Ventricular fibrillation associated with multi-vessel coronary spasms following radiofrequency ablation of atrial fibrillation and atrial flutter

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A 62-year-old male developed ventricular fibrillation associated with multi-vessel coronary spasms of the right coronary artery and left circumflex artery following a successful pulmonary vein isolation and cavotricuspid isthmus radiofrequency ablation procedure. Ventricular fibrillation should be considered as a life-threatening complication in patients with a history of vasospastic angina.

Case reports
A 62-year-old man was admitted for radiofrequency (RF) ablation of symptomatic paroxysmal atrial fibrillation and typical atrial flutter refractory to medical treatment. He had a history of syncope diagnosed as vasospastic angina pectoralis (VSAP) according to symptomatic ST changes that occurred during a provocation test of the coronary arteries 3 years before. He was free from any symptoms after taking

Figure 1  Coronary angiography. Severe stenoses were observed in the RCA (A) and LCX (B) and they improved with the administration of nitroglycerin (C and D).
calcium antagonists and nicorandil. He also took an antihypertensive drug and was a current smoker. Upon admission to our hospital for RF ablation, an echocardiogram revealed a structurally normal heart. Oral antianginal drugs were continued and sedation was maintained with the intravenous administration of dexmedetomidine hydrochloride during the ablation procedure. The RF ablation was performed with an 8 mm tip catheter, and the RF energy was delivered at a power setting of 25–35 W with a maximum temperature of 55°C. All four pulmonary veins (PVs) were successfully isolated and a bidirectional cavotricuspid isthmus (CTI) block was confirmed. He developed bradycardia transiently during the ablation, but no ST changes were observed. On the way to the medical ward after the procedure, he suffered a cardiopulmonary arrest. Cardiopulmonary resuscitation was started. Ventricular fibrillation (VF) was confirmed and terminated with a 300 J shock. Immediately after the cardioversion, the 12-lead electrocardiogram showed sinus tachycardia without any ST elevation, but 10 min later, it revealed ST elevation in Leads I, II, III, aVF, aVL, and V3–6, and non-sustained ventricular tachycardia. The intravenous administration of nitrates was started and the ST elevation improved. An urgent coronary angiography was performed, and it revealed severe stenosis of the right coronary artery (RCA) and left circumflex artery (LCX). An intracoronary bolus of nitroglycerin induced vasodilation of the coronary artery (Figure 1). He recovered without any neurological sequelae and received an implantable cardioverter-defibrillator for secondary prevention.

Discussion
Several mechanisms of the coronary artery spasms after the RF ablation were assumed. One possible mechanism is that the direct thermal trauma from the RF energy might have damaged the coronary artery and lead to coronary vasospasms. In the present case, the site of the RCA spasms was close to the site of the CTI ablation, but the site of the LCX was far from the site of the PV ablation. An intravenous nitrate infusion was administered before the coronary angiography, so we might have underestimated the extent of the coronary vasospasms.

The use of dexmedetomidine hydrochloride, which stimulates the α2 receptors and strain vagal tone, was probably related to the induction of the coronary artery spasms. Although α2 agonists do not induce constrictions of normal coronary arteries, they induce constrictions of the atherosclerotic segments and lead to a decrease in the coronary blood flow and the initiation of myocardial ischaemia in patients with endothelial dysfunction and established coronary artery disease.

Another mechanism may be autonomic imbalance-mediated coronary spasms. Coronary ganglionated subplexuses mainly come from the ganglia between the aorta and the pulmonary trunk, but partially from the para-sinoatrial ganglia, which are located primarily in the region lateral to the right PVs. Therefore, the RF energy applied during a PVI ablation might affect the ganglia and promote vasoconstrictions of the coronary arteries.

We should carefully monitor during the peri-operative period for the risk of any VF occurrence and consider administering an intravenous injection of nitroglycerin if patients have a past history of VSAP. The use of dexmedetomidine hydrochloride should be avoided in such a situation.

Conflict of interest: none declared.

References