Radiofrequency catheter ablation of a sporadically occurring ventricular arrhythmia originating from the right ventricular outflow tract: A novel arrhythmia induction strategy involving atrial fibrillation provocation

Taro Koya, MD, Taro Temma, MD, PhD, Masaya Watanabe, MD, PhD, Rui Kamada, MD, PhD, Toshiyuki Nagai, MD, PhD, Toshihisa Anzai, MD, PhD

From the Department of Cardiovascular Medicine, Faculty of Medicine and Graduate School of Medicine, Hokkaido University, Sapporo, Japan.

Introduction

Arrhythmias originating from the right ventricular outflow tract (RVOT) have been well described.[1] However, the influence of concomitant atrial fibrillation (AF) on the ventricular arrhythmia burden and severity has not been discussed. We present a unique case of a patient with a symptomatic RVOT arrhythmia and paroxysmal AF who received radiofrequency (RF) catheter ablation for the ventricular arrhythmia. AF induction increased the frequency of the ventricular arrhythmia, which enabled us to perform successful ablation using activation mapping for the targeted ventricular arrhythmia.

KEY TEACHING POINTS

- The induction of atrial fibrillation (AF) increased the frequency of an idiopathic right ventricular outflow tract (RVOT) arrhythmia.
- Only AF itself, not ventricular train pacing or isoproterenol infusion, raised the incidence of RVOT arrhythmia.
- The unique approach of ventricular arrhythmia induction by atrial fibrillation provocation might be helpful for the ablation cases in which the arrhythmia occurs infrequently.

Case report

A 33-year-old man with no history of structural heart disease was referred to our hospital owing to frequent premature ventricular contractions (PVCs) accompanied by palpitations. He had a prior history of a catheter ablation procedure for the RVOT PVCs, during which the prevalence of the PVCs had been infrequent. Although a pace mapping–guided ablation was performed, he reported suffering from the recurrence of symptomatic PVCs.

A 12-lead electrocardiogram (ECG) upon admission showed the monomorphic PVCs, which was similar to the morphology observed on a previous ECG. A left bundle branch block–type QRS morphology and an inferior axis with a V_4 transitional zone were present, suggesting an RVOT origin (Figure 1A). Another ECG tracing taken at the outpatient clinic showed an AF (Figure 1B) and more frequent PVCs and nonsustained ventricular tachycardia (NSVT) with the same QRS morphology as that observed when the patient was in sinus rhythm.

For the current procedure, the patient was in sinus rhythm and placed under local anesthesia. A few PVCs were elicited despite ventricular train pacing (minimum pacing cycle length 200 ms) or isoproterenol (1.0 μg/min) infusion (Figure 2A–C), which interfered with our ability to perform activation mapping for the PVCs. Next, we induced AF by rapid atrial pacing maneuvers, which resulted in a dramatic increase in the frequency of PVCs and incessant NSVT (Figure 2D). The activation map for targeting the ventricular arrhythmia was constructed using a 4-mm-tip electrode catheter (Navistar; Biosense Webster, Diamond Bar, CA) combined with anatomical images that were acquired using an intracardiac ultrasound catheter associated with the CARTO 3

KEYWORDS Atrial fibrillation; Catheter ablation; Nonsustained ventricular tachycardia; Premature ventricular contraction; Right ventricular outflow tract

(Heart Rhythm Case Reports 2020;6:411–414)

Address reprint requests and correspondence: Dr Taro Temma, Department of Cardiovascular Medicine, Faculty of Medicine and Graduate School of Medicine, Hokkaido University, Kita-15, Nishi-7, Kita-ku, Sapporo, Hokkaido 060-8638, Japan. E-mail address: tarotemma@g.ucla.edu.

https://doi.org/10.1016/j.hrcr.2020.03.018

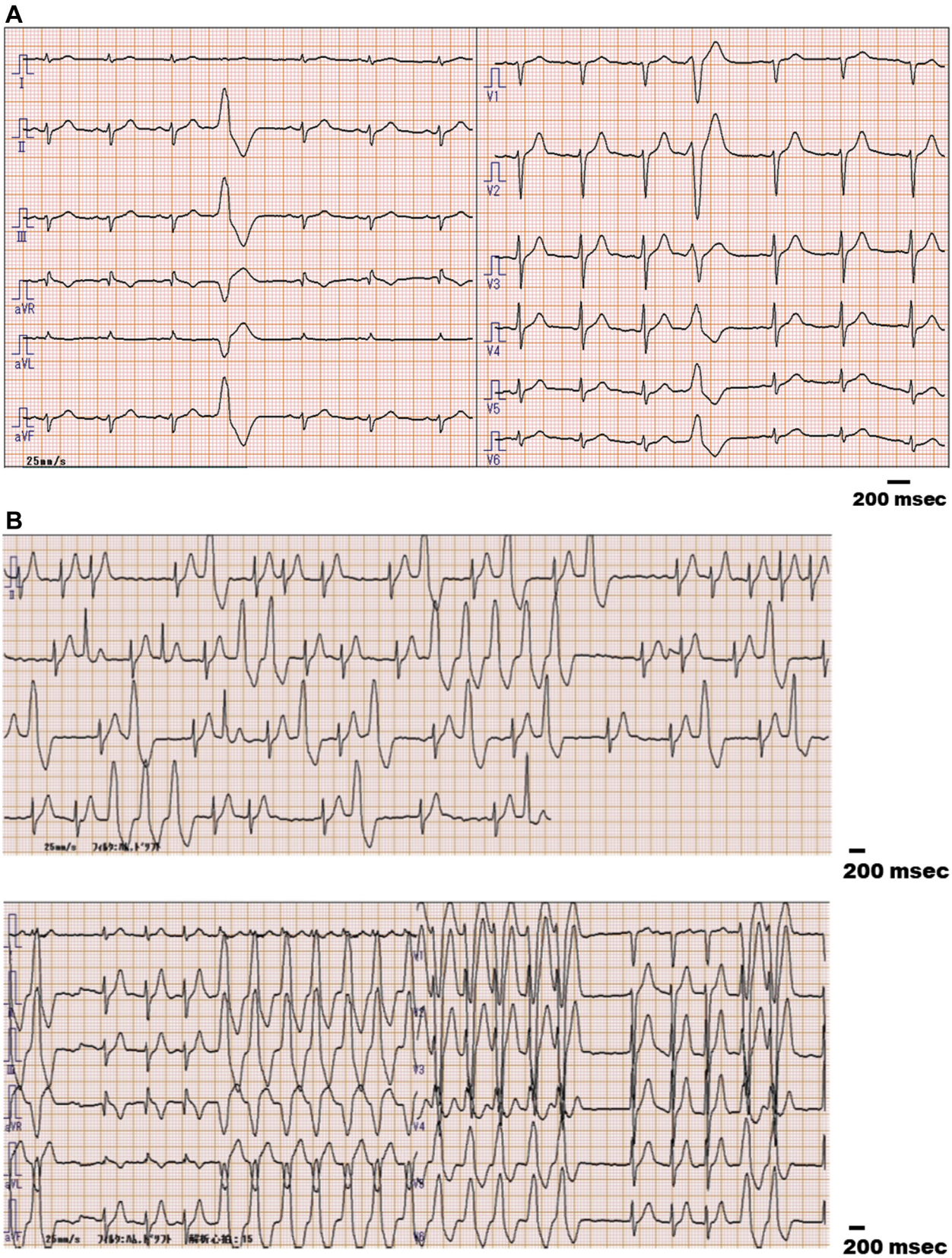

Figure 1 Electrocardiogram showing sinus rhythm (**A**) and atrial fibrillation (AF) (**B**). **A:** Monomorphic premature ventricular contractions (PVCs) with a left bundle branch block–type QRS morphology and inferior axis with V_4 transitional zone were observed in sinus rhythm. **B:** AF increased PVC frequency (top), and repetitive nonsustained ventricular tachycardia was documented (bottom).

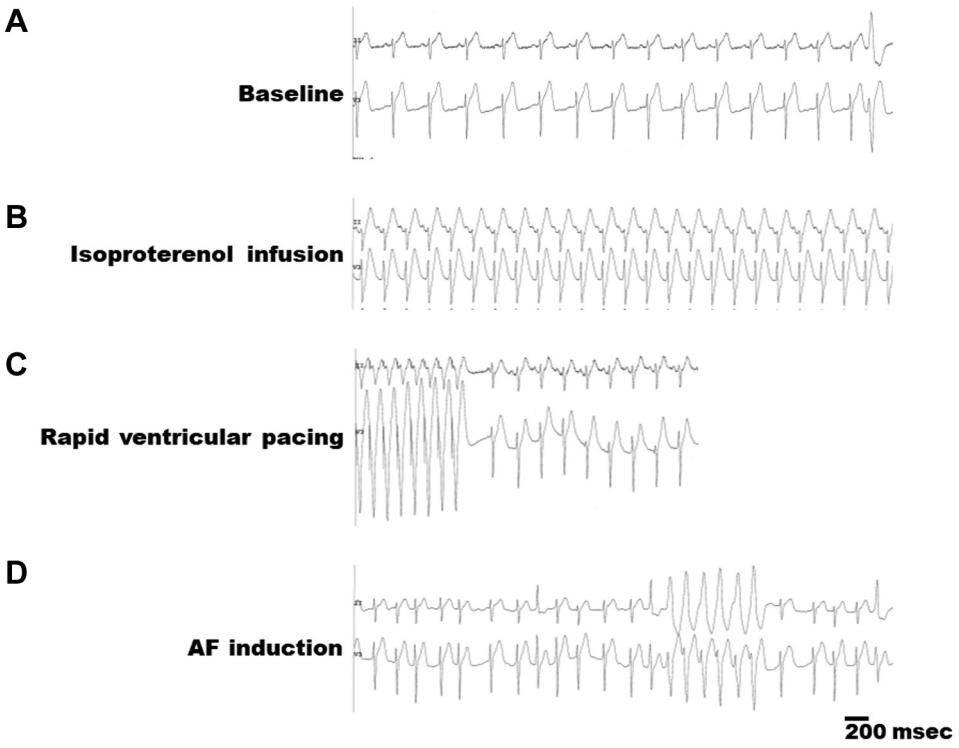

Figure 2 Electrocardiogram recorded in the electrophysiology laboratory: **A:** at baseline, showing sinus rhythm; **B:** after isoproterenol infusion; **C:** during rapid ventricular pacing; **D:** after atrial fibrillation (AF) induction.

system (CARTO 3; Biosense Webster, Diamond Bar, CA). The activation map showed that earliest activation was on the right ventricular free wall. RF applications (30–35 W, 50–55°C) were then delivered to the area that had the earliest activation (Figure 3). RF application stopped the ventricular arrhythmias. After that we could not induce any ventricular arrhythmias. The patient underwent cardioversion for the AF at the completion of the procedure. The patient has been free from recurrence of any ventricular arrhythmias for more than 7 months.

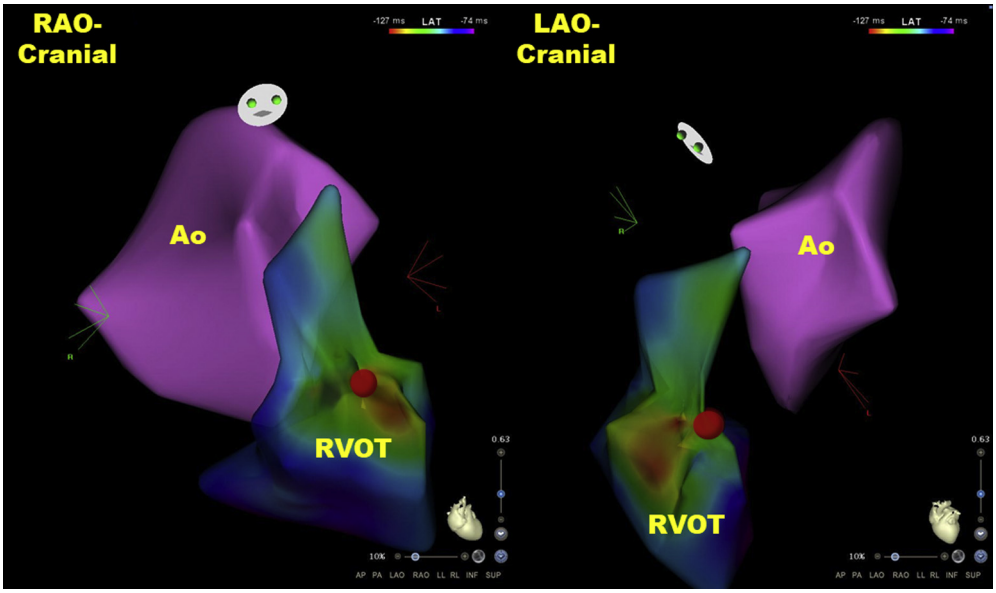

Figure 3 Activation map for targeting the ventricular arrhythmia in right anterior oblique cranial (RAO-cranial) and left anterior oblique cranial (LAO-cranial) views. The earliest activation was observed at the free wall of the right ventricular outflow tract (RVOT). The red dot indicates the site of the radiofrequency application sites. Ao = aorta.

Discussion

This case highlights an important clinical issue; that is, AF increased the prevalence of ventricular arrhythmias originating from the RVOT, which enabled the elimination of the RVOT arrhythmias via RF catheter ablation.

Most RVOT arrhythmias occur in patients without structural heart disease and are curable by ablative therapy.[2,3] However, the success rate of ablation often depends on the frequency of the arrhythmia during the procedure.[4] In this case, the patient had infrequent RVOT arrhythmias when in sinus rhythm during the ablation procedure, even after isoproterenol challenge. Interestingly, when AF was induced in the patient, more frequent PVCs and repetitive NSVT were observed and were reproducible, leading to the successful abolition of the ventricular arrhythmia. To the best of our knowledge, this is the first report that describes the advantage of AF induction to increase the prevalence of an RVOT arrhythmia that manifested as infrequent ectopic beats during the ablation procedure. This novel approach might be helpful for idiopathic RVOT arrhythmia ablation cases; further prospective studies examining this hypothetical strategy are needed in the future.

Two possible mechanisms of the increased prevalence of RVOT arrhythmias by AF are considered; 1 is calcium (Ca^{2+}) overload and the other is the altered autonomic nervous system tone. First, previous studies showed that AF adversely influenced calcium handling not only in the atrial myocardium but also in the ventricular myocardium.[5] And Ling and colleagues[6] also reported that the rhythmicity of AF impaired the Ca^{2+} handling. The irregularity of AF itself, not the burst pacing and isoproterenol infusion, might influence its handling more strongly. Therefore, we think that only AF-induced Ca^{2+} overload provoked triggered activity causing the RVOT arrhythmias in this case. Second, it has been reported that AF activates the autonomic nervous system in both animal model and human studies.[7,8] Furthermore, a recent study reported that arrhythmogenesis in the RVOT is associated with sympathetic fibers in the animal model.[1] Therefore, the increased sympathetic tone induced by AF would play an important role in the induction of RVOT arrhythmias. Moreover, in this case, the synergistic effect of these mechanisms by AF might cause RVOT arrhythmias.

Conclusion

We experienced a unique case of symptomatic RVOT arrhythmia, in which AF increased the frequency of the ventricular arrhythmia. The induction of AF might be useful for RF catheter ablation based on activation mapping in sporadic, but symptomatic, RVOT arrhythmia.

References

1. Lamba J, Redfearn DP, Michael KA, Simpson CS, Abdollah H, Baranchuk A. Radiofrequency catheter ablation for the treatment of idiopathic premature ventricular contractions originating from the right ventricular outflow tract: a systematic review and meta-analysis. Pacing Clin Electrophysiol 2014; 37:73–78.
2. Wang K, Hodges M. The premature ventricular complex as a diagnostic aid. Ann Intern Med 1992;117:766–770.
3. Takemoto M, Yoshimura H, Ohba Y, et al. Radiofrequency catheter ablation of premature ventricular complexes from right ventricular outflow tract improves left ventricular dilation and clinical status in patients without structural heart disease. J Am Coll Cardiol 2005;45:1259–1265.
4. Vestal M, Wen MS, Yeh SJ, Wang CC, Lin FC, Wu D. Electrocardiographic predictors of failure and recurrence in patients with idiopathic right ventricular outflow tract tachycardia and ectopy who underwent radiofrequency catheter ablation. J Electrocardiol 2003;36:327–332.
5. Wijesurendra RS, Casadei B. Atrial fibrillation: effects beyond the atrium? Cardiovasc Res 2015;105:238–247.
6. Ling LH, Khammy O, Byrne M, et al. Irregular rhythm adversely influences calcium handling in ventricular myocardium: implications for the interaction between heart failure and atrial fibrillation. Circ Heart Fail 2012;5:786–793.
7. Carnagarin R, Kiuchi MG, Ho JK, Matthews VB, Schlaich MP. Sympathetic nervous system activation and its modulation: role in atrial fibrillation. Front Neurosci 2018;12:1058.
8. Chang HY, Lo LW, Chen YR, et al. The autonomic neural mechanism of right ventricular outflow tract tachycardia. Auton Neurosci 2018;212:10–16.