Radiofrequency Energy Induced Ventricular Fibrillation in a Case of Idiopathic Premature Ventricular Contraction Originating from the Left Ventricular Papillary Muscle

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Abstract

A 15-year-old boy without structural heart disease was admitted for the treatment of frequent episodes of premature ventricular contractions (PVCs). Left ventricular mapping revealed that the origin of PVC was at the posterior papillary muscle. Diastolic small potentials were observed during sinus rhythm with a constant interval following QRS beats. This potential eventually coupled with the ventricular myocardium, resulting in the generation of PVC, and thus preceded QRS by 31 msec. Catheter ablation to this site induced non-sustained ventricular tachycardia, followed by transient ventricular fibrillation. Repeated application of radiofrequency energy eliminated PVC accompanied by the split of the diastolic potential.

Key words: premature ventricular contraction, ventricular fibrillation, papillary muscle, catheter ablation

Introduction

Idiopathic ventricular arrhythmia is considered to carry a favorable prognosis (1); however, sudden cardiac death has been reported (2). Recently, it has been shown that the left ventricular papillary muscle can give rise to ventricular arrhythmias in the normal heart (3, 4); however, the clinical significance and prognosis of ventricular arrhythmias originating from the left ventricular papillary muscle have not been fully elucidated. We report a case of premature ventricular contraction (PVC) originating from the left ventricular papillary muscle and showing transient ventricular tachycardia and fibrillation during radiofrequency energy application.

Case Report

A 15-year-old boy was admitted to our hospital for catheter ablation of PVC. The patient complained of palpitations due to PVCs that were worsened by exercise; however, he had not experienced syncope. No structural heart disease was detected on physical examination or transthoracic echocardiography. A 12-lead electrocardiogram (ECG) showed frequent repetitive PVC, but there was no abnormal ST elevation or QT prolongation (Fig. 1A). At baseline, monofocal PVC with a right bundle branch block and superior axis morphology were observed frequently (Fig. 1A). A total of 16,086 monofocal PVCs, including 253 couplet beats, were detected by Holter ECG. After left ventriculography, endocardial mapping was performed using a 20-pole electrode catheter (St. Jude Medical, Inc., St. Paul, MN) and a 7-Fr large-tip (4-mm in length) deflectable quadripolar electrode catheter (Biosense Webster, Inc., Diamond Bar, CA). Programmed ventricular stimulation did not induce ventricular tachycardia with or without the use of isoproterenol infusion. The earliest activation site during PVC was observed at the left ventricular posterior papillary muscle (Fig. 1B and 1C). Figure 1B shows the biplane left ventriculography. The area surrounded by small arrows shows the defect of contrast medium on left ventriculography, indicating the location of the posterior papillary muscle (Fig. 1B).
Figure 1C shows the earliest activation site during PVC observed at the posterior papillary muscle (Fig. 1C). During sinus rhythm, a small diastolic potential was observed with a constant coupling interval of 340 msec after the narrow QRS sinus beats (Fig. 2A). This small diastolic potential eventually coupled with the ventricular myocardium, resulting in the generation of PVC, and thus preceded the QRS by 31 msec at the left ventricular posterior papillary muscle (Fig. 2A). Meanwhile, a small potential was also observed 110 msec after the onset of QRS during sinus rhythm (open arrow in Fig. 2A). Radiofrequency energy application (20-25 W with a temperature limit set at 55°C for 60 seconds) to the earliest activation site induced non-sustained ventricular tachycardia with the same QRS morphology (Fig. 3), followed by transient ventricular fibrillation (Fig. 3), which terminated spontaneously by the cessation of radiofrequency energy application. Repeated energy application was then continued with careful observation and the frequency of spontaneous PVCs and induced ventricular tachycardia/fibrillation gradually decreased. After the 5th application of radiofrequency energy, spontaneous PVC was totally eliminated, accompanied by the split of the diastolic potential (Fig. 2B). PVC with the same morphology was not documented by Holter ECG recordings performed immediately and 1 month after the procedure. The patient was free from palpitations during the follow-up period of 17 months.

Discussion

Acceleration of tachycardia during effective radiofrequency energy application has been considered to be due to automaticity (5). Ventricular tachycardia or fibrillation induced by radiofrequency energy delivery in the present case might have been caused by the same mechanism of destruction of the abnormally automatic substrate, as suggested by Perry et al (5). A similar finding was also observed in a case of idiopathic ventricular tachycardia originating from the right ventricular outflow tract (6). Regarding the case of idiopathic ventricular tachyarrhythmia originating from the left ventricular papillary muscle, only one case report has described the occurrence of radiofrequency energy-induced ventricular fibrillation (7). Yamada et al reported that radiofrequency energy for idiopathic premature ventricular contractions arising from the left ventricular anterior papillary muscle induced ventricular fibrillation (7) and suggested automaticity as the mechanism of inducing ventricular fibrillation (7). Their case is similar to the present case; however, several points are different. First, the origin of PVC was ob-
Figure 2. Intracardiac electrograms showing electrograms at the successful ablation site located at the left ventricular posterior papillary muscle before (panel A) and after (panel B) ablation. See text for discussion. ABL: ablation catheter; LV: left ventricle, RV: right ventricle, Stim: stimulation

Figure 3. Non-sustained ventricular tachycardia (VT) with the same QRS morphology to PVC and transient ventricular fibrillation (VF) observed during radiofrequency energy (RF) application are shown.

served at the anterior papillary muscle in their case, whereas at the posterior papillary muscle in the present case. Second, diastolic potential was not observed in their case. Regarding the participation of Purkinje fibers in the ventricular tachyarhythmia originating from the left ventricular papillary muscle, different findings have been reported (3, 4). Good et al reported that the Purkinje potential was observed in 5 of 11 papillary muscle-related ventricular tachyarrhythmia patients,
and the tachycardia often originated at the Purkinje fiber-muscle interface (3), whereas no Purkinje potential was identified in 7 patients with ventricular tachycardia originating from the left ventricular posterior muscle (4). Thus, it is not clear whether Purkinje fibers contributed to the ventricular tachyarrhythmia originating from the papillary muscle. In the present case, the Purkinje potentials were recorded during sinus rhythm early and late after the QRS beat, which are different from the potential observed in the cases reported by Good et al (3). Previously, similar potential was found in fascicular ventricular tachycardia by Ouyang et al (8). They identified late diastolic potential during sinus rhythm which was generated by retrograde Purkinje conduction. They also showed that tachycardia termination was associated with the split of this diastolic potential (8), consistent with the findings in the present case. The clinical significance of the early potential observed 110 msec after QRS is not clear. However, it might be a part of the retrograde Purkinje activation within the Purkinje network as suggested previously (8, 9) because it disappeared after ablation. Although we could not determine the precise mechanism of the PVC because sustained tachycardia was not inducible before ablation, elimination of PVC following a split in the earliest diastolic potential at the PVC origin suggests critical participation of this Purkinje potential in PVC generation. The split of diastolic potential might be caused by the local conduction block at the ablation site and thus the second component may represent an electrogram in the localized blocked lesion created by radiofrequency energy. Although the mechanism of PVC is not clear, this localized block might eliminate the PVC by destroying the local reentry circuit or abnormal automatic substrate at diastolic potential recording site. Regarding the etiology of the diastolic potential, we postulated that it might be caused by retrograde Purkinje activation because it was observed with a constant interval after QRS beat both during sinus rhythm and ventricular pacing. However, it is possible that diastolic potential was caused by another mechanism such as automaticity.

It should also be noted that ventricular fibrillation may occur by the application of radiofrequency energy to the PVC originating from the left ventricular posterior papillary muscle. To the best of our knowledge, this is the first report showing ventricular fibrillation by the delivery of radiofrequency energy to the PVC origin, which is associated with probable Purkinje activation in the posterior papillary muscle.

References