Successful catheter ablation of ventricular fibrillation storm in acute myocardial infarction by eliminating triggering premature ventricular contraction at the remote site from the exit

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Introduction

Catheter ablation is an effective treatment for drug-refractory postinfarct ventricular fibrillation (VF) storm. Purkinje-related premature ventricular contractions (PVCs) are the main triggers of VF that develop in patients with post–myocardial infarction.[1–4] However, the detailed activation pattern of the Purkinje network preceding PVCs has not been well recognized. The axis of activation is considered to vary from inferior to superior depending on the area of origin from the Purkinje network and the exit site to the myocardium.[4,5] Here, we present a successful case of catheter ablation, targeting a Purkinje-related PVC that originated from the basal anterior left ventricular (LV) septum region and broke out at the apical posterior LV septum via a Purkinje network.

Case report

A 68-year-old male patient developed a VF storm 6 days after hospitalization for heart failure due to acute myocardial infarction. Emergency percutaneous coronary intervention for the left anterior descending artery was performed. Despite optimal medical therapy, including amiodarone, deep sedation, and mechanical cardiac support, VF storm recurred on day 14. As VF was reproducibly triggered by monomorphic PVCs that exhibited a right bundle branch block pattern, northwest axis deviation, and relatively short QRS duration of 127 ms, emergent catheter ablation was planned to target the triggering PVC. The morphology of the triggering PVC suggested the LV posterior septum origin; therefore, a multielectrode linear mapping catheter (DECANAV™; Biosense Webster, Irvine, CA) was placed along the LV posterior septum. The Purkinje potential preceded PVC onset by 150 ms (Figure 1A). The PVC exit was located at the apical LV posterior septum (Figure 1B). We mapped the posterior LV septum to find the Purkinje potential preceding the triggering PVC by 170 ms at the site close to the bifurcation of the left anterior fascicle (LAF) and left posterior fascicles (LPF) (Figure 2A). However, radiofrequency (RF) application at

KEY TEACHING POINTS

- Ventricular fibrillation (VF) storm was successfully treated by catheter ablation of trigger premature ventricular contraction (PVC) by targeting the earliest Purkinje potential preceding the QRS onset by as much as 270 ms.
- The QRS morphology of VF-triggering Purkinje-related PVCs may not necessarily be an excellent clue for approximating its origin. The PVC originated from residual Purkinje tissues at the basal anterior left ventricular (LV) septum and broke out at the apical posterior LV septum 51 mm apart.
- Detailed mapping of His–left posterior fascicle (LPF)–Purkinje potentials with a decapolar mapping catheter may facilitate identification of the origin of the triggering PVCs.
- A chevron pattern observed in the activation sequence of His-LPF-Purkinje potentials could help locate the earliest Purkinje potential at the basal anterior LV septum. This pattern suggested that the origin of triggering PVCs is linked to the branching fascicle or Purkinje fiber from the central LPF.

KEYWORDS Acute myocardial infarction; Catheter ablation; His; Left posterior fascicle; Purkinje; Ventricular premature contraction; Ventricular fibrillation

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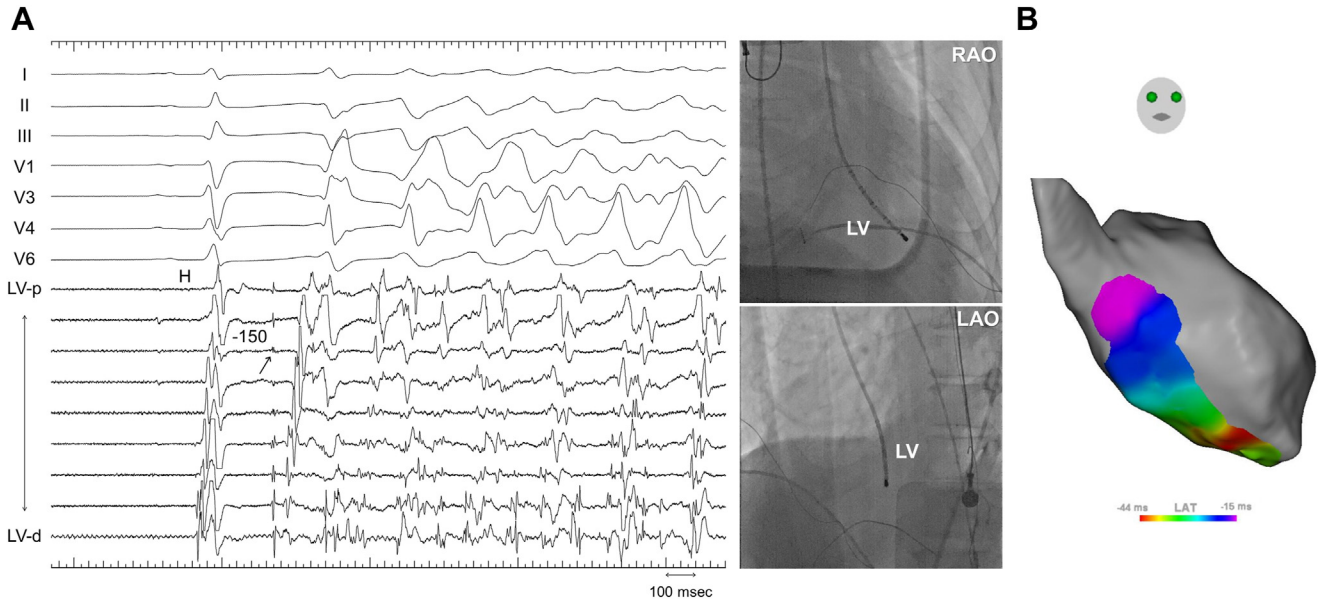

Figure 1 **A:** Electrocardiograms and intracardiac electrograms of the triggering premature ventricular contraction (PVC) before catheter ablation. The earliest Purkinje potential preceded the QRS onset by 150 ms. Fluoroscopy shows the location of a multielectrode linear mapping catheter placed along the left ventricle (LV) posterior septum. **B:** Activation map of the triggering PVC before catheter ablation. The PVC exit was located at the apical LV posterior septum. LAO = left anterior oblique; LAT = local activation time; RAO = right anterior oblique.

this site did not eliminate the triggering PVC. Considering the proximity of the earliest Purkinje and His bundle, we decided against primarily aiming for the earliest Purkinje potential; however, we modified the Purkinje network connecting the foci and exit of the PVC.[4] The Purkinje potentials that guided RF ablation were delivered to the mid-LV septum to transect the Purkinje network. After 13 additional RF applications, the triggering PVC was not suppressed, although minor changes were observed in PVC morphology, with 5 different types (Supplemental Figure 1), and in the activation pattern of the preceding Purkinje potentials. Figure 2B shows a chevron pattern in the distal His-LPF-Purkinje activation, with the earliest site preceding the QRS onset by 180 ms. This pattern suggested that the origin of triggering PVCs was linked to the branching fascicles or Purkinje fibers from the proximal LPF. We changed the strategy again to target the earliest Purkinje potential, which we finally identified to precede the PVC by 270 ms in the LV anteroseptal area at the scar border (Figure 3). RF application at this site eliminated PVCs and completely suppressed VF. The distance between the success site and PVC exit was 51 mm. The patient was free of VF during the subsequent 24 months.

Discussion

This case provides 3 important findings regarding catheter ablation of VF-triggering PVCs in acute ischemic heart disease. First, the Purkinje potentials at the success site

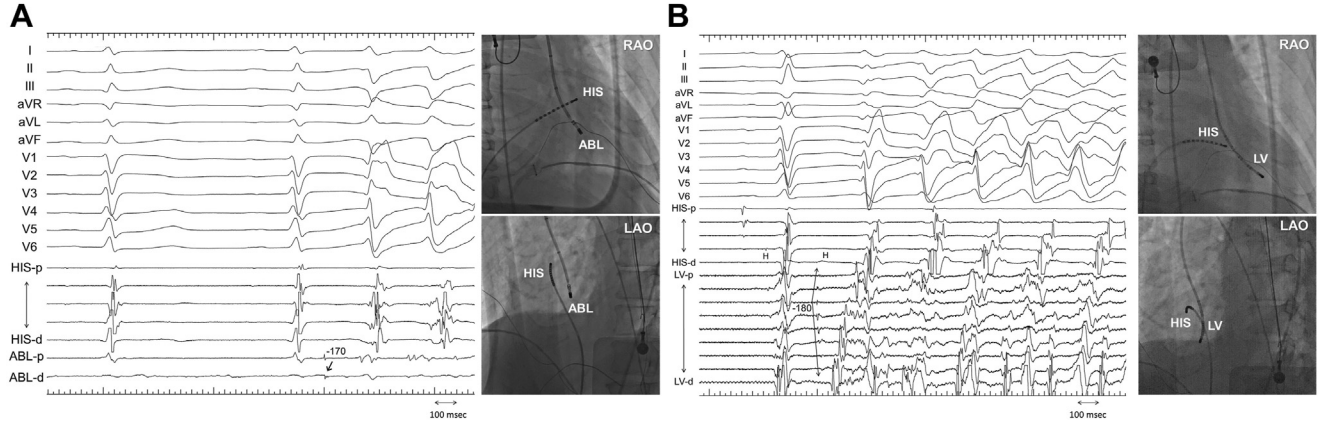

Figure 2 **A:** Twelve-lead electrocardiogram and intracardiac electrograms of the triggering premature ventricular contraction (PVC) at the first radiofrequency application site. The Purkinje potential preceded the triggering PVC by 170 ms. **B:** Twelve-lead electrocardiogram and intracardiac electrograms of the triggering PVC after 14 catheter ablation applications. The triggering PVC was preceded by a chevron pattern in the distal His–left posterior fascicle–Purkinje activation, with the earliest site preceding the QRS onset by 180 ms. Fluoroscopy shows the location of the multielectrode linear mapping catheters. ABL = ablation catheter; H = His potential; His = His catheter; LAO = left anterior oblique; LV = left ventricle; RAO = right anterior oblique.

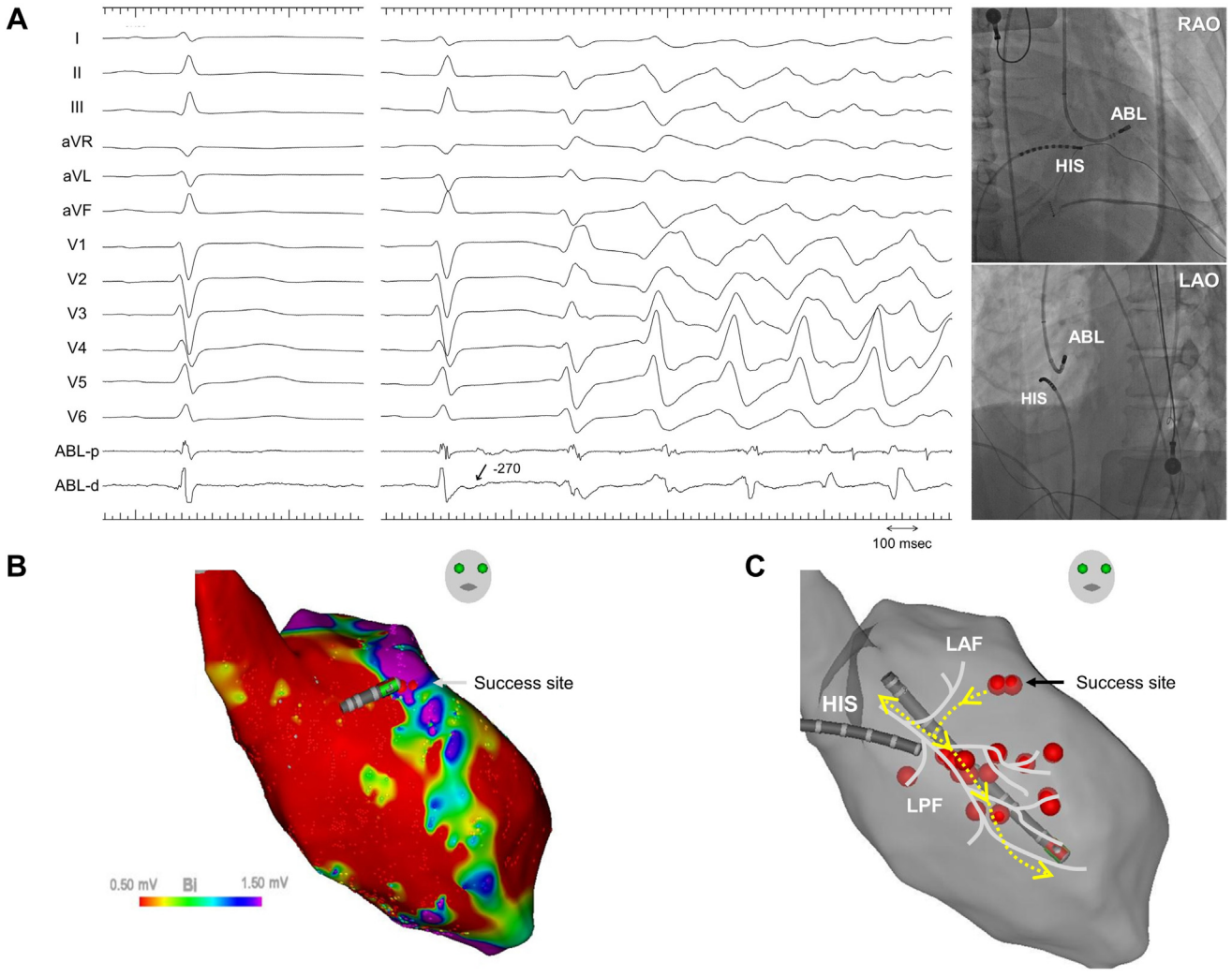

Figure 3 **A:** Twelve-lead electrocardiogram and intracardiac electrograms of the triggering premature ventricular contraction (PVC) at the successful radiofrequency (RF) application site. The Purkinje potential preceded the triggering PVC by 270 ms at the left anterior fascicle (LAF) region where RF application eliminated the PVCs. This abnormal potential could only be recorded before the PVC. **B:** The bipolar voltage map of the left ventricle (LV). The successful ablation site was located at the scar border zone in the voltage map. **C:** Schematic of the proposed PVC circuit. The PVC originated from Purkinje fiber at the basal anterior LV septum, and the impulse retrogradely came into the proximal left posterior fascicle (LPF), traveled via the damaged Purkinje network in the LPF region, and broke out to the apical LV posterior septum while retrogradely activating the His bundle. The yellow dotted line and arrows indicate the presumed pathways that trigger the PVC. Red tags indicate all RF application sites. ABL = ablation catheter; Bi = bipolar voltage map; His = His catheter; LAO = left anterior oblique; RAO = right anterior oblique.

extremely preceded the QRS onset of the triggering PVC, and the success site was located in the basal anterior LV septum 51 mm away from the PVC exit, which was in the apical posterior LV septum. Second, a chevron pattern observed in the activation sequence of His-LPF-Purkinje potentials could help locate the earliest Purkinje potential. Additionally, modification of the Purkinje network between the origin and exit sites did not work.

The earliest Purkinje potentials are considered to be the primary target of catheter ablation.[1–4] The preceding time of the earliest Purkinje potentials was reported to be 126–160 ms[1] and 68 ± 20 ms[2] in previous literature. In this case, we observed an extremely long preceding time of 270 ms at the success site. This difference may be explained by the anatomical distance between the foci and the breakout site of the PVC. The PVC originated from Purkinje fiber at the basal anterior LV septum, and the impulse came into the proximal LPF, traveled via the Purkinje network in the LPF region, and broke out to the apical LV posterior septum while retrogradely activating the His bundle (Figure 3C). The latency existing from the earliest ventricular potential within the scar to the myocardial breakout site may have also contributed to this delay. The QRS morphology of PVC can be an excellent clue for approximating its origin.[4,5] However, this was not the case in the present patient. The PVC morphology was indicative of apical LV posterior septal origin; however, the success site was basal LV anterior septum. The chevron pattern of the distal His-LPF-Purkinje activation (Figure 2B) suggested that the impulse entered the proximal LPF retrogradely via the LAF, intermediate branching fascicles, or Purkinje fibers.[6] Subsequently, the basal LV septum was mapped to identify the earliest Purkinje

potential at the LV anterior site. Since the 12-lead electrocardiogram during sinus rhythm indicated preserved antegrade conduction in the LAF, its retrograde activation with antegrade conduction block exclusively for the triggering PVC was unlikely. Thus, although the triggering PVC originated from a Purkinje fiber in the anteroseptal region located closer to LAF than LPF, it was possibly connected to the proximal LPF via an intermediate branching fascicle and/or the Purkinje network. However, it remains possible that the origin was located in a branch of the LAF and retrogradely activated it without distal antegrade conduction owing to functional block, resulting in the chevron activation pattern. The absence of preceding Purkinje potential at the success site during sinus rhythm may suggest unidirectional block or superimposing of a much greater ventricular potential on the smaller Purkinje potential. It should be noted that the origin of triggering PVCs could be located in an area remote from the exit.

Modification of the Purkinje network between the foci and the exit may be an alternative approach when the earliest site cannot be determined or is located close to the His bundle.[4] However, this approach failed in the present case. Multiple RF applications in the mid posterior septum area only changed PVC morphology. This demonstrates an extended complicated Purkinje network between the proximal LPF and PVC exit, and RF applications in this area only modified the course and exit. Recently, "Purkinje de-networking" has emerged as an optional treatment of VF.[7] The strategy proposed is to ablate all Purkinje potentials between LAF and LPF in the sense of tissue homogenization. The triggering PVC in the present case could have been treated with this technique because it is likely that the success site as well as the conducting pathway to the exit of the PVC were included within this target region.

Conclusion

We report the uncommon case of Purkinje-related PVC-triggered VF in a patient with acute myocardial infarction. Catheter ablation successfully targeted an extremely preceding Purkinje potential recorded in the remote area from the PVC exit.

Appendix

Supplementary Data

Supplementary data associated with this article can be found in the online version at [10.1016/j.hrcr.2023.04.002].

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