

Recurrent and late-onset coronary spasms after cryoballoon ablation procedure in a patient with atrial fibrillation



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Introduction

The feasibility of cryoballoon ablation for treating paroxysmal atrial fibrillation (AF) has been reported. On the other hand, several complications specific to ablation using a cryoballoon may be expected. The prior reports of coronary spasms during procedures describe some mechanisms of coronary spasms; however, those mechanisms remain unknown. We describe a case of recurrent and late-onset coronary spasms after cryoablation procedure that may suggest a possible mechanism not only via the cooling effect on the coronary artery but also via autonomic nervous imbalance by the affected ganglionated plexi located close to the pulmonary veins.

Case report

A 73-year-old man with persistent AF was admitted to our institute for catheter ablation. His persistent AF improved to paroxysmal AF with antiarrhythmic drugs. The patient did not have any coronary risk factors and had bronchial asthma, which was under control with medication. His electrocardiogram (ECG) exhibited AF on admission with a moderate ventricular response of about 75 beats per minute. A transesophageal echocardiogram revealed no intracardiac thrombi.

Catheter ablation using a cryoballoon catheter was performed under moderate sedation with dexmedetomidine. A single transseptal puncture was performed with a radio-frequency needle over an 8 F SL-0 sheath, and then the sheath was exchanged for a Medtronic Flexcath Advance LA 15 F sheath guided by a guidewire inserted into the left atrium. Pulmonary vein (PV) isolation using a 28-mm second-generation cryoballoon (Medtronic, Inc, Minneapolis, MN) was performed in the order of the right superior PV,

right inferior PV, left superior PV (LSPV), and left inferior PV. Each single cryoballoon application was performed for 3 minutes. A total of 2 cryoenergy applications were delivered to both the LSPV and the left inferior PV. In the right inferior PV, a total of 3 cryoenergy applications and 2 additional cryoenergy applications using an 8-mm-tip cryocatheter (Freezor MAX; Medtronic) were needed to complete the PV isolation. Bidirectional linear block of the cavotricuspid isthmus (CTI) was completed with 4 cryoenergy applications for 2 minutes each, using an 8-mm-tip cryocatheter. The whole procedure was performed without any complications. The 12-lead ECG exhibited normal sinus rhythm without any ST changes at the end of the procedure.

While the patient was being kept at rest for 2.5 hours after the ablation procedure, he suddenly developed chest pain, and the 12-lead ECG showed a striking ST elevation in leads II, III, aVF, V₅, and V₆. An intravenous coronary vasodilator immediately improved his chest pain and ST elevation, as shown in [Figure 1](#). Further, after the intravenous vasodilator was stopped 3.5 hours after the procedure, he experienced chest pain again, as well as ST elevation, as shown in [Figure 2](#). After the intravenous vasodilator was restarted, emergent coronary angiography was performed. That coronary angiography revealed no organic coronary stenosis, and no ST elevation was observed. The 12-lead ECG exhibited normal findings the next day. He had no recurrence of angina pectoris for 10 months after the procedure on nicorandil.

Discussion

We describe a case with recurrent late onset of coronary spasms after PV isolation using a cryoballoon and CTI block by a cryocatheter. Electrocardiographic ST elevation and chest pain repeatedly occurred about 3 hours after the procedure. The patient did not have any coronary risk factors, including habitual smoking and beta-blocker agent use, that might have been associated with coronary spasms.

The coronary spasms during the cryoablation procedure were thought to be caused by both direct and indirect effects. Some cases of coronary spasms related to PV isolation by radiofrequency ablation have been reported that suggested

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KEY TEACHING POINTS

- Coronary spasms can occur during cryoballoon ablation in spite of there being no coronary risk.
- Late-onset coronary spasm also could occur repeatedly, which may suggest that its mechanism is through an imbalance in the autonomic nervous system via affected ganglionated plexus located close to pulmonary veins.
- Patients must be monitored not only during the ablation procedure but also after the procedure.

direct thermal effects on the coronary artery and also a possible indirect effect that could induce coronary spasms through the autonomic nervous system.^{1,2} A case of near-fatal left main coronary spasms following a cryoablation procedure was also reported.³ That report suggested the possibility of an indirect effect on the coronary artery because an ST-T change was already observed after starting the first cryoenergy application for the LSPV, which is somewhat distant from the left main coronary artery.

We also previously reported another case of coronary artery spasms during a cryothermal cavotricuspid isthmus ablation and suggested that one of the mechanisms was a direct cooling effect by the cryoablation on the right coronary artery located beneath the CTI.⁴

In this case, the ST elevation did not occur during the CTI ablation procedure using the cryocatheter; however, ST elevation occurred 2.5 hours after the entire ablation procedure had ended. Furthermore, the ST elevation recurred again after the patient was taken off the vasodilator 3.5 hours after the procedure. This case reports the late and recurrent onset of ST elevation with chest pain, which may suggest that the possible mechanism of the coronary spasm was attributable not only to the direct effect of the cooling on the coronary artery but also to the indirect effect of an imbalance in the autonomic nervous system. Inflammation after a cryoenergy application may induce an imbalance in the autonomic nervous system. The autonomic change has been previously reported^{5–8} as a mechanism of coronary spasms with a dynamic fluctuation in the vagal activity and following sympathetic nervous system activity via the affected ganglionated plexus located close to the pulmonary veins.

The relationship between a vagal response and ablation procedures involving the LSPV has been previously reported, and the incidence of a vagal response during a cryoballoon ablation is higher than that during radiofrequency ablation.^{9,10} However, in this case the patient did not have any atrioventricular block or asystole during the cryoablation procedure of the left PV. We previously reported¹¹ a reasonable approach during cryoablation targeting the right PVs prior to the LSPV to prevent any vagal response, including asystole, during the ablation of the LSPV. We also performed the cryoablation of the LSPV

following the right PVs to prevent any bradycardia during the procedure in this case.

The other mechanism was thought to be a direct and/or indirect effect of cooling on the coronary artery by cryoenergy application. Coronary spasms may be induced by cold stimulation. According to previous reports,^{12–14} the sensitivity of the cold pressor test is reported to be 11%–33% of variant angina. However, it is unknown whether or not coronary spasms could be provoked by the specific cooling effect of the cryoablation.

An air embolism occurring during the procedure could be one of the causes of ST elevation during a procedure. Care was taken to prevent an air embolism from occurring during the cryoballoon ablation procedure. In this case, we did not find any air bubbles in the heart on fluoroscopy with contrast media. Furthermore, the patient's chest pain and ST elevation occurred 3 hours after the procedure, when he was still at rest in a supine position. We believe that an air embolism as the cause of the coronary spasms was unlikely in this case.

Dexmedetomidine could be one of the factors affecting the coronary spasms. Dexmedetomidine has an agonist effect in central alpha 2-adrenergic receptors, which may pharmacologically induce a vasoconstriction; however, the incidence of coronary spasms induced by dexmedetomidine is rare. We usually use dexmedetomidine for mild sedation during cryoablation procedures. In this case, we started with dexmedetomidine 4–6 $\mu\text{g/kg/h}$ for 10 minutes at the beginning of the cryoablation, and then a dose of 0.6 $\mu\text{g/kg/h}$ was maintained to obtain mild sedation during the procedure. The patient's chest pain occurred more than 3 hours after the dexmedetomidine had been stopped and he had already awakened from the anesthetic by the end of the procedure. Because the half-life of dexmedetomidine is 2.39 ± 0.71 hours, the influence of dexmedetomidine on his coronary spasms might have been small.

Although there are some mechanisms responsible for inducing coronary spasms, it is also important to recognize the racial differences in the prevalence of coronary artery spasms. It has been previously reported that the susceptibility to coronary spasms is more frequent in Asian countries, and multivessel spasms are especially more frequent and there is a higher inducibility of coronary spasms with acetylcholine and ergonovine injections than in Western countries.^{15–18} Further investigation and a larger investigation of the incidence of coronary spasms solely during cryoablation procedures may be needed.

We report a case of a late and recurrent onset of ST elevation associated with chest pain after a cryoballoon ablation procedure. Given the relationship between the time course and mechanism involving cryolesion tissue injury,¹⁹ the inflammation after the cryoenergy application around the ganglionated plexus may have induced an imbalance of the autonomic nervous system, which could have been the mechanism of the coronary spasms after the cryoballoon ablation procedure.

Conclusion

This case highlighted that it is possible for coronary spasms to occur during cryoablation procedures in spite of there

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