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Case Report

Early transient recurrence of ventricular fibrillation after catheter ablation of premature ventricular contraction from Purkinje fibers in two patients with myocardial infarction

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ABSTRACT

Catheter ablation (CA) targeting premature ventricular contraction (PVC) from Purkinje fibers can be an effective therapy for refractory ventricular fibrillation (VF) after myocardial infarction (MI). We experienced two cases in which catheter ablation targeting PVC initiating VF after percutaneous coronary intervention (PCI) in post-MI patients was effective despite transient early recurrences of VF. The first patient (a 68-year-old woman with MI) developed drug-refractory VF 3 days after PCI to the left anterior descending artery (LAD) and left circumflex artery. CA targeting Purkinje potential preceding PVC at the infarcted area eliminated both the PVCs and VF. Three days after the procedure, the VF attacks relapsed by a different type of PVC. However, the VF responded to conventional treatments and disappeared thereafter. In the second patient (an 83-year-old woman with old MI), refractory VF attacks occurred after PCI to the LAD. CA targeting Purkinje potential preceding two distinct types of PVC successfully suppressed the VF. Although the VF relapsed 2 days after CA, it was suppressed by conventional treatment and disappeared the next day.

<**Learning objective:** We report two patients with MI in whom VF attacks were treated by CA targeting triggering PVCs, and these completely disappeared after the process of transient early VF recurrences. Two cases demonstrate that additional CA may not always be necessary even if VF has relapsed within several days after PVC elimination. When recurrences of VF attacks after the first CA are not so frequent, we will be able to observe patient for several days without performing additional CA.>

Introduction

Ventricular tachycardia (VT) and ventricular fibrillation (VF) after myocardial infarction (MI) are occasionally drug-refractory, repetitive, and lethal. According to some previous studies, catheter ablation (CA) targeting premature ventricular contractions (PVCs), which originate from the Purkinje fiber network and trigger VT/VF, may be effective as an immediate therapy for patients with MI [1].

We herein report two patients with MI and refractory VF attacks in whom early transient recurrence of VF was observed after CA.

Case report

Case 1

A 68-year-old woman was transferred to another hospital because of severe dyspnea. Coronary artery disease was highly suspected as a cause of heart failure based on abnormal electrocardiogram (ECG) findings (poor R progression and ST-segment elevation from V1 to V4) and motion abnormality of the left ventricular (LV) anterior wall on echocardiography. Emergent coronary angiography (CAG) was therefore performed. Severe diffuse stenoses of the left main trunk to left anterior descending

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artery (LAD) and left circumflex artery were observed. Almost complete revascularization was achieved by percutaneous coronary intervention (PCI) with drug-eluting stents to these lesions.

Following PCI, continuous hemodiafiltration and intra-aortic balloon pumping were performed to treat the heart failure and maintain coronary perfusion. Despite improvement in the heart failure, frequent VFs developed 3 days after PCI. Deep sedation, intravenous antiarrhythmic agents (amiodarone, a beta-blocker, and lidocaine), and percutaneous cardiopulmonary support were unable to suppress the VFs for 8 days. The patient was transferred to our hospital to control the refractory VF attacks. On admission to our hospital, an echocardiogram showed severe hypokinesis of the anterior and inferior wall with an LV ejection fraction of 35%. A 12-lead ECG showed poor R progression and ST-segment elevation from V1 to V4 during sinus rhythm and frequent PVCs with a morphology of superior axis and complete right bundle branch block pattern (Fig. 1A), which triggered the VF attacks.

Catheter ablation

We performed CA 11 days after PCI, aiming to eliminate the PVCs triggering VF under the guidance of an electroanatomic mapping system (CARTO; Biosense, Diamond Bar, CA, USA). During mapping around the infarcted area (LV mid-inferior wall), the earliest Purkinje potential (320 ms delay from the onset of QRS) which preceded 80 ms from the onset of the PVC was recorded. PVCs were completely eliminated after several applications of radiofrequency (RF) energy ranging from 30 to 40 W at the site, After CA, conduction delay of 320 ms was not changed, but no further PVC was observed. This finding suggests conduction block between myocardium and VF triggering Purkinje fiber (Fig. 1A). We performed pace mapping at the successful site, but morphology of paced QRS was not identical to that of the triggering PVC.

Post-catheter ablation

After the ablation procedure, the VF and triggering PVC disappeared for 5 days. However, after extubation on the sixth day, VF was triggered by a distinctly different type of PVC and relapsed three times (Fig. 1B, C). Immediately after this event, CAG was performed again, and it did not show significant coronary re-stenosis. Deep sedation with re-intubation was effective without the need for any additional antiarrhythmic agents to suppress the VF. The VF did not recur even after re-extubation. An implantable cardioverter-defibrillator (ICD) was implanted thereafter, and the patient developed no additional VT/VF attacks for 6 years.

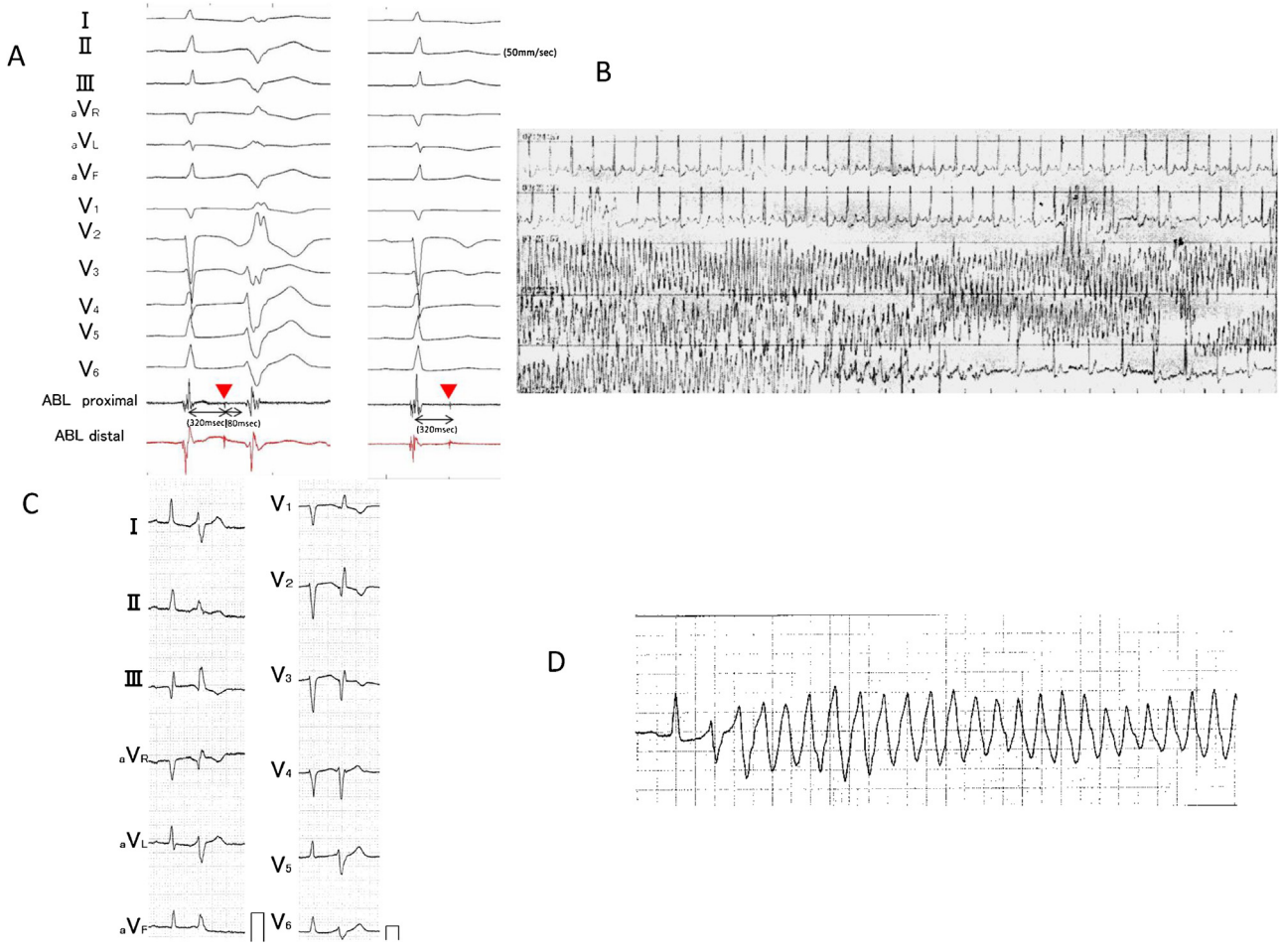

Fig. 1. Case 1. (A) 12-lead electrocardiogram (ECG) shows poor R progression from V1 to V4 during sinus rhythm and frequent premature ventricular contractions (PVCs) that triggered ventricular fibrillation (VF). Catheter ablation at the site with a Purkinje potential preceding the onset of the PVC by 80 ms eliminated the PVCs and VF (left). After catheter ablation, the Purkinje potential appeared with significant delay during sinus rhythm, suggesting disconnection between the Purkinje fibers and myocardium (right). (B) ECG monitoring (corresponds to lead I) at the onset of VF before catheter ablation. (C) A 12-lead ECG showing different type of PVC which triggered VF after catheter ablation. (D) ECG monitoring (corresponds to lead I) at the onset of VF after catheter ablation. VF transiently recurred due to the appearance of another type of PVC after ablation.

Case 2

An 83-year-old woman presented to the emergency department at our hospital because of a 3-month history of progressive shortness of breath. She was diagnosed with heart failure due to recent onset of MI; an ECG showed an abnormal Q wave and ST elevation in V1–V4 (Fig. 2A), and echocardiography demonstrated akinesis with thinning of the anterior LV wall with an ejection fraction of 30%. Her creatine kinase level was within the normal range.

We performed CAG the same day and found severe stenosis in the proximal portion of the LAD. Subsequent PCI was successfully performed on the LAD lesion with a drug-eluting stent. No elevation of creatine phosphokinase was observed after PCI, and her heart failure began to improve with the medical treatment. However, on the second day, VF was repeatedly triggered by two different types of PVCs, and deep sedation, intravenous amiodarone, and a beta-blocker were not effective. Repeated CAG revealed neither restenosis nor new lesions. Fig. 2A and B shows PVC-1 (inferior axis with right bundle branch block pattern) and PVC-2 (inferior axis with left bundle branch block pattern) triggering the VF.

Catheter ablation

We performed CA on the fifth day under the guidance of the CARTO system. During the ablation procedure, both PVCs occurred spontaneously. The electrogram at the infarcted lesion (LV anterolateral wall) showed a sharp presystolic Purkinje potential that preceded the onset of PVC-1 by 46 ms on the surface ECG (Fig. 2C). QRS morphology of pace mapping from this site was not identical to that of triggered PVC. We recorded electrocardiograms at other endocardial sites during sinus rhythm, we could recognize delayed Purkinje potential only at the origin of PVC. During RF energy application at the site, PVC firing along with transient polymorphic VT was observed before complete elimination of PVC-1 with a disappearing Purkinje potential. PVC-2 showed that it originated from the LV apical septum and that the Purkinje potential preceded its onset by 31 ms on the surface ECG. RF energy delivery at the sites eliminated PVC-2. Purkinje potentials during sinus rhythm disappeared after RF energy application at both sites. We delivered RF current ranging from 30 to 40 W at maximum.

Post-catheter ablation

The day after CA, VF triggered by a slightly different type of PVC occurred three times after extubation. Deep sedation effectively suppressed the PVCs and VF without additional medications. Three days later, despite termination of sedation, no further PVCs or VF attacks were observed. The patient and her family declined an ICD. She remained alive with no cardiac events for 2 years.

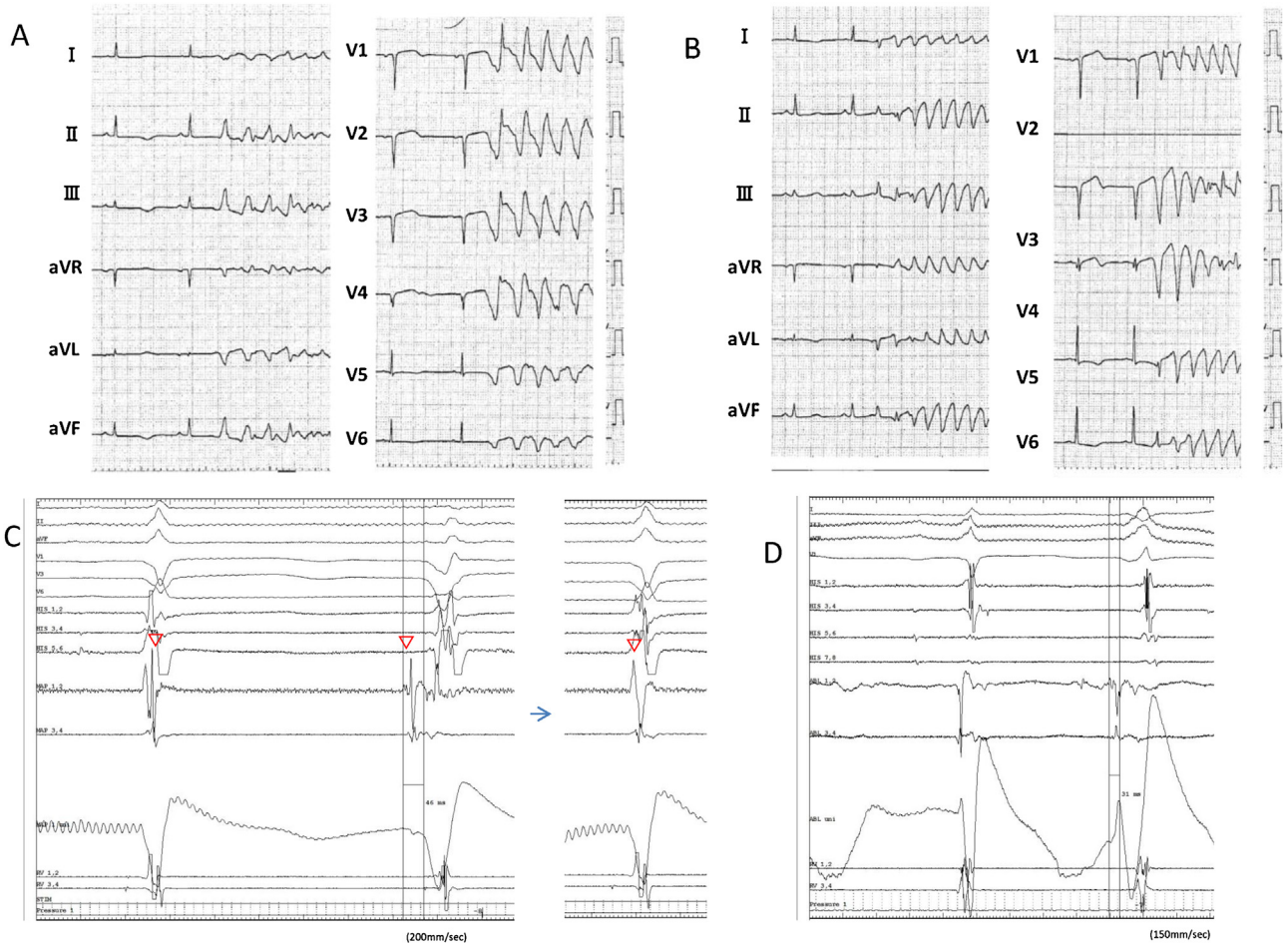

Fig. 2. Case 2. (A, B) 12-lead electrocardiogram leads shows a QS pattern from V1 to V4 during sinus rhythm and ventricular fibrillation (VF) that was induced by two different types of premature ventricular contractions (PVCs). (C) Local electrograms demonstrated a Purkinje potential that was overlapped by a neighboring ventricular potential during sinus rhythm, but it preceded the onset of PVC-1 by 46 ms (left-panel ▽). After catheter ablation at this site, the Purkinje potentials during sinus rhythm disappeared (right-panel ▽), and the PVCs and VF were inhibited. (D) Local electrograms demonstrated a Purkinje potential which preceded 31 ms from the onset of PVC-2 (▽).

Discussion

We have herein described two patients with MI in the post-acute phase in whom frequent and refractory VF attacks were treated by CA targeting the PVCs triggering VF, and these VF attacks completely disappeared after the process of transient early VF recurrences. According to our experience with these two cases, additional CA may not always be necessary even if VF attacks have relapsed within several days after PVC elimination. Relapsed VFs may be prone to respond to medical treatments that had been ineffective before CA and might subside spontaneously.

According to previous research, frequent VT/VF attacks after MI are occasionally triggered by particular PVCs originating from Purkinje fibers and refractory to standard medical therapies [1]. CA aiming to eliminate the PVCs is highly effective to overcome such a critical situation. The cavital blood supply and large amount of glycogen storage allow the Purkinje fibers to be more resistant to ischemia than the neighboring myocardium. The surviving Purkinje fibers adjacent to the infarcted area are prone to give rise to abnormal automaticity (triggered activity) or significant conduction delay, which facilitates formation of a reentrant circuit within the Purkinje network combined with a complex anatomical structure. Creating the lesions using RF energy delivery at the origin of the PVC suppresses arrhythmic activity or disconnects the Purkinje fibers from the PVC origin. In case 1, we demonstrated that the delayed Purkinje potential still existed after successful CA, and the coupling interval of Purkinje potential before and after CA was constant. These observations mean that delayed conduction from neighboring myocardium to Purkinje fiber remained, but conduction from Purkinje fiber to the exit of PVC was eliminated. On the other hand, in case 2, culprit origin of Purkinje potential itself was eliminated by CA. Different responses to CA in the present cases are intriguing, but these can be explained by either mechanism of abnormal automaticity (triggered activity) or reentry.

In our two cases, refractory VF was initiated after revascularization to the culprit lesions of MI in the post-acute phase, and CAG during the VF episodes revealed neither restenosis nor new stenotic lesions. Reperfusion to surviving Purkinje fibers produces various and dramatic changes in electrophysiologic conditions, resulting in abnormal electrical activities such as automaticity or oscillatory afterpotential (triggered activity) [2]. Furthermore, incomplete recovery of a conduction block within the Purkinje fibers may produce heterogeneous propagation of excitation, which promotes the possibility of reentry formation.

Along with disappearance of different types of triggering PVCs, VF attacks were inhibited in both cases. The precise mechanism of transient recurrence of VF attacks (i.e. spontaneous disappearance of relapsed VF) after CA is unclear. In the present cases, the morphologies of the PVCs after CA were different from the previous ones, and the VF eventually responded to standard medical therapies. These phenomena may indicate that the electrophysiologic properties of the PVCs and VF were modified by CA. Multiple RF power delivery close to the critical substrates of VF might create local inflammation, which might facilitate transient proarrhythmic conditions. We sometimes experience early transient recurrence of atrial arrhythmia after pulmonary vein isolation (PVI) for treatment of atrial fibrillation. Most clinical studies evaluating the efficacy of PVI have used a 3-month blanking period because atrial arrhythmias are likely to subside spontaneously early after the PVI procedure [3]. Acute post-ablation inflammation is thought to be responsible for very early (within 7 days) recurrence of atrial arrhythmia after PVI. Our two patients with VF after PCI had clinical manifestations similar to that after PVI, and the same pathophysiological mechanism may be suggested. These two cases indicate that repeat CA may not always be necessary for early relapsed VF after CA targeting PVCs initiating VF in post-MI patients.

Conclusion

We experienced two cases in which CA targeting PVCs initiating VF after PCI in post-MI patients was effective despite transient early relapsed VF.

Conflict of interest

The authors declare that there is no conflict of interest.

References

[1] Ferrier GR, Moffat MP, Lukas A. Possible mechanisms of ventricular arrhythmias elicited by ischemia followed by reperfusion. Studies on isolated canine ventricular tissues. Circ Res 1985;56:184–94.
[2] Nogami A. Purkinje-related arrhythmias. J Arrhythm 2011;27:6–27.
[3] Liang JJ, Dixit S, Santangeli P. Mechanisms and clinical significance of early recurrences of atrial arrhythmias after catheter ablation for atrial fibrillation. World J Cardiol 2016;8:638–46.