Journal of Cardiology Cases 20 (2019) 221–224

Contents lists available at ScienceDirect

Journal of Cardiology Cases

journal homepage: www.elsevier.com/locate/jccase

Case Report

Coronary vasospasm caused by intravenous infusion of dexmedetomidine: Unrecognized pitfall of catheter ablation procedures of atrial fibrillation

Koichi Furui (MD), Itsuro Morishima (MD, PhD)*, Yasunori Kanzaki (MD), Hideyuki Tsuboi (MD, PhD, FJCC)

Department of Cardiology, Ogaki Municipal Hospital, Ogaki, Japan

ARTICLE INFO

Article history:
Received 19 February 2019
Received in revised form 14 July 2019
Accepted 16 August 2019

Keywords:
Atrial fibrillation
Catheter ablation
Coronary artery
Vasospasm
Sedation
Dexmedetomidine

ABSTRACT

Coronary vasospasm is an emerging potentially lethal complication of catheter ablation for atrial fibrillation (AF), however, its mechanism in this setting has not been well elucidated.

A 55-year-old man with symptomatic paroxysmal AF underwent pulmonary vein isolation under sedation with propofol. The procedure was completed without any complications. Fifteen months later, a repeated session was performed because of AF recurrence. Initially, a high-dose infusion of dexmedetomidine instead of propofol was administered to introduce sedation. Then, an ST-segment elevation developed in the inferior leads and the diagnosis of coronary vasospasm was made by urgent coronary angiography. A comparison of the procedural details between the first and second sessions identified dexmedetomidine, an α-2 adrenergic agonist with a short distribution half-life, as a potential cause of coronary vasospasm seen only in the second session in the same individual. Since it has been shown that α-2 adrenoreceptor-mediated vasoconstriction can involve the coronary circulation, it is thus possible that a stimulation of α-2 adrenergic receptors induced by dexmedetomidine caused a coronary vasospasm.

The present case provides new insights into dexmedetomidine-induced vasospasm. Physicians should be aware of this potentially lethal side effect of dexmedetomidine which is increasingly used in the current AF ablation practice.

<**Learning objective:** Dexmedetomidine has become widely used during catheter ablation for atrial fibrillation since it is generally regarded as a safe drug for sedation and analgesia with fewer respiratory depressant effects compared to other agents. However, it should be noted that dexmedetomidine may cause a coronary vasospasm, especially at the time of an initial high loading-dose infusion. Physicians should be aware of this potentially lethal side effect of dexmedetomidine.>

Introduction

In contemporary catheter ablation procedures for atrial fibrillation (AF), sedation is typically required because patients undergoing the procedures must stay immobile on the table for several hours even though they may experience occasional pain due to repeated stimulation from the ablation. Among the available sedative drugs, dexmedetomidine—an α-2 adrenergic agonist with a short distribution half-life—has been deemed a safe sedative drug providing sedation and analgesia with less respiratory depressant effects compared to other agents [1,2]. Regarding the potential complications of catheter ablation, case reports demonstrating coronary spasm as a potential lethal complication of catheter ablation for AF have emerged [3–9]. The occurrence of a coronary spasm during catheter ablation seems to be multifactorial, and its underlying mechanisms have not been fully determined. The clarification of these mechanisms can be expected to contribute to the prevention of coronary spasms during catheter ablation. Here we describe the case of a patient who experienced a coronary spasm during catheter ablation for his AF. The initial high-dose intravenous infusion of dexmedetomidine was highly suspected to be responsible for the spasm.

* Corresponding author at: Department of Cardiology, Ogaki Municipal Hospital, 4-86 Minaminokawa-cho, Ogaki 503-0864, Japan.
E-mail address: morishima-i@muc.biglobe.ne.jp (I. Morishima).

https://doi.org/10.1016/j.jccase.2019.08.010
1878-5409/

Case report

A 55-year-old man with symptomatic paroxysmal AF and a history of hypertension and hypercholesteremia was admitted to our institution for catheter ablation. His height was 175 cm and body weight was 84 kg. He had had no episodes suggestive of coronary spastic angina (CSA) prior to the procedure. After written informed consent was obtained, pulmonary vein isolation was performed with a 28-mm second-generation cryo-balloon catheter (Medtronic, Minneapolis, MN, USA) with the patient under sedation with propofol. Cavo-tricuspid isthmus linear ablation was also performed, by the irrigation catheter. The procedure was completed without any complications.

A repeated session of catheter ablation was planned 15 months later, because the patient had experienced recurrent episodes of AF. The transesophageal echocardiogram performed before the session did not reveal any intracardiac thrombi, patent foramen ovale, or atrial septal defect. The 12-lead electrocardiogram (ECG) conducted upon the patient's arrival at the electrophysiological laboratory showed normal ST-segments (Fig. 1A) and the blood pressure was 149/94 mmHg. We initiated a high-dose infusion of dexmedetomidine (4 μg/kg/h) instead of propofol at 8:40 am over the subsequent 15 min to introduce sedation. We then changed the dose of the drug to a maintenance-dose (0.4 μg/kg/h). We simultaneously administered fentanyl using the same protocol as in the first session (4 μg/kg/h for the initial 15 min followed by a maintenance dose of 0.4 μg/kg/h). We placed three sheaths in the right femoral vein, one in the right jugular vein, and one in the right femoral artery (Fig. 2). The blood pressure had dropped to 125/61 mmHg and the patient had become drowsy, indicating the effects of dexmedetomidine.

Then, before the trans-septal puncture, the surface ECG showed an ST segment elevation in inferior leads (Fig. 1B) at 9:01 am, suggesting acute myocardial ischemia.

Urgent coronary angiography showed a severe narrowing at the ostium of right coronary artery (Fig. 1C). Immediately, 5 mg of isosorbide dinitrate was administered in the right coronary artery, which successfully reversed the coronary narrowing and ST segment elevation (Fig. 1D,E). Since there was no evidence showing any air or thrombotic embolism in the coronary angiograms, we concluded that the patient's transient coronary stenosis was caused by a coronary vasospasm. While nitroglycerin was continuously infused, the radiofrequency catheter ablation was completed without an ST segment re-elevation.

Discussion

This case report indicates an intravenous administration of dexmedetomidine, an α-2 adrenergic receptor stimulant, as a potential cause of coronary vasospasm during catheter ablation for AF. Coronary vasospasm is a rare but potentially fatal complication associated with catheter ablation. According to several previous reports, the proposed mechanisms of coronary vasospasm related to catheter ablation include direct thermal effects on the coronary artery [3], indirect effects via cryoenergy-induced blood cooling [4], and an autonomic nervous system imbalance caused by the affected ganglionated plexus through a thermal or cooling injury [5–8]. It has been shown that α-2 adrenoreceptor-mediated vasoconstriction can involve the coronary circulation, especially in the presence of atherosclerosis and endothelial dysfunction [10]. It is thus possible that a stimulation of α-2 adrenergic receptors induced by dexmedetomidine causes a coronary vasospasm, and in

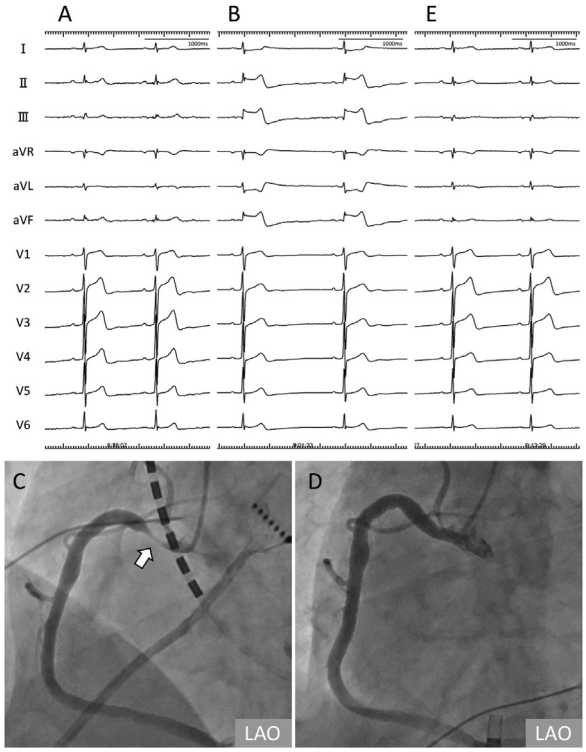

Fig. 1. Changes in 12-lead electrograms (ECGs) and coronary angiograms. (**A**) ECG at baseline. (**B**) ECG after the intravenous loading of dexmedetomidine. Marked ST-segment elevation in the inferior leads was seen. (**C**) Right coronary angiogram obtained while ST-segment elevation was seen. Severe narrowing was identified in the ostium of the right coronary artery. (**D**) Right coronary angiogram obtained after an intra-coronary injection of isosorbide dinitrate. The coronary narrowing was completely resolved, indicating that the coronary spasm was the cause of the ST-segment elevation. (**E**) ECG after the intra-coronary injection of isosorbide dinitrate. The ST-segment elevation was completely resolved.

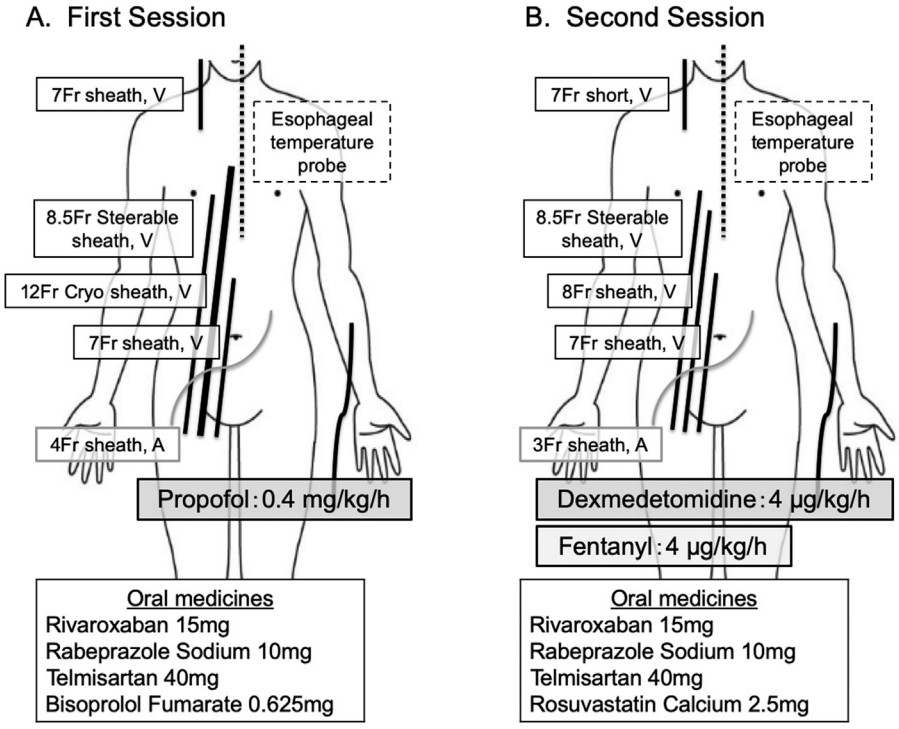

Fig. 2. Comparison of the procedural and pharmacological details observed before the trans-septal puncture between the first (A) and second (B) ablation sessions. The procedures were the same between the sessions including the sites of puncture, number of sheaths placed, and the esophageal temperature probe used. Propofol was used in the first session for sedation; dexmedetomidine was used in the second session.
A, artery; V, vein.

fact, dexmedetomidine was used in the recently reported cases of coronary vasospasm that occurred during catheter ablation [6–9]. However, this remains merely a hypothesis, and there is no clinical evidence to support it at this time.

The present case gives new insights into this hypothesis. The patient had never experienced CSA (including at the initial AF ablation procedure) but he experienced a coronary vasospasm at the beginning of the second-session catheter ablation, soon after the loading administration of dexmedetomidine. A comparison of the procedural details between the first and second sessions may help identify the cause of coronary vasospasm (Fig. 2). The patient had not been prescribed a coronary vasodilator such as a calcium channel blocker at either session. He had taken a β-blocker (which has the potential to induce a coronary artery spasm) at only the first session. The same type of esophageal temperature probe was placed in the esophagus for both sessions. The insertion of the sheaths may have caused a vagal response. However, the puncture sites and the number of sheaths were exactly the same in the first and second sessions. Larger sheaths were used in the first session compared to those used in the second session (Fig. 2).

The above background of the second session seems to be less favorable for coronary spasm compared to the first session. Regarding the patient's sedation, neither propofol nor fentanyl has pharmacological properties that induce or prevent coronary artery spasms. On the other hand, the effects of dexmedetomidine were apparent at the onset of coronary vasospasm as shown by a drop in blood pressure and a mild sedative state. In light of the above considerations, we concluded that dexmedetomidine administered with loading doze was highly likely to be responsible for the coronary vasospasm that occurred in the patient's second session of catheter ablation for his AF. In addition, because of the coexisting hypertension and hypercholesteremia, the patient may have had endothelial dysfunction of the coronary arteries and have been prone to develop coronary spasm when exposed to a potential vasoconstrictor.

A limitation of this case report is that we have not confirmed the reproducibility to show the causal association between coronary spasm and dexmedetomidine infusion. It is not ethically acceptable to repeatedly use dexmedetomidine in a patient in whom vasospasm occurred immediately after the drug administration.

In summary, an intravenous administration of dexmedetomidine, an α-2 adrenergic agonist, may cause a coronary spasm during catheter ablation for AF. Physicians should be aware of this potentially lethal side effect, especially at the time of an initial loading dose infusion to the patient. Further research is expected to elucidate the causal association between coronary vasospasm and an intravenous infusion of dexmedetomidine.

Conflict of interest

The authors declare that there is no conflict of interest.

Acknowledgment

None.

References

[1] Calkins H, Hindricks G, Cappato R, Kim Y-H, Saad EB, Aguinaga L, et al. 2017 HRS/EHRA/ECAS/APHRS/SOLAECE expert consensus statement on catheter and surgical ablation of atrial fibrillation: executive summary. J Arrhythm 2017;33:369–409.
[2] Gerstein NS, Young A, Schulman PM, Stecker EC, Jessel PM. Sedation in the electrophysiology laboratory: a multidisciplinary review. J Am Heart Assoc 2016;5. pii:e003629.

[3] Takahashi Y, Jais P, Hocini M, Sanders P, Rotter M, Rostock T, et al. Acute occlusion of the left circumflex coronary artery during mitral isthmus linear ablation. J Cardiovasc Electrophysiol 2005;16:1104–7.
[4] Lehrmann H, Potocnik C, Comberg T, Peck W, Salama A, Schneider J, et al. Near-fatal coronary artery spasm during cryoballoon pulmonary vein isolation: an unreported complication. Circ Arrhythm Electrophysiol 2014;7:1273–4.
[5] Hishikari K, Kuwahara T, Takahashi A, Isobe M. Severe coronary artery spasm during radiofrequency ablation for atrial fibrillation. Int J Cardiol 2014;172:e513–5.
[6] Watanabe T, Hachiya H, Miyazaki S, Nakamura H, Taniguchi H, Iesaka Y. Recurrent and late-onset coronary spasms after cryoballoon ablation procedure in a patient with atrial fibrillation. Heart Rhythm Case Rep 2016;2:421–4.
[7] Kaneshiro T, Matsumoto Y, Nodera M, Kamioka M, Suzuki H, Takeishi Y. Spastic occlusion of coronary artery during cryoballoon pulmonary vein isolation. Heart Rhythm Case Rep 2017;3:509–12.
[8] Honda N, Takase S, Tashiro H. Severe coronary artery spasm repeatedly induced after left pulmonary vein isolation in patient with atrial fibrillation. Heart Rhythm Case Rep 2018;4:501–5.
[9] Kagawa Y, Fujii E, Fujita S, Yamada N, Ito M. Three cases of vasospastic angina following catheter ablation of atrial fibrillation. J Arrhythm 2017;33:511–3.
[10] Heusch G, Baumgart D, Camici P, Chilian W, Gregorini L, Hess O, et al. Alpha-adrenergic coronary vasoconstriction and myocardial ischemia in humans. Circulation 2000;101:689–94.