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CASE REPORT

An Unexpected Complication: Myocardial Infarction Due to Ablation of Left Ventricular Outflow Tract Extrasystole

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Summary

Clinically insignificant premature ventricular complexes (PVCs) are a relatively common arrhythmia that can also be observed in healthy individuals. However, when the frequency of PVCs increases, it may lead to certain clinical symptoms and cardiac abnormalities. While radiofrequency catheter ablation is a highly effective treatment method, its invasive nature can give rise to some undesired complications. This report discusses a rare complication that can occur during PVC ablation and its management.

Keywords: ablation; coronary; premature ventricular complex

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Premature ventricular complexes (PVCs) are a common arrhythmia that can be detected in the majority of healthy individuals using a 48-hour Holter monitor.[1] While PVCs in the absence of underlying structural heart disease are generally considered as benign ventricular arrhythmias, they can also lead to serious adverse outcomes such as cardiomyopathy, impaired quality of life, and sudden cardiac death.[2] PVCs are frequently asymptomatic but may result in symptoms such as palpitations, dyspnea, presyncope, and fatigue. Radiofrequency (RF) catheter ablation is commonly indicated to alleviate symptoms in patients with PVC-induced cardiomyopathy (PIC). Complications of catheter ablation procedures for PVCs are observed in 0% to 5% of cases, primarily attributed to issues related to vascular access.[3] Coronary artery occlusion, as a complication, is quite rare. Herein, we present a case report of acute left anterior descending (LAD) artery thrombosis following RF catheter ablation of PVC, which was successfully treated with percutaneous coronary intervention.

Case Report

A 53-year-old man was admitted to the cardiology outpatient clinic with complaints of palpitations. The electrocardiogram (ECG) revealed sinus rhythm with frequent PVCs in all ECG leads (Figure 1A). Transthoracic echocardiography indicated an ejection fraction of approximately 25%. The patient's history revealed that coronary angiography had been performed three months prior due to left ventricular systolic dysfunction, and non-critical stenosis was detected. Given the presence of frequent PVCs, an existing cardiomyopathy, and the absence of an alternative etiology for the cardiomyopathy, a diagnosis of idiopathic PIC was established. Subsequently, catheter ablation was planned to eliminate the burden of PVCs. The patient was brought to the catheter laboratory after obtaining informed consent. Following the intravenous (i.v.) administration of 8000 units of heparin, a continuous infusion of heparinized saline (1000 U/h) was initiated through a peripheral vein to maintain the activated clotting time (ACT) within the range of 300 to 400 seconds to prevent thrombus formation. ACT values were monitored at 15-minute intervals. Additional intravenous heparin boluses were administered in response to ACT values.

Left ventricular outflow tract region mapping was performed using the CARTO 3 System (Biosense Webster), an electroanatomical navigation system, and a Thermocool Smarttouch catheter. Following the basal ECG, the earliest activation site of the PVC was transaortically mapped to the left coronary cusp of the aortic valve. Radiofrequency energy was applied in this area (35 Watts/60s), resulting in PVC termination. A contact force-sensing catheter was utilized during the ablation procedure, with contact forces ranging from 12 g to 20 g across a total of 8 lesions. Coronary angiography during the ablation procedure revealed no proximity to the left main coronary artery (LMCA) (12.8 mm apart from LMCA) (Figure 1B, 1C). After radiofrequency ablation, the patient was transferred to the coronary intensive care unit for monitoring. The patient received 100 mg of acetylsalicylic acid post-ablation. The post-procedure ECG showed normal sinus rhythm without PVCs. However, 12 hours later, hemodynamically unstable ventricular tachycardia was observed on the patient's monitor, and electrical cardioversion was performed. The ECG revealed ST-segment elevation in precordial leads (Figure 2A). An emergent coronary angiogram confirmed anterior myocardial infarction with acute occlusion of the left anterior descending artery (LAD) (Figure 2B). Intravenous nitroglycerin was initiated to rule out coronary spasm. The thrombotic occlusion was crossed with a guide wire, and a 3.0 x 33 mm everolimus-eluting stent was implanted without complications, restoring TIMI-3 flow (Figure 2C). The patient was then returned to the coronary intensive care unit, where standard medical treatment was initiated, and he was discharged three days later.

Discussion

Multicenter studies have demonstrated a high success rate combined with a low complication rate in catheter ablation, making it the preferred therapeutic option for idiopathic outflow tract ventricular arrhythmias. The success rate of PVC ablation procedures ranges from approximately 80% to 95%.[4] The majority of complications are associated with the vascular access site, such as hematoma, pseudoaneurysm, or arteriovenous fistula. Reported complications of catheter ablation for PVCs include major events at a rate of 2.4%, encompassing aortic dissection, atrioventricular block, cardiac tamponade, damage to the coronary arteries, and neurologic complications.[3]

Different mechanisms can be proposed to explain coronary damage after RF ablation. Ischemic complications during ablation with normal coronary arteries on angiography are thought to result from focal spasm induced by RF energy. Spasm is assumed to be the most common cause, especially when RF energy is delivered within the coronary sinus due to its proximity to the epicardial surface of the heart.[5] However, vasospasm typically occurs during or immediately after the procedure and responds to intravenous nitroglycerin. In our case, the onset of symptoms occurred 12 hours after ablation, and the unresponsiveness to intravenous nitroglycerin suggests that LAD occlusion may occur due to a different mechanism.

The accidental delivery of intracoronary RF current may lead to serious thrombotic complications, such as acute or subacute occlusion. However, in this case, RF energy was not delivered into the coronary arteries. Alternatively, direct trauma to a coronary artery may occur while crossing the aortic valve with the RF catheter, potentially resulting in intimal dissection and thrombus formation.[6] This hypothesis is considered the most suitable explanation for LMCA occlusion, although in our patient, occlusion was observed only in the mid-portion of the LAD artery.

Another explanation for thrombosis is the indirect thermal effect trauma, involving edema and intimal injury due to RF ablation. However, in our case, the distance between the ablation site and the coronary arteries was confirmed through coronary angiography and 3D mapping during the procedure. No significant stenosis was observed in the LAD artery, and it was sufficiently distant from the ablation site. When performing ablations in high-risk areas, a thorough understanding of anatomy and the distance to the LMCA is crucial. Additionally, it is advisable to employ short energy delivery with a power not exceeding 20 W and with low contact force (< 20 grams) to minimize the risk of creating a deep lesion.[7] Embolization of thrombus formation at the RF ablation site into the coronary microcirculation may be another possible mechanism. However, in our case, the procedure was performed under appropriate anticoagulation.[8]

In our case, adrenergic discharge, possibly induced by pain during RF application or in-hospital

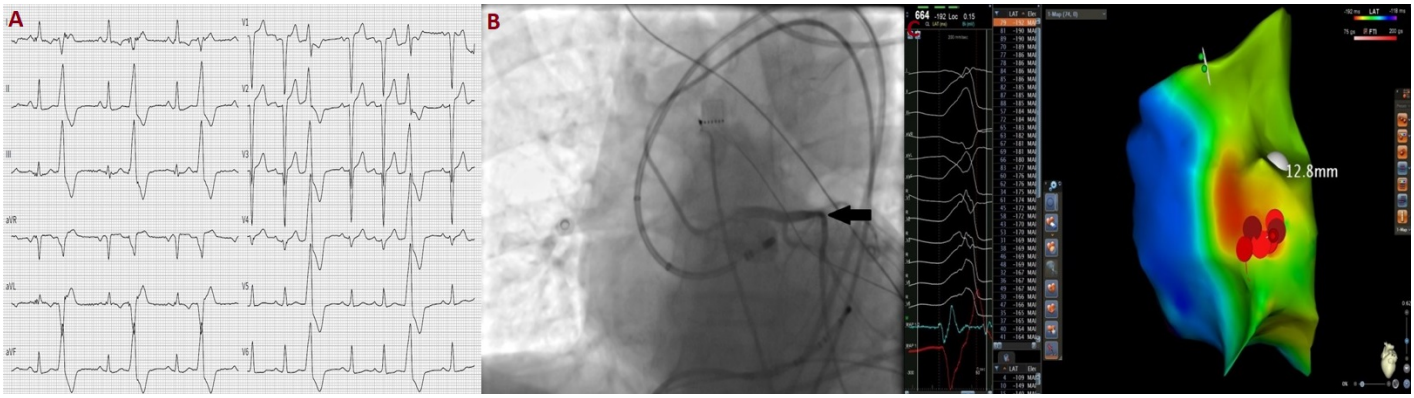

Figure 1. Sinus rhythm with frequent PVCs in all ECG derivations (A). Transaortic approach in electrophysiological mapping. Coronary angiography during the ablation procedure reveals no critical atherosclerotic lesions in either coronary artery. The black arrow indicates an atherosclerotic plaque in the mid-portion of the left anterior descending artery (B). In the 3D mapping, the distance between the left main coronary artery and the radiofrequency ablation catheter was measured as 12.8 mm. The white dot represents the left main coronary artery.
ECG, electrocardiogram; PVCs, premature ventricular complexes

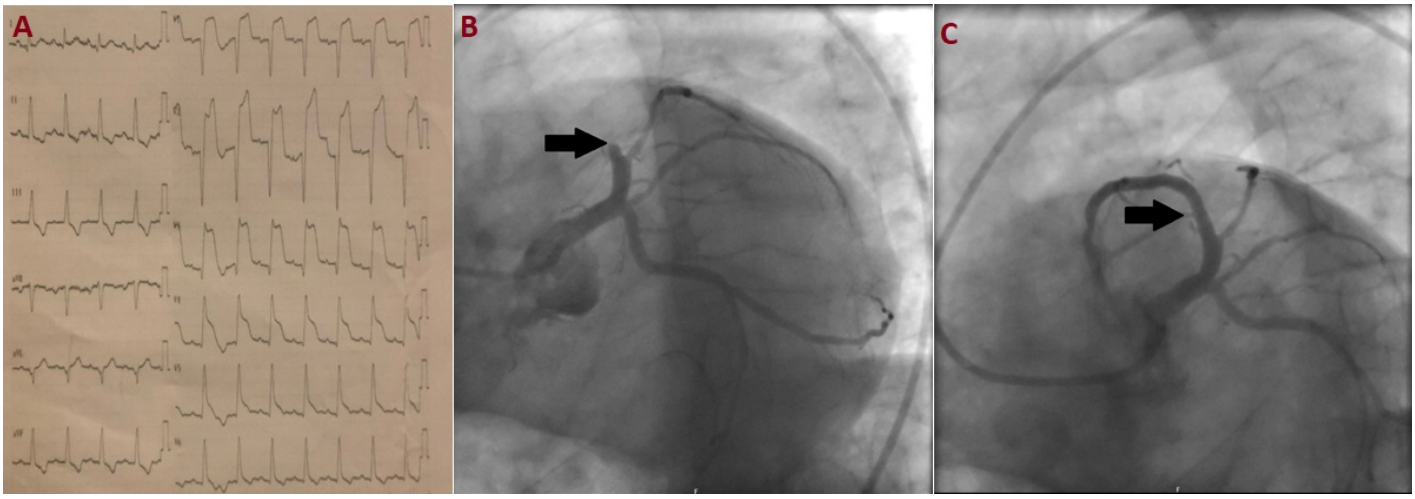

Figure 2. Twelve-lead ECG shows ST elevation in precordial leads (A). Left anterior oblique caudal view of the left coronary angiogram. The black arrow denotes total occlusion in the mid-portion of the left anterior descending artery (B). Left anterior oblique caudal view of the left coronary angiogram. The black arrow denotes TIMI-3 flow after stent implantation (C).
ECG, electrocardiogram

stress-related anxiety, may lead to plaque rupture in the LAD artery, subsequently causing transmural myocardial infarction.

Coronary damage and subsequent myocardial ischemia may be responsible for ventricular arrhythmias or sudden unexplained deaths in the acute and subacute phases after RF ablation. Despite the overall low risk of coronary artery-related complications with RF ablation, this case once again highlights the importance of performing RF applications carefully with minimal catheter manipulation and under appropriate anticoagulation, especially during left-sided procedures. Following left-sided ablation procedures, patients should be monitored for at least 24 hours for potential complications.

Conflict of Interests

None

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References

1- Ng GA. Treating patients with ventricular ectopic beats. Heart. 2006;92(11):1707-1712.

2- Lavalle C, Mariani MV, Piro A, et al. Electrocardiographic features, mapping and ablation of idiopathic outflow tract ventricular arrhythmias. J Interv Card Electrophysiol. 2020;57(2):207-218.

3- Latchamsetty R, Yokokawa M, Morady F, et al. Multicenter Outcomes for Catheter Ablation of Idiopathic Premature Ventricular Complexes. JACC Clin Electrophysiol. 2015;1(3):116-123.

4- Cronin EM, Bogun FM, Maury P, et al. 2019 HRS/EHRA/APHRS/LAHRS expert consensus statement on catheter ablation of ventricular arrhythmias. Heart Rhythm. 2020;17(1):e2-e154.

5- Yalin K, Golcuk E, Bilge AK, Umman S, Adalet K. Successful stenting of a left main coronary artery occlusion as a complication of RF ablation for Wolff-Parkinson-White syndrome. Pacing Clin Electrophysiol. 2012;35(2):e43-46.

6- Hope EJ, Haigney MC, Calkins H, Resar JR. Left main coronary thrombosis after radiofrequency ablation: successful treatment with percutaneous transluminal angioplasty. Am Heart J. 1995;129(6):1217-1219.

7- Benhayon D, Nof E, Chik WW, Marchlinski F. Catheter Ablation in the Right Ventricular Outflow Tract Associated With Occlusion of Left Anterior Descending Coronary Artery. J Cardiovasc Electrophysiol. 2017;28(3):347-350.

8- Dinckal H, Yucel O, Kirilmaz A, Karaca M, Kilicaslan F, Dokumaci B. Left anterior descending coronary artery occlusion after left lateral free wall accessory pathway ablation: what is the possible mechanism? Europace. 2003;5(3):263-266.