Case Report

Three cases of vasospastic angina following catheter ablation of atrial fibrillation

Yoshihiko Kagawa, Eitaro Fujii*, Satoshi Fujita, Norikazu Yamada, Masaaki Ito

Department of Cardiology and Nephrology, Mie University Graduate School of Medicine, 2-174 Edobashi, Tsu, Mie 514-8507, Japan

ABSTRACT

Pulmonary vein isolation is an effective treatment for patients with atrial fibrillation (AF). Although vasospastic angina (VSA) is not a common complication after ablation of AF, we report 3 cases of VSA following ablation of persistent AF. Two of the 3 patients felt chest pain following pulmonary vein isolation, and complex fractionated atrial electrogram ablations were performed. ST elevation in the inferior leads and atrioventricular block occurred because of severe coronary vasospasm. In the third patient, the electrocardiography monitor detected transient ST elevation within an hour after ablation. Treatment of VSA may be required following catheter ablation of AF.

© 2017 Japanese Heart Rhythm Society. Published by Elsevier B.V. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

1. Introduction

The safety of catheter ablation of atrial fibrillation (AF) has been established. Nevertheless, the following cases demonstrate that we must remain vigilant regarding the occurrence of vasospastic angina (VSA) after ablation of persistent AF.

2. Case report

2.1. Case 1

A 53-year-old man was referred to our hospital for radiofrequency ablation of persistent AF. He underwent extensive pulmonary vein isolation (EPVI) and complex fractionated atrial electrogram (CFAE) ablation. At the end of the procedure, he felt chest pain with ST segment elevation and advanced atrioventricular block (Fig. 1). Emergency coronary angiography demonstrated coronary vasospasm in the multi-vessels (Fig. 2). After injection of intravenous nitroglycerin, impressive vasodilation of all vessels was observed.

2.2. Case 2

A 69-year-old man with a history of hypertension was diagnosed as having persistent AF for 6 months. EPVI and CFAE ablation were performed. During CFAE ablation, electrocardiography demonstrated ST elevation (Fig. 1) followed by complete atrioventricular block and cardiac arrest. Emergency coronary angiography demonstrated narrowing of multi-vessels, and vasodilation was observed after applying nitroglycerin.

2.3. Case 3

Four years ago, a 68-year-old man underwent EPVI and CFAE ablation of persistent AF. At that time, he felt chest pain, but coronary angiography revealed no stenotic lesions. He was admitted to our hospital for ablation of recurrent persistent AF. EPVI, CFAE ablation, and superior vena cava (SVC) isolation were performed because the SVC was identified as a trigger for the AF. One hour after ablation, the electrocardiography monitor detected ST segment elevation for 3 minutes, without any other symptoms (Fig. 1). The provocative test using intracoronary ergonovine maleate injection, which was performed 2 days after ablation, demonstrated VSA (Fig. 2). He was treated with a calcium-channel blocker and had no chest pain in the follow up period.

3. Discussion

Tada et al. first reported VSA within 12 days after paroxysmal AF ablation [1]. Although VSA is not a typical complication following AF ablation, the cases above also show that we should be ready to treat it [2]. An air embolism or a thrombosis of the coronary artery may cause ST-segment elevation. However, the coronary angiography did not demonstrate either air emboli or thrombi in our cases. Coronary vasospasm may be caused by the imbalance of the autonomic nervous system after ablation rather than a thrombotic event.
than by indirect thermal trauma from radiofrequency energy applications near the coronary artery.

Yamashita et al. reported that catheter ablation promotes vasoconstriction of the right coronary artery, which might precipitate VSA. They evaluated 20 patients who underwent left atrial catheter ablation and found that the basal minimal lesion diameter of the proximal segment of the coronary artery was smaller, and the basal tone was greater, after ablation in the right coronary artery [3]. Marked changes in autonomic nervous activity and imbalances between sympathetic and parasympathetic activities after left atrial catheter ablation might cause vasoconstriction or vasospasm of the right coronary artery.

Additionally, patients with paroxysmal AF may coincidentally exhibit coronary artery spasm. Kawakami et al. evaluated 17 patients with paroxysmal AF (AF group) and 34 patients without paroxysmal AF (control group). Coronary artery spasm was induced in 13 patients (76.5%) before AF ablation in the AF group, while being induced in only 3 patients (8.8%) in the control group [4].

Finally, dexmedetomidine is an alpha-2-adrenoreceptor agonist that possesses sedative, analgesic, and anxiolytic properties, and has been used during procedures in our patients. Alpha-2-adrenoreceptors-mediated vasoconstriction can involve coronary circulation, especially in the presence of atherosclerosis and endothelial dysfunction and high sympathetic tone [5]. Therefore, it is
possible that stimulation of alpha-2-adrenoreceptors using dexmedetomidine may lead to vasospasm.

4. Conclusion

Coronary vasospasm may occur following ablation of persistent AF. Early detection of ST elevation is necessary to avoid hemodynamic collapse.

Conflict of interest

All authors declare no conflict of interest related to this study.

References

[1] Tada H, Naito S, Oshima S, et al. Vasospastic angina shortly after left atrial catheter ablation for atrial fibrillation. Heart Rythm 2005;2:867–70.
[2] Nishizaki M Life-threatening arrhythmias leading to syncope in patients with vasospastic angina. J Arrhythm, 2017, in press.
[3] Yamashita E, Tada H, Tadokoro K, et al. Left atrial catheter ablation promotes vasoconstriction of the right coronary artery. Pacing Clin Electrophysiol 2007;30:998–102.
[4] Kawakami T, Ohno H, Tanaka N, et al. The relationship between paroxysmal atrial fibrillation and coronary artery spasm. Pacing Clin Electrophysiol 2014;37:591–6.
[5] Heusch G, Baumgart D, Camici P, et al. alpha-adrenergic coronary vasoconstriction and myocardial ischemia in humans. Circulation 2000;101:689–94.