Catheter Ablation for Electrical Storm as Polymorphic Ventricular Tachycardia in a Case with Ischemic Cardiomyopathy after Coronary Artery Bypass Graft

Yasuhiro Yoshiga MD, Akihiko Shimizu MD, Shinsuke Suzuki MD, Naoki Sugi MD, Toshihide Omiya MD, Makoto Ono MD, Masaaki Yoshida MD, Masunori Matsuzaki MD

Division of Cardiology, Department of Medicine and Clinical Science, Yamaguchi University Graduate School of Medicine
Division of Cardiology and Faculty of Health Sciences, Yamaguchi University Graduate School of Medicine

We report a case involving a patient with ischemic cardiomyopathy who developed polymorphic ventricular tachycardia (PVT) and ventricular fibrillation storm after coronary artery bypass graft. Because PVT was initiated by various right bundle branch type premature ventricular contractions (PVCs), we assessed the relatively monomorphic ventricular tachycardia (MVT) during PVT. Electroanatomical mapping revealed that the earliest ventricular activation of the MVT was located in the scar border zone at the posterior septum of the left ventricle. Stable potentials which preceded the MVT were observed. Catheter ablation for the preceding potential suppressed the maintenance of the PVT, although triggered PVCs appeared frequently. Catheter ablation was effective as a bailout therapy in a patient with PVT-induced cardiomyopathy after cardiac operation.

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Key words: Polymorphic ventricular tachycardia, Radiofrequency catheter ablation, Purkinje fiber, Ischemic heart disease, Coronary artery bypass graft

Introduction

Recently, premature ventricular contraction (PVC) originating from the Purkinje system has been reported as a trigger of polymorphic ventricular tachycardia (PVT) and ventricular fibrillation (VF) in ischemic heart disease.1-3) These triggered PVCs of the ventricular tachyarrhythmia could be eliminated by focal energy delivery during catheter ablation. It is reported that the Purkinje fibers may not only trigger, but are part of the reentry circuit of monomorphic ventricular tachycardia (VT) in patients with post-infarction VT, in particular those with a relatively narrow QRS complex VT.4,5) On the other hand, it is reported that delayed potential-guided ablation is effective for electrical storm of multiple VT as a bailout therapy.6) In patients with ischemic heart disease, the mechanism of the
maintenance of PVT has not yet been clarified. An incessant or long-lasting tachycardia may cause both cardiac structural and electrical remodeling, and lead to left ventricular dysfunction (so-called tachycardia induced cardiomyopathy). We describe a patient with incessant PVT induced by Purkinje-related PVC in whom catheter ablation not only suppressed the PVT, although the initiating PVCs appeared frequently, but also improved the cardiac function.

**Case Report**

A 69-old-year male with ischemic cardiomyopathy (ICM: left main trunk (LMT) + 3 vessels disease) and mitral valve regurgitation (MR: III degree) underwent coronary artery bypass graft (CABG: left internal thoracic artery (LITA) to left anterior descending artery (LAD), saphenous vein grafts (SVG) to diagonal branch and circumflex artery (Cx) sequenclally, SVG to right coronary artery (RCA)) and mitral valve replacement (MVR). Despite hemodynamic improvement, no prolongation of the QT intervals, normal electrolytes, no recurrence of the pre-operative VT, and no catecholamine, he exhibited incessant PVT and VF 2 days after surgery. Antiarrhythmic drugs such as lidocaine, mexiletine, landiolol, nifekalant, or amiodarone, and deep sedation were unable to control the PVT/VF. Intravenous verapamil was administered and was transiently effective. Coronary angiography showed all grafts to be patent. Although cardiac function improved a day after CABG (left ventricular ejection fraction (LVEF): 25% to 30%, cardiac output (CO): 4.81/min), the repetitive PVT/VF reduced the CO (4.81/min to 1.81/min) and the LVEF (30% to 15%) 4 days after surgery. Next, we administrated oral amiodarone and performed intra-aortic balloon pumping (IABP) and percutaneous cardiopulmonary support (PCPS) for hemodynamic failure. Despite oral amiodarone and mechanical support for 9 days, we could not control the PVT/VF. Therefore, we decided to perform both an electrophysiological study (EPS) and catheter ablation for the treatment of PVT/VF.

![Figure 1](image-url)  
**Figure 1** Premature ventricular contractions (PVCs) and polymorphic ventricular tachycardia (PVT).  
A: The morphologies of the PVCs. Although all of the PVCs were right bundle branch block, the morphologies of each of the PVCs was slightly different. B: An electrocardiogram during polymorphic ventricular tachycardia. Relatively monomorphic ventricular tachycardia (MVT) appeared during PVT. The MVT was right bundle branch block, superior axis.  
S: sinus rhythm, V: premature ventricular contraction
Multipolar catheters were positioned from the left femoral vein into the right ventricle and His bundle, and from the right subclavian vein into the right atrium. Left ventricular (LV) electroanatomical mapping was performed with a 7F, 4-mm-tip electrode (Biosense-Webster Inc, California) via a retrograde transaortic approach. The morphology of the PVT and initiating PVCs were right bundle branch block type, but each of the initiating PVCs had a varying morphology, coupling interval and QRS duration (Figure 1A). Because we could not map the initiating PVCs, we mapped the relatively monomorphic VT during PVT (Figure 1B). The earliest ventricular activation site of the relatively monomorphic VT was located at the distal portion of the LV posterior septum and scar border zone. According to the LV voltage map during sinus rhythm, the LV septum and inferior wall exhibited a very low voltage and the Purkinje potentials were observed within the scar border zone. The potential of the earliest ventricular activation site had an initial fragmented component. The preceding potentials were observed at the low voltage area during a relatively monomorphic VT. The preceding poten-
tials were stable only near the earliest ventricular activation site (Figure 2). During sinus rhythm, the Purkinje potentials were found at the same site (Figure 3). The changes in the potential intervals preceded the changes in the RR intervals during relatively monomorphic VT. Bundle branch reentry was ruled out by the absence of a His bundle depolarization preceding the QRS complex during the relatively monomorphic VT. The PVT terminated during the monomorphic VT by radiofrequency ablation (with temperature limited to 55 degree and maximum power limited to 30 W) for the stable earliest preceding potential (Figure 2: site B, Figure 3, Figures 4A and B). The PVT could not be maintained after the radiofrequency ablation, although triggered PVCs appeared frequently (Figure 4C).

No complications including bundle branch block were observed after the procedure. After the radio-frequency ablation there was no further PVT or VF recurrence. The disappearance of PVT increased the CO (1.81/min to 4.51/min) and the LVEF (15% to 30%), and we could remove the PCPS and IABP 3 days after the procedure. The patient recovered well from heart failure and was discharged after an implantable cardioverter-defibrillator (ICD) implantation and amiodarone was administrated to prevent sudden cardiac death and ICD shock, because the other non-sustained VT appeared and VF was induced in the EPS 4 months after procedure. At 16 months follow-up, a defibrillator interrogation showed no further episodes of PVT/VF with oral Amiodarone (200 mg/day).

Discussion

It has been already reported that post CABG VTs were related to the severe underlying coronary artery disease and preoperative triggering factors, such as acute ischemia, electric disorders and sudden hemodynamic impairment. However, in our patient, there were no ischemia, electric disorders and post operative hemodynamic impairment immediately after operation.

First, it was very important to diagnose whether the VT in our patient was related to the Purkinje network. The preceding potential was observed at the successful ablation site during sinus rhythm and VT. Further, this potential preceded the QRS complex during VT rather than sinus rhythm. Furthermore, we could not detect the scar region in the endocardium and record the delayed potential at the successful ablation site. These findings suggested

Figure 3 Local electrograms and the surface electrograms during the initiation of polymorphic ventricular tachycardia (PVT) at His position and the ablation site.

Left panel shows the His potential during the initiation of PVT. His potential is preceding the QRS complex during sinus rhythm, but not VT. Arrows indicated His potentials.

Right panel shows the local electrograms at the ablation site during the initiation of PVT. The first beat in the left panel is a sinus beat with a Purkinje potential that precedes the ventricular activation. The second beat (**) is a premature ventricular contraction that is preceded by Purkinje potential. Arrows indicated Purkinje potentials.

TVA: tricuspid annulus, HBE_{dis}: distal His bundle electrogram. HBE_{prox}: proximal His bundle electrogram. Map_{dis}: distal mapping catheter, Map_{prox}: proximal mapping catheter, A: atrial potential
that the VT in our patient was related to the Purkinje network. However, His potential is not present during relatively monomorphic VT although it is present before the ventricular wave in PVC. Therefore we cannot differentiate VT-induced by Purkinje-related PVC from Purkinje related VT.

Some report show VF or PVT storm with ischemic heart disease treated successfully by catheter ablation targeting the PVCs that triggers VF.\textsuperscript{1-3} These reports emphasize the importance of the Purkinje network system as a trigger of VF or PVT. However, Kobayashi Y et al. reported the variety of PVCs as trigger of PVT and the participation of Purkinje network as maintenance of PVT.\textsuperscript{9} On the other hand, some reports suggest that the scar related VT is the mechanism of electrical storm in the patients with ischemic heart disease.\textsuperscript{6} In our patient we could not map the initiating PVCs because of the various morphologies that appeared. Instead, we mapped the relatively monomorphic VT during PVT. The radiofrequency ablation for the stable earliest preceding potential terminated the relatively monomorphic VT, and, thereafter the PVT could not be maintained, although triggered premature ventricular contractions appeared frequently.

The mechanisms of Purkinje fiber-related VT may be reentry, abnormal automaticity, or triggered activity. In animal models, it is suggested that both triggered activity and micro-reentry cause PVT and VF in Purkinje fibers located within the scar border.
zone. The triggered activity from the surviving distal Purkinje system in the scar border zone is required for the development of PVT/VF\textsuperscript{10,11} and micro-reentry can also cause PVT/VF, because the coexisting scar and viable myocardium create the circuit for the electrical reentry.\textsuperscript{12} It is suggested that Purkinje-muscle reentry is important as a mechanism of PVT in a 3-dimensional model of the ventricles, too.\textsuperscript{13} In our patient, because of the situation of frequent and unstable PVT and VF episodes, we could not perform a programmed electrical stimulation (induction, termination, and entrainment) before the ablation, and clarify the underlying mechanisms. Furthermore, we could not perform the detail EPS and verify the mechanism in our patient because he underwent EPS during PCPS and IABP therapy.

We made the activation map by CARTO system during monomorphic VT between PVT to find the earliest activation point. Because there is no report about this method, it needs further exploration.

Although the short PVT and triggered PVCs appeared immediately after catheter ablation, these ventricular arrhythmias disappeared gradually in two weeks. The mechanisms of disappearance of short PVT and triggered PVCs are not clear, but it may be related to the hemodynamic improvement and the chronic effect of amiodarone.

In our patient, we had to introduce IABP and PCPS because of cardiac function worsened by the PVT/VF storm and an unstable hemodynamic condition. We improved the hemodynamic condition by a catheter ablation, which terminated the maintenance of the PVT, and we were able to remove the mechanical support. Therefore, catheter ablation for PVT/VF under mechanical support may be used as a bailout therapy for cardiomyopathy induced by frequent PVT/VF.

**Conclusion**

We could prevent the maintenance of PVT by catheter ablation to the stable preceding potential as a target during monomorphic VT. Catheter ablation to terminate the maintenance of PVT may therefore be useful as a bailout therapy for the electrical storm of PVT/VF with ischemic heart disease.

**References**