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SPOTLIGHT

Acute occlusion of the left main coronary artery following impedance rise after high-frequency catheter ablation

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A 47-year-old male, with a medical history of depression and hypertension, presented with palpitations. Electrocardiogram (ECG) findings revealed the presence of a premature ventricular complex (PVC; Figure 1), characterized by an inferior axis, right bundle branch block morphology, and a negative lead I, V2 pattern break, indicative of a left ventricular summit origin. Subsequent Holter ECG monitoring documented a PVC comprising 40% of daily beats, prompting hospital admission for catheter ablation due to inadequate response to pharmacotherapy. Transthoracic echocardiography revealed a slight enlargement of the left ventricle by a reduced ejection fraction of 47%. The PVC was spontaneously evident during the electrophysiological study. Advancement of the catheter into the great cardiac vein (GCV) toward the anterior interventricular vein revealed ventricular activation on bipolar electrogram 27 ms preceding the QRS

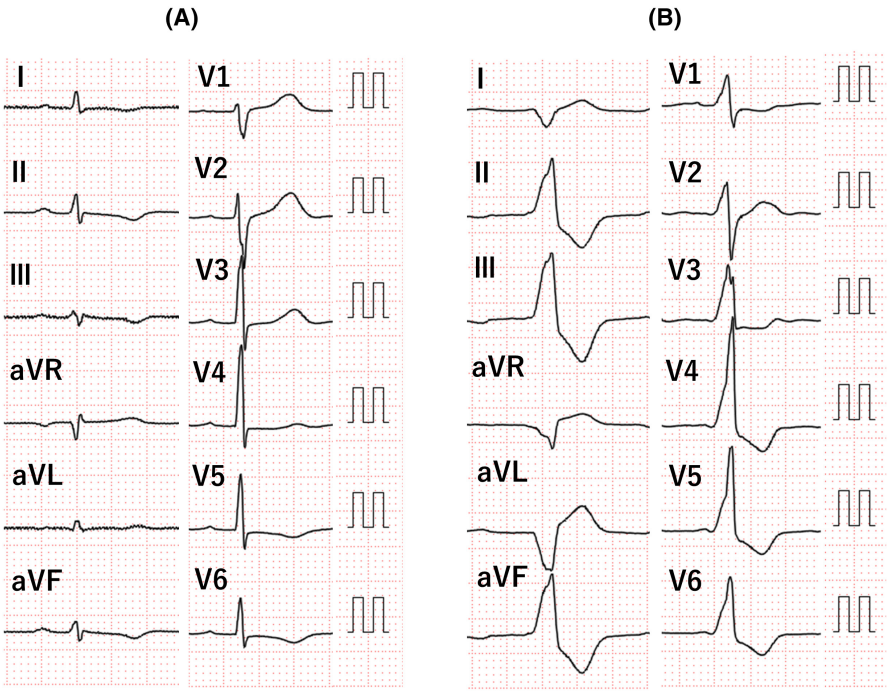

FIGURE 1 Twelve-lead electrocardiograms: (A) Sinus rhythm and (B) premature ventricular tachycardia. Notable observations include an inferior axis and right bundle branch morphology.

onset, accompanied by a unipolar QS pattern (Figure 2A,C). Direct catheter ablation was deemed hazardous due to high resistance at the site, prompting aortic cusp mapping. The earliest PVC activation was localized at the inferior portion of the left coronary cusp (LCC). Although the unipolar electrogram was nonideal (rS morphology), an early bipolar signal was identified (21 ms preceding QRS complex onset), and the site was anatomically opposite to the GCV, where the earliest ventricular activation during the PVCs was recorded (Figure 2B,D). Ablation was performed using a quadripolar catheter equipped with a 3.5-mm irrigated distal electrode, set at 35°C with a peak power output of 30 W. During the radio frequency (RF) application, the premature PVC was transiently terminated but recurred after deactivation of the RF energy. In the third catheter ablation session, ablation was promptly halted due to a sudden impedance increase at 50 s (139 → 245 Ω). In summary, RF was applied to the LCC thrice, cumulatively lasting 153 s, and was associated with an impedance surge.

At 1 min after the final RF application, the patient reported chest discomfort. An emergent coronary angiography (CAG) revealed acute stenosis at the ostium of the left coronary artery (LCA), which remained unresolved by intracoronary nitroglycerin injection (Figure 3A). The ECG indicated ST-segment elevation in the anterior leads. A subsequent coronary angiogram revealed occlusion of the left main artery (Figure 3B). Postangiogram, ventricular fibrillation ensued; defibrillation was executed, followed by the emergency placement of a drug-eluting stent in the left main artery (Figure 3C). Despite stenting, hypotension persisted; a follow-up CAG identified a 99% stenotic lesion within the stent (Figure 3D), which was treated with balloon dilation (Figure 3E). However, stenosis persisted post-dilation (Figure 3F). Later, intravascular ultrasound (IVUS) revealed intimal thickening and tissue protrusion within the stent (Figure 3G). After another balloon dilation of the stent, the patient was monitored in the catheterization lab for 10 min until coronary angiography affirmed the absence of stenosis progression within the stent (Figure 3H). On Postoperative Day 3, the patient was successfully weaned of assisted circulation, and a subsequent CAG revealed no in-stent stenosis. The patient was discharged on Postoperative Day 7. At the next outpatient visit, the burden of PVCs had not changed, and he continued to take beta-blockers and calcium channel blockers. 6 years postprocedure, the patient remains in good health with no evidence of stent restenosis.

In this case, a standard catheter ablation targeting the optimal electrophysiological site of the GCV was abandoned because of anatomical obstacles; thus, ablation from the anatomically opposite site at the LCC cusp was attempted. Impedance escalated during the aortic cusp ablation, leading to a subsequent occlusion of the LCA. Although an emergency stent was implanted, acute occlusion necessitated further balloon dilation.

Impedance rose during aortic cusp ablation in this particular case. The resistance component is affected by the contact degree between the electrode tip and myocardial tissue. As tissue resistivity is approximately double that of blood, cumulative resistance correlates with the tissue–catheter tip interface and increases with greater tissue contact force.[1] Hence, when a catheter

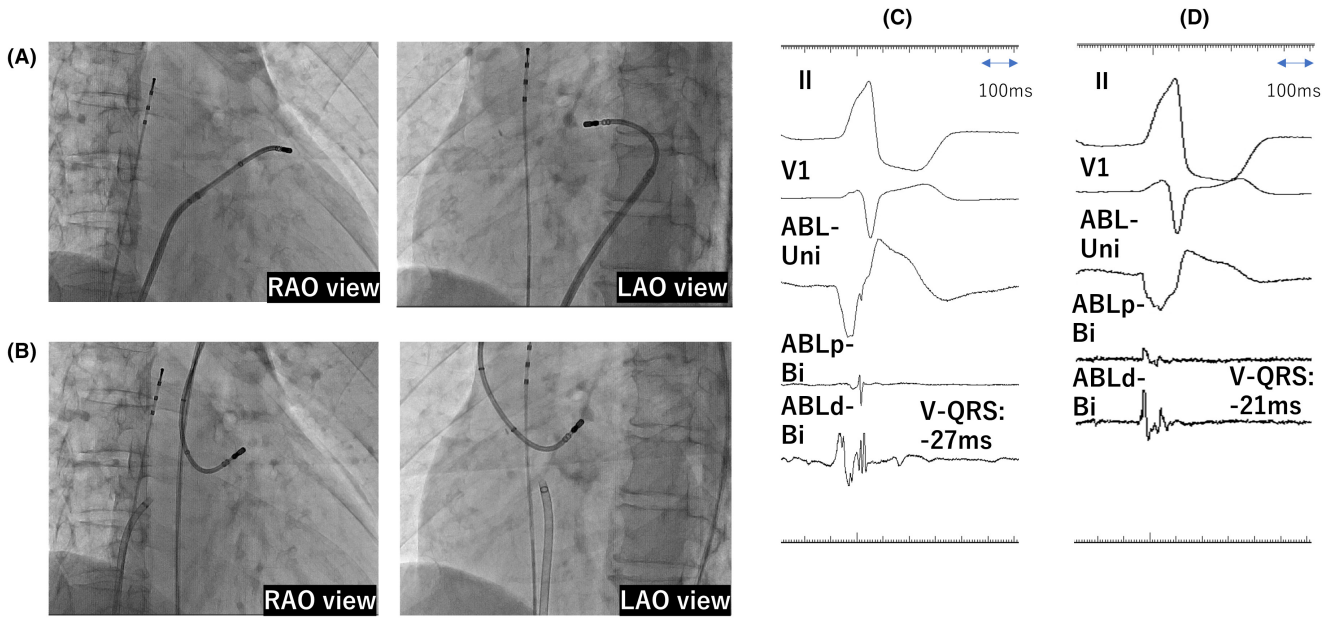

FIGURE 2 Fluoroscopic images of ablation sites and intracardiac electrograms during premature ventricular complexes (PVC): (A) Catheter placement during ablation at the great cardiac vein (GCV). (B) Catheter placement during ablation at the left coronary cusp (LCC). (C) Surface electrocardiographic tracings and intracardiac electrogram from the anterior interventricular vein (AIV) during PVC. The local ventricular activation at the distal bipolar electrode of the catheter revealed an initial upright R wave preceding the onset of the QRS complex by 27 ms, while the simultaneous recording from the unipolar electrode displayed a QS pattern. (D) Surface electrocardiographic tracings and intracardiac electrogram at the LCC during PVC revealed an initial upright R wave at the distal bipolar electrode of the ABL preceding the QRS complex onset by 21 ms. Simultaneously, the unipolar electrode exhibited an rS pattern.

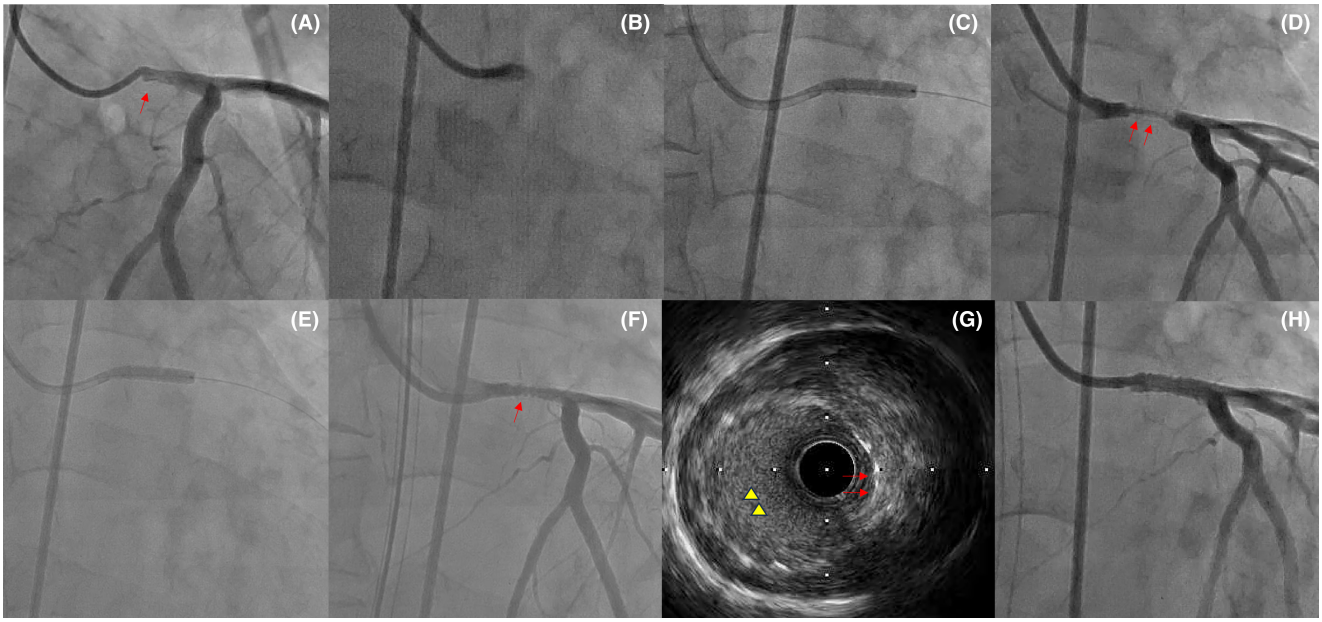

FIGURE 3 (A) Fluoroscopic right anterior oblique view illustrating left main coronary artery (LMCA) stenosis (indicated by a red arrow). (B) Coronary angiography depicting interrupted blood flow in the left coronary artery. (C) Following this, a stent was inserted, leading to restored flow. (D) Coronary angiography shortly after revealed restenosis at the LMCA stent site (red arrow). (E) Subsequent balloon dilation within the stent was conducted. (F) Coronary angiography displayed stent dilation with remaining stenosis (red arrow). (G) Intravascular ultrasound indicated intimal thickening (red arrow) and tissue prolapse within the stent (yellow arrow). (H) Following balloon dilation, coronary blood flow was re-established.

is introduced into a coronary artery, resistance is likely to surge due to enhanced catheter–tissue contact and reduced blood circulation. Elevations in impedance within both saline and blood environments have been linked with tissue charring and disruption of the endocardial surface.[2] Consequently, energy delivery should be promptly halted upon detecting an impedance rise. Two potential causes underlie the occlusion of the LCA. The first is associated with repeated RF application at the LCA ostium, leading to heat conduction and subsequent thermal injury. To prevent damage to the coronary arteries, the ablation site must be at least 8 mm away from the coronary ostium. Therefore, we needed to confirm the location of the ostium by obtaining the left coronary artery imaging before performing the ablation.[3] The second possibility is due to temporary catheter insertion into the LCA. Given the observed impedance rise at the conclusion of the ablation in this instance, the ablation catheter's entry into the coronary artery appears probable. Animal studies have demonstrated that RF ablation (RFA), when administered adjacently and in a parallel manner to the artery, led to lesions confined to the media. However, RFA applied directly and perpendicularly to the artery resulted in marked intimal hyperplasia and intravascular thrombosis.[4] IVUS findings indicated intimal hyperplasia, further suggesting transient catheter insertion into the LCA ostium. To prevent catheter insertion into the coronary artery, it is important to confirm the entrance of the coronary artery using contrast. However, monitoring impedance is also crucial for cusp ablation.

In the present study, stent occlusion was observed poststent placement. Although there have been reports of stenting after ablation-induced coronary artery injury, there have been no reports of acute occlusion after stenting.[5] The cause may be intimal hyperplasia or thrombus formation induced by catheter ablation; thus, careful observation for thrombus formation after stenting is necessary.

This case underscores the necessity of impedance monitoring during aortic cusp ablation and the need for vigilance toward acute occlusion following stent placement due to ablation-induced coronary artery obstruction.

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CONFLICT OF INTEREST STATEMENT

N/A.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available from the corresponding author, TK, upon reasonable request.

ETHICS APPROVAL STATEMENT

N/A.

PATIENT CONSENT STATEMENT

The patient signed the informed consent statement.

CLINICAL TRIAL REGISTRATION

N/A.

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