Multiform Premature Ventricular Contractions and Polymorphic Ventricular Tachycardia Caused by Purkinje Activity with Slow Conduction in Idiopathic Ventricular Fibrillation

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Abstract

In several cases with idiopathic ventricular fibrillation (VF), VF was initiated by premature ventricular contractions (PVCs) from the Purkinje system. However, the precise characteristics of the Purkinje activity in patients with idiopathic VF remain unclear. We performed an electrophysiological study in a patient with idiopathic VF and examined the correlation between the Purkinje potential and the incidence of PVCs/polymorphic ventricular tachycardia (PMVT). In this case of idiopathic VF, the Purkinje activity caused multiform PVCs and PMVT. The Purkinje activity and slow conduction of Purkinje fibers are associated with the occurrence of multiform PVCs and PMVT.

Key words: Purkinje, ventricular fibrillation, polymorphic ventricular tachycardia, premature ventricular contraction, slow conduction

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Introduction

It has been reported that idiopathic ventricular fibrillation (VF) is induced by premature ventricular contractions (PVCs) with very short coupling intervals originating from the Purkinje system (1-4). We performed an electrophysiological study (EPS) in a patient with idiopathic VF and examined the correlation between the Purkinje potential and the incidence of PVCs.

Case Report

A 29-year-old woman was referred to our hospital on account of frequent episodes of VF initiated by PVCs with a short coupling interval and several implantable cardioverter-defibrillator (ICD) discharges (Fig. 1A). The patient had been diagnosed with idiopathic VF at 26 years of age due to the occurrence of spontaneous VF episodes, a normal QT interval and no evidence of Brugada-type electrocardiogram (ECG) findings. Cardiac echocardiography, right and left ventriculography, coronary angiography and a myocardial biopsy demonstrated no abnormalities. An ICD was immediately implanted after diagnosis, and treatment with oral atenolol and disopyramide was initiated.

ECG recordings obtained after admission showed multiform PVCs (Fig. 1B) and non-sustained polymorphic ventricular tachycardia (PMVT). All PVCs had a right bundle branch block configuration with various axis deviations and QRS durations. After obtaining the patient’s informed consent, EPS was performed under local anesthesia. A 7-French 20-pole electrode catheter (1-3-1 mm inter electrode spacing; Cordis Webster, Diamond Bar, USA) was positioned at the left ventricular (LV) septum, and a quadripolar electrode catheter was positioned at the bundle of His recording region (Fig. 2A). During EPS, PVCs and non-sustained PMVT were frequently observed. Under a sinus rhythm, serial Purkinje potentials were recorded immediately before the onset of QRS at the LV septum (Fig. 2B). Although the PVCs exhibited multiple configurations, diastolic Purkinje...
Figure 1. (A) ECG showing PMVT initiated by a PVC (●) with a short coupling interval (250 ms). (B) Surface ECG showing multiform PVCs. All PVCs had a right bundle branch block configuration with various axis deviations and QRS durations.

Figure 2. (A) Catheter position. Fluoroscopic right anterior oblique (RAO) and left anterior oblique (LAO) views of the 20-pole electrode catheter positioned at the left ventricular septum (LV) and the quadripolar catheter positioned at the bundle of His region (HBE). (B) Surface ECG and intracardiac electrograms demonstrating multiform PVCs (●) and diastolic Purkinje potentials (DPs; arrow). The configuration of each PVC was different; however, a DP with an identical sequence always preceded each PVC. During sinus rhythm, serial Purkinje potentials were recorded immediately before the onset of QRS.

potentials (DPs) with an identical sequence recorded at the LV septum always preceded each PVC (Fig. 2B, 3A). Occasionally, the DP was blocked, and no PVC appeared (Fig. 3A). The DPs also preceded each QRS complex of PMVT (Fig. 3B).

In order to examine the relationships between the preceding sinus cycle length, DP and multiform PVCs, we measured the QRS duration, H0-H1 interval (which reflects the preceding sinus cycle length), H1-DP interval, H1-QRS interval and DP-QRS interval in each PVC (Fig. 4). We found no relationships between the H0-H1 interval and H1-DP interval (Fig. 4A). However, inverse correlations were found between the H1-DP interval and DP-QRS interval (r=-0.76, p<0.0001) (Fig. 4C) and between the H1-QRS interval and
activation of the Purkinje fiber caused multiform PVCs. The incidence of PVCs. The results of this study are as follows: the relationship between Purkinje activation and the characteristics of the abnormal Purkinje activation and EPS; therefore, it was possible to determine the precise episodes of abnormal activation of the Purkinje fiber during PMVT. The Purkinje activation was independent of the preceding normal sinus activation, as no relationships were found between the preceding sinus cycle length (H0-H1) and the H1-DP interval. Accordingly, activation of the Purkinje fiber is associated with automaticity, and the H1-DP interval is variable.

The occurrence of multiform PVCs with various QRS durations is associated with the alteration of multiple exits and variability of the refractory period in the Purkinje network and Purkinje-muscle junction. The variable sequence of local LV electrograms observed in each PVC may also represent the alteration of exit sites and dispersion of refractoriness in the Purkinje network and Purkinje-muscle junction (Fig. 2B). Because the exit sites were not uniform, we were unable to determine the properties of the decremental conduction from the DP to the exit site. However, due to the prolonged DP-QRS interval, a conduction delay between the abnormal Purkinje activity and the exit site may have been present in this patient.

Discussion

In some cases of idiopathic VF, specific PVCs with a very short coupling interval induce PMVT and VF; catheter ablation to treat the PVCs originating from Purkinje fibers can be used to eliminate this type of idiopathic VF (1-5). However, the precise characteristics of arrhythmogenic Purkinje activation have not been described in previous reports.

In the present case, we were able to record frequent episodes of abnormal activation of the Purkinje fiber during EPS; therefore, it was possible to determine the precise characteristics of the abnormal Purkinje activation and examine the relationship between Purkinje activation and the incidence of PVCs. The results of this study are as follows: [1] activation of the Purkinje fiber caused multiform PVCs and PMVT; [2] slow conduction was observed in the Purkinje fiber and/or Purkinje-muscle junction; [3] the incidence of PVCs with a very short coupling interval was associated with a longer QRS duration following non-sustained PMVT. The Purkinje activation was independent of the preceding normal sinus activation, as no relationships were found between the preceding sinus cycle length (H0-H1) and the H1-DP interval. Accordingly, activation of the Purkinje fiber is associated with automaticity, and the H1-DP interval is variable.

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The DP, a trigger of PMVT of more than a four-beat run, exhibited a very short H1-QRS interval and an extremely long QRS duration of the initial ventricular ectopic beat (Fig. 4D). We speculate that the PVCs with a remarkably short coupling interval were accompanied by heterogeneity QRS duration of the PVCs (r=-0.80, p<0.0001) (Fig. 4D). Only very short H1-QRS intervals caused 4- to 6-beat PMVT runs.

The PVCs and non-sustained PMVT were completely eliminated following radiofrequency (RF) catheter ablation at the recording sites of the DPs in the mid-LV septum (Fig. 3C). A total of 14 RF applications were delivered to the area with the earliest DP and around the earliest DP site in which a DP was recorded. During the follow-up period of 96 months, three episodes of ICD discharge were recorded. However, after the administration of oral atenolol and disopyramide, no ICD discharges were observed.

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Figure 3. (A) Surface ECG and intracardiac electrograms demonstrating multiform PVCs (●) and DPs (arrow). The second DP was blocked. (B) Surface ECG and intracardiac electrograms demonstrating a 6-beat run of non-sustained PMVT (●). A Purkinje potential (arrow) preceded each QRS complex of PMVT. Widening of the ventricular activity was observed on the second and fourth beats of NSVT. This finding possibly represents a conduction delay in the myocardium. (C) Catheter position. Fluoroscopic RAO and LAO views of the ablation catheter (arrow) positioned at the left ventricular septum.
QRS duration

Figure 4. (A) Measurement parameters on EPS. ● Indicates a PVC. (B) Relationship between the H0-H1 and H1-DP intervals. No relationships were found between the two parameters. This finding suggests that the Purkinje activation was independent of the preceding normal sinus activation. (C) Relationship between the H1-DP and DP-QRS intervals. An inverse relationship was observed (r = -0.76, p<0.0001). □ Indicates a non-sustained PMVT run with more than four beats. (D) Relationship between the H1-QRS interval and QRS duration of the PVCs. An inverse relationship was observed (r=-0.80, p<0.0001). □ Indicates a non-sustained PMVT run with more than four beats. Only very short H-QRS intervals caused 4- to 6-beat PMVT runs.

and dispersion of refractoriness in the Purkinje network or Purkinje-muscle junction, which may have caused reentrant activity. However, we cannot deny the possibility that focal rapid activity in the Purkinje network caused the PMVT. In any case, the Purkinje network may play an important role in both the initiation and perpetuation of PMVT and subsequent VF.

In this case of idiopathic VF, the Purkinje activity in the LV caused multiform PVCs and PMVT. Abnormal Purkinje activity and slow conduction of the Purkinje fiber and/or the Purkinje-muscle junction were observed in this patient.

The authors state that they have no Conflict of Interest (COI).

References

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