Purkinje de-networking using a ripple map with a novel multielectrode mapping catheter for a ventricular fibrillation storm in a patient supported by an Impella device after an acute coronary syndrome

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Introduction

A ventricular fibrillation (VF) storm can follow an acute coronary syndrome (ACS) and is often initiated by premature ventricular contractions (PVCs) from the surviving bundles in the Purkinje system.[1] A previous study showed that Purkinje de-networking (PDN) is an effective rescue approach in patients with a drug-refractory VF storm.[2] However, while PDN may be relatively easy to perform, it poses a risk of complete left bundle branch block (cLBBB), as seen in a previous study where 25% of patients developed new cLBBB during PDN.[3] In this report, we describe a patient with a drug-refractory VF storm following ACS under Impella support in whom we successfully performed PDN using the ripple map (RM) feature integrated into the CARTO 3 system (Biosense Webster, Diamond Bar, CA) to identify the bifurcation of the main left bundle into the left anterior and posterior fascicles.

KEY TEACHING POINTS

- By using the ripple map during Purkinje de-networking, it might be possible to identify the bifurcation point of the left anterior and posterior branches, which could potentially help to prevent complete left bundle branch block.
- The Octaray catheter might be useful for mapping Purkinje potentials precisely and rapidly and might serve as a potential indicator of success by allowing comparison of the inducibility of ventricular fibrillation triggered by movement of the catheter in the left ventricle before and after ablation.
- In preprocedural planning, consideration should be given to whether to perform catheter ablation with Impella support, weighing the primary benefit of hemodynamic assistance against the primary drawbacks of electromagnetic interference. If electromagnetic interference occurs, it can be resolved by decreasing the P level of the Impella device.

Case report

A 58-year-old woman with a history of type 2 diabetes mellitus and dyslipidemia was brought to our hospital with ACS and cardiogenic shock. An emergent coronary angiogram revealed chronic total occlusion of the proximal left anterior descending artery and a 99% stenosis in the proximal portion of left circumflex artery. An Impella CP device (Abiomed, Danvers, MA) was inserted via the left femoral artery before performance of percutaneous coronary intervention (PCI) to stabilize hemodynamics during the procedure. A drug-eluting stent was placed through the left main trunk to the left circumflex artery and a drug-coated balloon was deployed in the left anterior descending artery. TIMI 3 flow was confirmed on completion of the procedure (Figure 1A). After PCI, the patient's hemodynamics showed gradual improvement and were maintained with the Impella CP device set at P-4.

VF occurred during PCI, requiring deep sedation with propofol and fentanyl and continuous intravenous administration of amiodarone. However, even with deep sedation and

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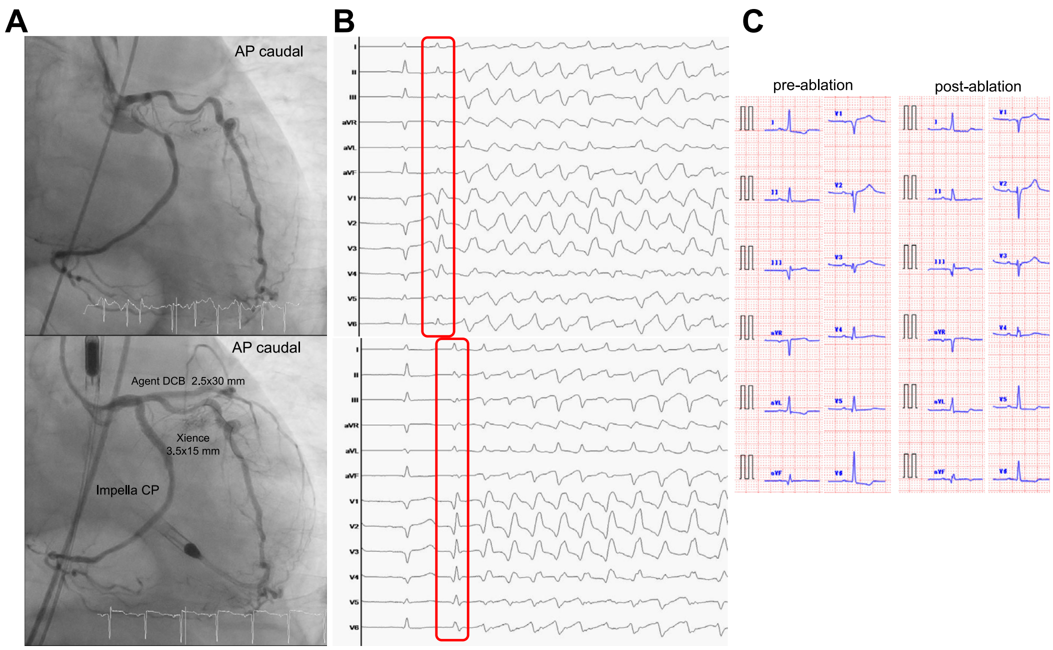

Figure 1 **A:** Coronary angiograms (upper panel, preintervention; lower panel, postintervention). **B:** Twelve-lead electrocardiograms (ECGs) showing 2 representative types of triggered premature ventricular contractions before the ablation procedure. **C:** Pre- and postablation ECGs.

infusion of amiodarone, the patient experienced repeated episodes of VF originating from multiple types of triggered PVCs (Figure 1B), leading to a diagnosis of VF storm on day 5.

Based on their relatively narrow appearance, a monophasic R wave in lead I, a discordant pattern in the inferior leads, and right bundle branch block–like characteristics, we speculated that the triggered PVCs originated from the Purkinje fibers in the left posterior fascicle area. Initially, we attempted right ventricular overdrive pacing using a temporary pacemaker to suppress the PVCs. However, owing to hemodynamic deterioration caused by left ventricular dyssynchrony as a result of right ventricular apical pacing, the decision was taken to treat the VF storm by catheter ablation. The patient's PAINESD score was 20 because she met the criteria for ischemic cardiomyopathy, New York Heart Association class III, VF storm, and diabetes (high-risk category); the PAINESD score categorizes a patient at risk for hemodynamic decompensation during ventricular ablation.[4] Therefore, we decided to perform the catheter ablation while continuing the Impella CP support. The ablation procedure was performed using a transseptal approach because of previous insertion of the Impella CP device via a retrograde transaortic approach.

When the performance of an Impella CP is set at P-4, the device causes magnetic interference with the CARTO 3 system, resulting in an inability to acquire positional information and perform mapping. After lowering the Impella CP setting from P-4 to P-2, we performed endocardial electroanatomical mapping of the left ventricle (LV) during sinus rhythm using an Octaray multielectrode mapping catheter (2-2-2 mm spacing; Biosense Webster) with a visualized steerable sheath (Vizigo sheath; Biosense Webster).

We were able to identify the location where the main branch of the left bundle bifurcates into the left anterior fascicular (LAF) branch and the left posterior fascicular (LPF) branch using the RM (Figure 2A and Supplemental Video 1). We avoided application of radiofrequency energy to the bifurcation point of the LAF and LPF branches identified using the RM, thereby preventing cLBBB. Furthermore, upon examining the voltage map with a setting of 0.5–1.5 mV to distinguish normal tissue from damaged tissue, we observed a low-voltage area in the LPF region (Figure 2A). When the Octaray catheter was brought into the vicinity of this area, VF was readily induced by movement of the catheter. The triggered PVCs displayed a morphology that was similar but not identical to those observed preoperatively. During VF, the Purkinje potential preceded the local myocardial potential (Figure 3A, where the shorter cycle length compared to the local ventricular signal suggests a maintenance Purkinje signal; and Figure 3B, where their same cycle lengths suggest a passive Purkinje signal). Furthermore, in some instances of catheter-induced VF, a prepotential preceding the QRS complex was observed during the triggered PVCs and occurred at the same location where delayed potentials had previously been recorded during sinus rhythm (Figure 3C and 3D).

We performed PDN and intensive ablation in the border zone of the low-voltage area of the LPF region where positioning of the Octaray catheter easily induced VF (Figure 2B). Ablation was performed over 40–60 seconds with a power of 30–35 W. We confirmed noninducibility of VF and ventricular tachycardia (VT) even during movement of the Octaray catheter within the LV, ventricular burst pacing of the LV, and programmed stimulation with up to the

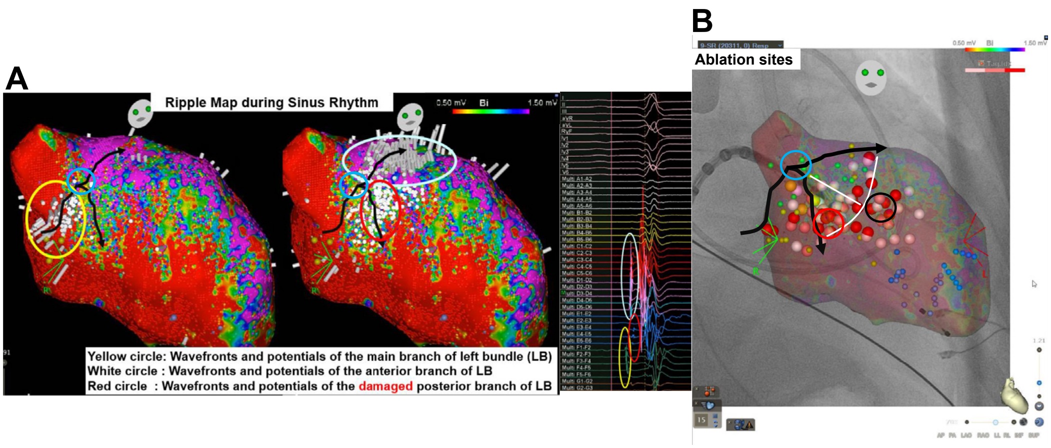

Figure 2 Left ventricular endocardial mapping. **A:** Ripple map (RM) on the endocardial voltage map of the left ventricle. The left panel shows the moment when the ripple bars are present on the left bundle main branch. The right panel shows the moment when the ripple bars are present on the left anterior and posterior branch. The black arrow represents the direction of activation of the His-Purkinje system identified by the RM. The blue circular area represents the bifurcation point of the left anterior and posterior branch. **B:** Location of ablation sites on the left ventricular endocardial voltage map. The white line indicates the location where ablation was performed as Purkinje de-networking. The regions marked with black circles represent the sites where maintenance Purkinje potentials were recorded using the Octaray catheter, leading to relatively extensive ablation toward the apical side.

third extrastimulus, and completed the ablation procedure without any complications, including cLBBB (Figure 1C).

The Impella device was removed on day 8, and the patient was transferred from the intensive care unit to a general ward on day 11. A dual-chamber implantable cardioverter-defibrillator (Resonate EL; Boston Scientific, Marlborough, MA) was placed on day 28 for secondary prevention. The patient was discharged home on day 35. There have been no episodes of VF or VT or of deterioration of heart failure during a year of follow-up.

Discussion

To our knowledge, this is the first report of a combination of PDN and ablation of surviving Purkinje fibers arising from the scar border zone using an RM for a VF storm in a patient supported by an Impella CP device after an ACS.

VF that occurs shortly after a myocardial infarction primarily engages the Purkinje network. During VF, the Purkinje potential precedes the local myocardial potential, and the mechanism is thought to involve triggered activity or random reentry within the Purkinje network. While the precise mechanism via which PDN suppresses VF remains unclear, it is presumed to involve disruption of reentry circuits in the Purkinje network. This disruption may hinder perpetuation of reentrant excitation waves within the network, potentially preventing maintenance of VF.[5]

The following methodology was proposed in a past report on PDN. The left bundle branch and Purkinje potentials should be identified and marked with colored tag points. Next, a virtual triangle is constructed with its apex defined by the proximal left bundle branch and its base delineated by the most distal Purkinje potentials. The anterior and posterior fascicles are represented by the margins of this triangle.[2]

Initially, a linear ablation was performed at the base of the triangle, followed by another ablation perpendicular to the base, aligned with the symmetrical axis of the virtual isosceles triangle, while preserving both the left bundle branch and the 2 fascicles. PDN as an anatomical ablation method does not rely on arrhythmia inducibility and mapping, which can be difficult or not feasible owing to hemodynamic instability. However, it has been reported that 25% of patients who undergo catheter ablation for VF using PDN develop cLBBB.[3]

The RM, incorporated in CARTO 3, is useful for understanding the flow of excitation propagation in the His-Purkinje system because it reflects the actual propagation of excitation without any interpretation of the acquired points on the map. Upon activation of the RM, the direction of activation of the His-Purkinje system, including the bifurcation point of the LAF and LPF, can be clearly delineated before formation of the QRS wave.

In addition to PDN, we performed intensive ablation in the border zone of the low-voltage area of the LPF region. Applying these 2 techniques is beneficial to reduce the risk of VF recurrence. Previous studies found that the culprit Purkinje sources were usually distributed over the border zone of the ischemic scar at the left ventricular septum.[6] This finding is consistent with our observations. Komatsu and colleagues[1] also reported that atrioventricular block and cLBBB occurred in approximately 10% of patients who underwent catheter ablation for a refractory VF storm after myocardial infarction.

In our patient, given that the border zone was also distributed in the proximal portion of the LPF, it was essential to perform ablation while recognizing the distance to the left main branch using the RM. When ablating the border zone of the ischemic scar, identifying the left main branch, LAF, and LPF using the RM might help to prevent occurrence of atrioventricular block and cLBBB.

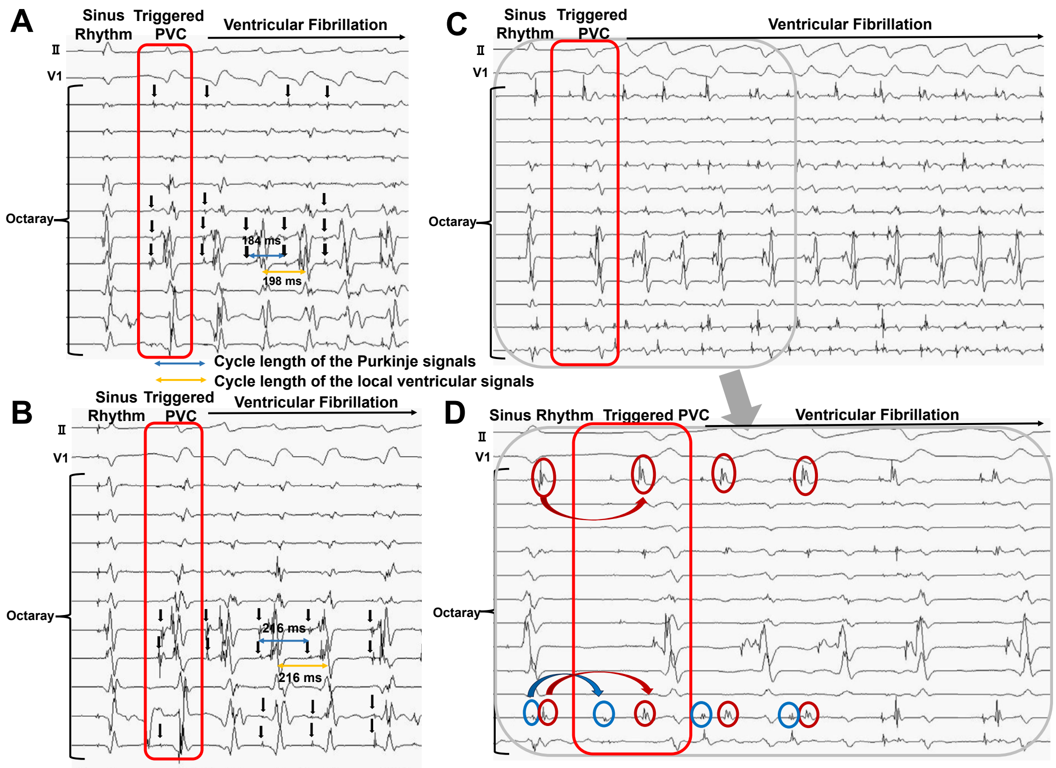

Figure 3 Intracardiac electrograms during catheter-induced or spontaneously occurring ventricular fibrillation. **A, B:** Representative examples of diastolic Purkinje potentials (*black arrow*) during ventricular fibrillation (**A:** maintenance Purkinje signals, indicated by the black circle in Figure 2B; **B:** passive Purkinje signals). **C, D:** A prepotential preceding the QRS complex was observed during triggered premature ventricular contraction at the location where delayed Purkinje potentials were previously recorded during sinus rhythm (indicated by the red circle in Figure 2B). PVC = premature ventricular contraction.

We found that when the Octaray catheter was placed near the culprit Purkinje fibers close to the border zone, it consistently induced a transition from PVC to VF (Figure 3). After ablation, catheter-induced PVC occurred when the Octaray catheter was moved within the LV but did not progress to VF. This observation could potentially be interpreted as an indicator of success. However, there is a concern that runs of VF may be induced mechanically by moving the Octaray catheter within the left ventricle. In cases where a PVC-induced VF storm is deemed hemodynamically unstable, the Optrell catheter (Biosense Webster), if available, can be particularly suitable owing to less ectopy. The timing of introduction of adjunctive circulatory support is a crucial consideration when performing catheter ablation for VT or VF in a patient with structural heart disease. A previous study found that mortality was significantly higher in a rescue Impella insertion group than in a preemptive Impella insertion group and a no insertion group during catheter ablation for VT.[7]

If there are concerns regarding hemodynamic stability, we recommend proceeding with catheter ablation while the Impella device is inserted. Our patient, who was in the acute phase following ACS, was managed in the intensive care unit with Impella CP support, and catheter ablation was performed while maintaining that support.

Electromagnetic interference (EMI) is an important consideration in such procedures. Several studies have reported instances of EMI when performing VT ablations, especially when using a multiaxial flow device (MFD) and magnet-based mapping systems like the CARTO 3.[8] EMI is most often observed when performing mapping in close proximity to the motor of the MFD. In the case of magnet-based mapping systems, the rotational motion of the impeller inside the MFD can generate EMI, potentially compromising accurate localization of catheters, acquisition of mapping points, and integration of respiratory compensation algorithms.[8]

There has been a report of severe EMI being resolved by reducing the performance of the Impella from P-8 to P-6.[9] However, in our case, because of EMI even at P-4, the performance of the Impella was reduced further to P-2 (Supplemental Video 2). Susceptibility to EMI is influenced by the distance between the mapping catheter and the MFD monitor.

The decision whether or not to perform catheter ablation with Impella support should be included in the preprocedural planning, taking into account the primary advantage of hemodynamic assistance, the primary drawbacks of EMI, and the inability to use a transaortic approach.

Conclusion

We have performed catheter ablation using PDN and ablation of the culprit Purkinje fibers arising from the scar border zone under Impella support in a patient with a drug-refractory VF storm after ischemia. This case demonstrates that using an RM to identify the bifurcation point of the LAF and LPF might be beneficial in preventing damage to the conduction system during PDN and ablation of the culprit Purkinje fibers.

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Appendix

Supplementary Data

Supplementary data associated with this article can be found in the online version at https://doi.org/10.1016/j.hrcr.2023.12.011.

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