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SPOTLIGHT

Idiopathic premature ventricular contractions originating from the distal Purkinje fiber network of the right bundle branch

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A 45-year-old woman with a several-month history of palpitation and progressive dyspnea on exertion was referred to our hospital. At the time of referral, frequent premature ventricular contractions (PVCs) were found in the 12-lead ECG (Figure 1). Echocardiogram indicated a structurally normal heart without valvular disease. Our 24-h Holter ECG showed a high burden of PVCs (26% of all beats), but not couplet PVCs or nonsustained ventricular tachycardia (NSVT). She had no history of syncope or family history of sudden cardiac death. The PVCs had left bundle branch block (LBBB) morphology, inferior axis, and precordial R-wave transition at V5. More specifically, rSR′ in aVL and a QS pattern in aVR and V1 were found in the 12-lead ECG.

Upon the patient's consent, catheter ablation for the PVCs was performed. A 14-polar electrode catheter was introduced from the right femoral vein (RFV) and positioned from the right ventricle apex to the His bundle area. An irrigated contact force (CF) ablation catheter (ThermoCool SmartTouch SF; Biosense Webster) was also inserted into the RFV and advanced into the right ventricle via an 8.5-Fr deflectable sheath (Agilis; St. Jude Medical). A surface ECG

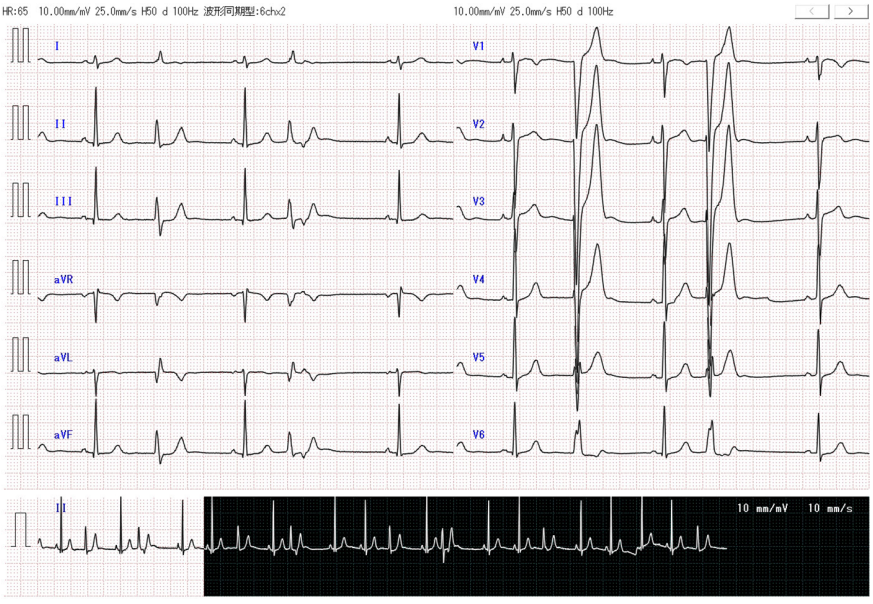

FIGURE 1 12-lead Electrocardiogram (ECG) of the patient before catheter ablation

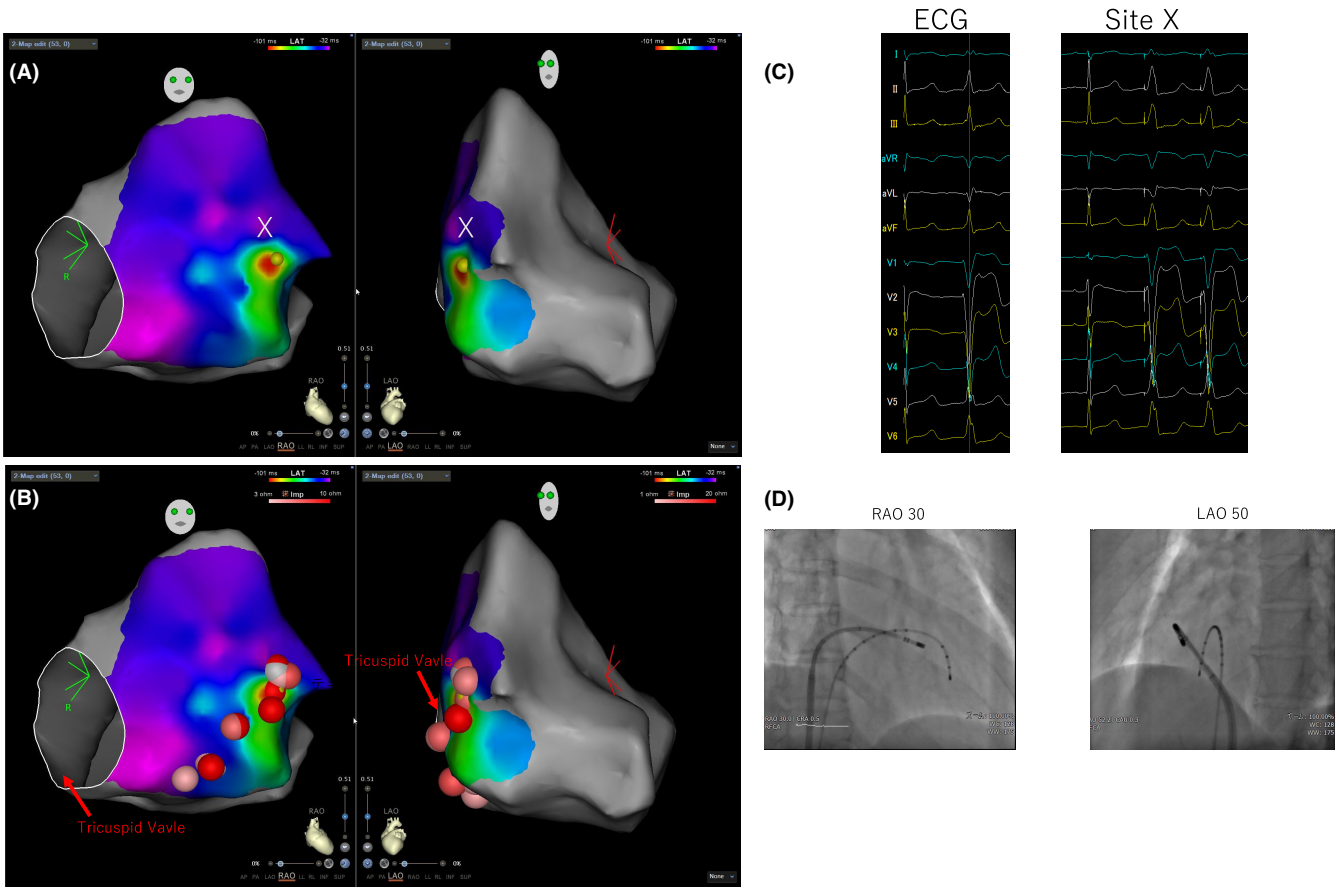

FIGURE 2 CARTO3 activation mapping of the right ventricle. (A) Pace mapping was applied to Site X indicated here. (B) The red squares indicate ablation points in/around Site X. (C) Comparisons between clinical premature ventricular contractions (PVCs) and pace mapping. Left: clinical PVCs; right: pace mapping from Site X. (D) Fluoroscopic images of ablation catheter delivered at Site X. Initial ablation was performed here

and intracardiac electrograms were recorded by the RMC5000 system (Nihon Kohden) during the procedure. During PVCs, activation mapping of the target PVC was created using the CARTO3 system (Biosense Webster). Based on the 12-lead ECG findings, pace mapping was applied to Site X on the free wall of the right ventricle (RV) (Figures 2A,B). This time, pace mapping gained almost *perfect pace map* with 95% similarity according to the PaSo software (Figure 2C). Figure 3A is the surface electrogram and intracardiac electrogram from the ablation catheter at Site X. When the target PVC appeared, the local bipolar activation potential preceded the QRS onset by 31 ms and unipolar potential from the catheter showed QS pattern, confirming that Site X was sufficiently close to the PVC origin. Interestingly, two kinds of isolated Purkinje potentials, 150–160 and 190–200 ms after the QRS onset, were recorded during sinus rhythm (SR) (Figure 3A). This suggests that Site X was located around the distal network of the right bundle branch. We subsequently performed catheter ablation on Site X. Radiofrequency energy from 25 to 40 Watt was applied for 120 s to obtain an impedance drop of 10Ω at Site X (Figure 2D). PVC firing was observed right after the ablation started (Figure 3C). PVCs completely disappeared after 120 s of additional ablation. The post-ablation intracardiac electrocardiogram showed that the Purkinje potentials with small delays (150–160 ms) disappeared, while the ones with large delays (190–200 ms) remained (Figure 3D). This suggests that Purkinje potentials with large delays were bystander potentials. Neither right bundle branch block (RBBB) nor prolonged PQ interval was observed after the procedure. This confirms that the ablation site was located on the distal Purkinje fiber network, not the main branch of the right bundle. After confirming target PVCs could not be provoked even through isoproterenol infusion, our session was ended.

Catheter ablation (CA) has been established as an effective treatment of PVCs. Previous studies report that about 8% of the patients had VT/PVCs originating from the RV inflow (e.g., the tricuspid annulus).[1] A late pericardial transition with an rSR′ in aVL and a QS pattern in aVR and V1 that were found in our 12-lead ECG are characteristics of PVCs arising from the RV inflow; our pace and activation mapping indeed confirmed the origin in the RV inflow.[2]

Activation mapping on the ablation site showed a rare pattern that was not reported in the previous works: local activation potentials (Purkinje potentials) happen much later than the QRS onset during SR (150–160 or 190–200 ms later). PVCs originating from the Purkinje fibers without structural heart disease usually trigger serious prognosis of idiopathic ventricular fibrillation (VF). Previous papers reported that more than 80% of idiopathic VF

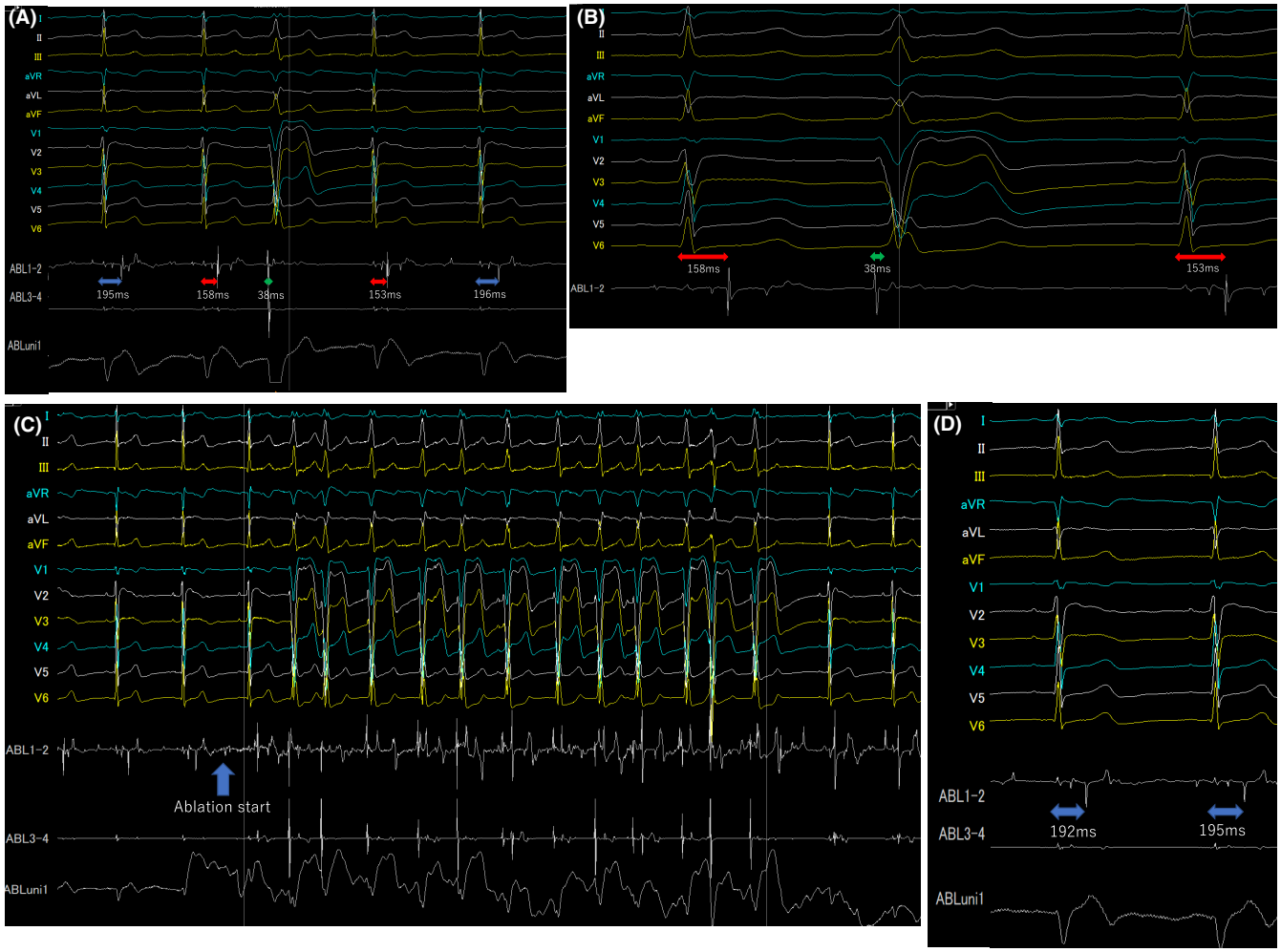

FIGURE 3 (A) Surface electrogram and intracardiac electrogram recorded by the ablation catheter positioned at Site X. (B) Zoom-in figure of Purkinje potentials recorded by the ablation catheter. (C) Intracardiac electrocardiogram right after catheter ablation started. We observed premature ventricular contraction firing. (D) Intracardiac electrocardiogram after catheter ablation

cases originated from the Purkinje fibers.[3] In those cases, Purkinje potentials coincided with or preceded (by around 10 ms) the QRS onset during SR. On the other hand, those potentials preceded the QRS onset by at least 20 ms during PVCs. Catheter ablation could be a useful tool to suppress the recurrence of VF in those cases. To the best of our knowledge, our clinical case illustrated for the first time that idiopathic frequent PVCs without VF provocation were originated from the distal network of the right bundle branch, and Purkinje potentials were recorded later than the QRS onset during SR.

Figure 4A–C illustrates one possible mechanism in the Purkinje fiber network that explain the delay of Purkinje potentials during SR. Similar to a previously reported case,[4] we suspect that Purkinje potentials were delayed during SR because antegrade activation to the ablation site through the proximal Purkinje fibers was blocked (indicated by the purple squares) for some reason (e.g., damage in the Purkinje fibers). Different from the previous case, however, RBBB did not happen because the block was not on the main branch of the right bundle. The ablation site was activated only retrogradely over the distal Purkinje fibers. It is likely that there are two pathways with different lengths: the shorter pathway (Pathway X) corresponds to small delays (150–160 ms), and the longer one (Pathway Y) to large delays (190–200 ms). Purkinje potentials with large delays were only observed after catheter ablation (Figure 3D), indicating that Pathway X was successfully ablated, and PVCs disappeared. This implies that activation during PVCs travels through Pathway X into the intrinsic myocardium, as illustrated in Figure 4 (middle). Indeed, Purkinje potentials preceded the QRS onset by 38 ms because of this mechanism during PVCs (Figure 3A). Comparing these preceding Purkinje potentials during PVCs and the potentials with small delays during SR, we see that the wave forms (Figure 3B) strongly suggest bi-directional potentials around the ablation site: RS-like waves (upward first) during PVCs and QR-like waves (downward first) during SR. This bidirectional nature of the potentials strongly suggests a conduction block in the proximal fibers of Site X, as opposed to a conduction delay. Finally, Figure 4D shows another possibility during SR that has two conduction blocks that are close to each other. Those two conduction blocks would correspond to the two types of delays

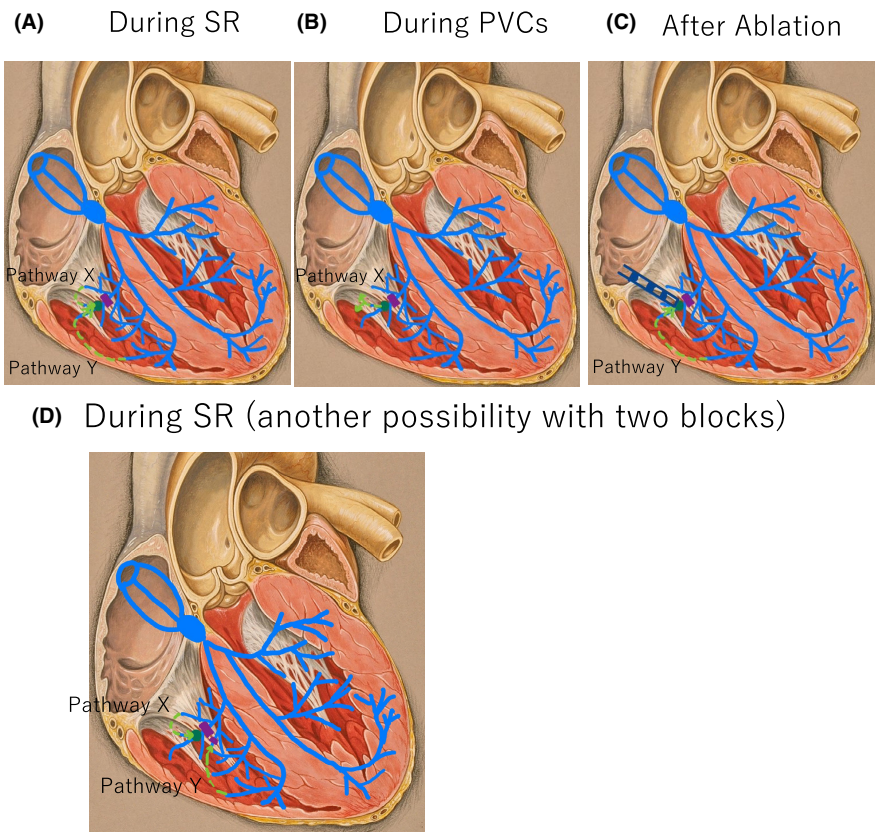

FIGURE 4 Illustration of mechanisms that cause two types of delayed Purkinje potentials. The Purkinje fiber network (drawn in blue) arborizes at the distal sites. The dotted lines denote intramuscular propagation. The purple boxes indicate potential conduction blocks. (A) During sinus rhythm before catheter ablation. (B) During premature ventricular contractions (PVCs). (C) After catheter ablation. Pathways X and Y correspond to small (causing PVCs) and large delays. (D) Another possible mechanism with two conduction blocks

CONFLICT OF INTEREST

The authors declare no conflict of interest for this article.

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