holter ECG recording was performed 4 times during the follow-up period, but ventricular tachycardia was not documented.

Discussion

Ventricular tachycardia originating from the RVOT is characterized by left bundle branch block morphology with an inferior frontal plane axis, often requires adrenergic stimulation for induction, and can be terminated by adenosine, beta-blocker, and verapamil administration (1, 8-11). Idiopathic ventricular tachycardia originating from the RVOT in patients without structural heart disease is considered benign, and radiofrequency catheter ablation has become an effective therapeutic option for these arrhythmias (12-16). However, recent reports have shown that malignant idiopathic polymorphic ventricular tachycardia/ventricular fibrillation may occasionally originate from the RVOT, the same site of origin as the “benign” RVOT (6, 7). In the present case, 12-lead ECG showed normal QT intervals both before and after polymorphic ventricular tachycardia. The ST elevation and right bundle branch block morphology that can be seen in Brugada syndrome were not detected. The signal-averaged electrocardiogram showed an absence of the late potential. The coupling interval was not as short as those described in patients with short-coupled variant of torsade de pointes (17) or idiopathic ventricular fibrillation (18, 19). In idiopathic benign RVOT ventricular tachycardia, previous studies reported that endocardial activation time before the onset of surface QRS at successful ablation sites ranged from 26±11 msec (16) to 46±5 msec (20). In the present case, the earliest activation time was 32 msec before QRS onset at the anteroseptal right ventricular outflow tract, which falls within the range of the previous studies. These findings suggest that ventricular tachycardia typically regarded as a benign entity may actually be malignant.

The mechanism of idiopathic polymorphic ventricular tachycardia/ventricular fibrillation originating from the RVOT is unclear. Previously, it has been suggested that the mechanism of idiopathic ventricular tachycardia arising from the RVOT is triggered activity (21, 22). Kusano et al reported that rapid pacing from the RVOT reproduced polymorphic change of the QRS wave on ECG, therefore they suggested that polymorphic ventricular tachycardia origi-
nates from a single focus and chaotic ventricular conduction around the focus causes polymorphic ECG changes (23). Also, Noda et al suggested that rapid firing due to triggered activity or microreentry arising from a single focus leads to “fibrillatory” conduction, and causes polymorphic ventricular tachycardia/ventricular fibrillation (7).

Therapeutic approaches for this condition are limited, and only ICD (implantable cardioverter defibrillator) can save lives. However, although ICD is a powerful device for terminating polymorphic ventricular tachycardia/ventricular fibrillation, it cannot prevent initiation or the ensuing syncope. Previous reports suggested that radiofrequency catheter ablation was an effective treatment option for idiopathic ventricular arrhythmias from the RVOT (7, 23, 24). We also performed radiofrequency catheter ablation by targeting the initiating PVC and were able to get a satisfactory result. However, it is uncertain whether this ablation only eliminated a trigger for polymorphic ventricular tachycardia, or whether it ablated a common arrhythmogenic site responsible for both PVCs and polymorphic ventricular tachycardia. If we were only able to ablate a trigger, it is possible that the substrate for polymorphic ventricular tachycardia is still present, but the inability to induce polymorphic ventricular tachycardia by programmed stimulation in the present case suggests a modification of the arrhythmogenic substrates by radiofrequency energy. However, some studies have reported that the recurrence of episodes was found after the catheter ablation of idiopathic ventricular tachycardia/ablation (6). These findings suggest that the implantation of ICD is recommended even after successful ablation of ventricular tachycardia/fibrillation.

References