The efficacy of radiofrequency catheter ablation for menstruation-dependent incessant ventricular tachycardia: A case report

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Introduction

Menstrual cycles have been reported to relate to some arrhythmias. The changes in sex hormone levels during the menstrual cycle influence the frequencies and symptoms of arrhythmia.[1,2] Sex hormones are known to affect cardiac ion channels and cause sex differences in cardiac electrophysiological characteristics. Previous studies revealed sex differences in the regulation of QTc interval[3] and the effect on this feature of the electrocardiogram (ECG) of menstrual cycle–related changes.[4]

Idiopathic ventricular arrhythmia originating from the right ventricular outflow tract (RVOT) is known to be more common in female subjects.[5] Although previous reports[6,7] suggested that sex hormonal effects could be one of the possible mechanisms for sex differences in ventricular arrhythmia, the pathologic mechanisms remain unclear. A menstruation-dependent premature ventricular contraction (PVC) originating from the RVOT has been reported.[8] However, the relationships between the menstrual cycle and incessant outflow tract ventricular tachycardia (VT), and the efficacy of catheter ablation, have not been investigated.

Here, we report a premenopausal case with an incessant form of menstruation-dependent nonsustained VT originating from the RVOT, which was effectively treated with radiofrequency (RF) catheter ablation.

Case report

A 41-year-old woman was referred to our hospital for periodic palpitations. A baseline ECG showed normal sinus rhythm; however, nonsustained monomorphic VT occurred during an exercise ECG. She did not have any family history of heart disease or sudden cardiac death. She was not taking any medicines. Her echocardiography showed no structural abnormalities and computed tomography showed that her coronary arteries were normal. A 24-hour Holter ECG showed monomorphic PVCs (6720 beats/day) and no VT. We prescribed daily bisoprolol (2.5 mg/day) and her palpitations were well controlled.

Two months later she was admitted to our hospital owing to abruptly worsening palpitations and presyncope. Continuous ECG monitoring revealed incessant (17 episodes/day) nonsustained monomorphic VT and PVCs (3037 beats/day). The morphology of both VT and PVCs was inferior axis, left bundle branch block, R/S transition in lead V_3, and notched QRS in inferior leads (Figure 1). Based on these characteristics, we suspected that the origin of VT and PVCs was the RVOT free wall. Her menstruation started on the day after admission. We focused on the relationship between menstrual cycle and the arrhythmia burden. We confirmed by additional careful investigation of her history that her symptoms reproducibly occurred just before and at the beginning of menstruation. We could reduce her symptoms by continuous infusion of lidocaine (60 mg/hour). We performed catheter ablation on hospital day 8 to avoid menstruation. We considered that the mechanism of this VT was triggered activity. Our ablation target was the trigger PVC, which was shown as the first beat of VT. At the beginning of the session VT was not incessant and there were few

KEY TEACHING POINTS

- Outflow tract ventricular tachycardia in premenopausal women can be periodically incessant, during certain parts of the menstrual cycle.
- Menstruation-dependent arrhythmia is supposed to be associated with the cyclic change of the sex hormone levels, especially estrogen.
- Radiofrequency catheter ablation is an effective treatment for this arrhythmia, and performing the ablation procedure at the most inducible timing during the menstrual cycle is important for success.

KEYWORDS Catheter ablation; Incessant form; Menstruation; Outflow tract ventricular tachycardia; Sex differences
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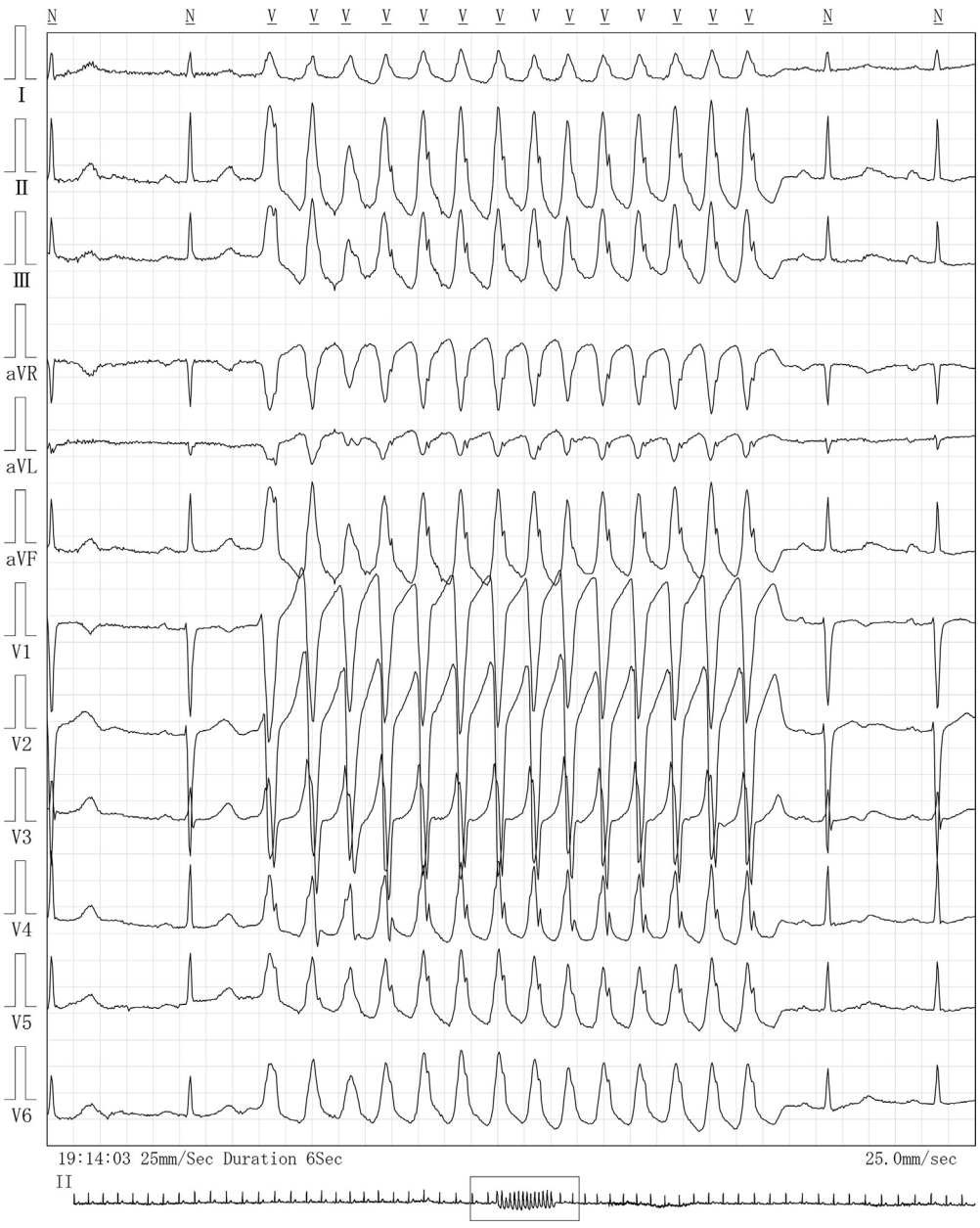

Figure 1 Twelve-lead electrocardiogram of clinical ventricular tachycardia with inferior axis, left bundle branch block, R/S transition in V_3 lead, and notched QRS in inferior leads.

PVCs. Therefore, we started intravenous isoproterenol infusion to perform activation mapping. VT could be easily induced, and the number of PVCs was increased. The earliest activation site was at the posterior attachment of the free wall in the RVOT, where local activation preceded QRS onset by 40 ms and a pace map matched the trigger PVC of the clinical VT (Figures 2 and 3). The RF application had an accelerated response and successfully eliminated VT. After bonus ablations, the session was terminated, with VT now noninducible by any programmed electrical stimulated pacing, with or without isoproterenol infusion.

She did not have any symptoms for about a month after discharge; however, she was admitted again owing to palpitations and presyncope just before the next menstrual period. A 12-lead ECG revealed that her nonsustained VT recurred with slight morphological changes. We added mexiletine (300 mg/d) to bisoprolol (2.5 mg/d) and her symptoms became bearable.

Six months later, she revisited our emergency center with incessant VT. We planned the second session of catheter ablation for her drug-resistant VT on day 3 of hospitalization. Her menstruation began on the day we performed catheter ablation. At the beginning of the session, VT was incessant, and PVCs occurred in a bigeminal pattern. We constructed an activation map using a multielectrode mapping catheter (PentaRay, Biosense Webster Inc., Diamond Bar, CA) and a 3D mapping system (Carto 3, Biosense Webster Inc.). We found the earliest activation point in the posterior part of the RVOT and a pace map performed at the point was matched to the trigger PVC of the clinical VT (Figure 2). This area was

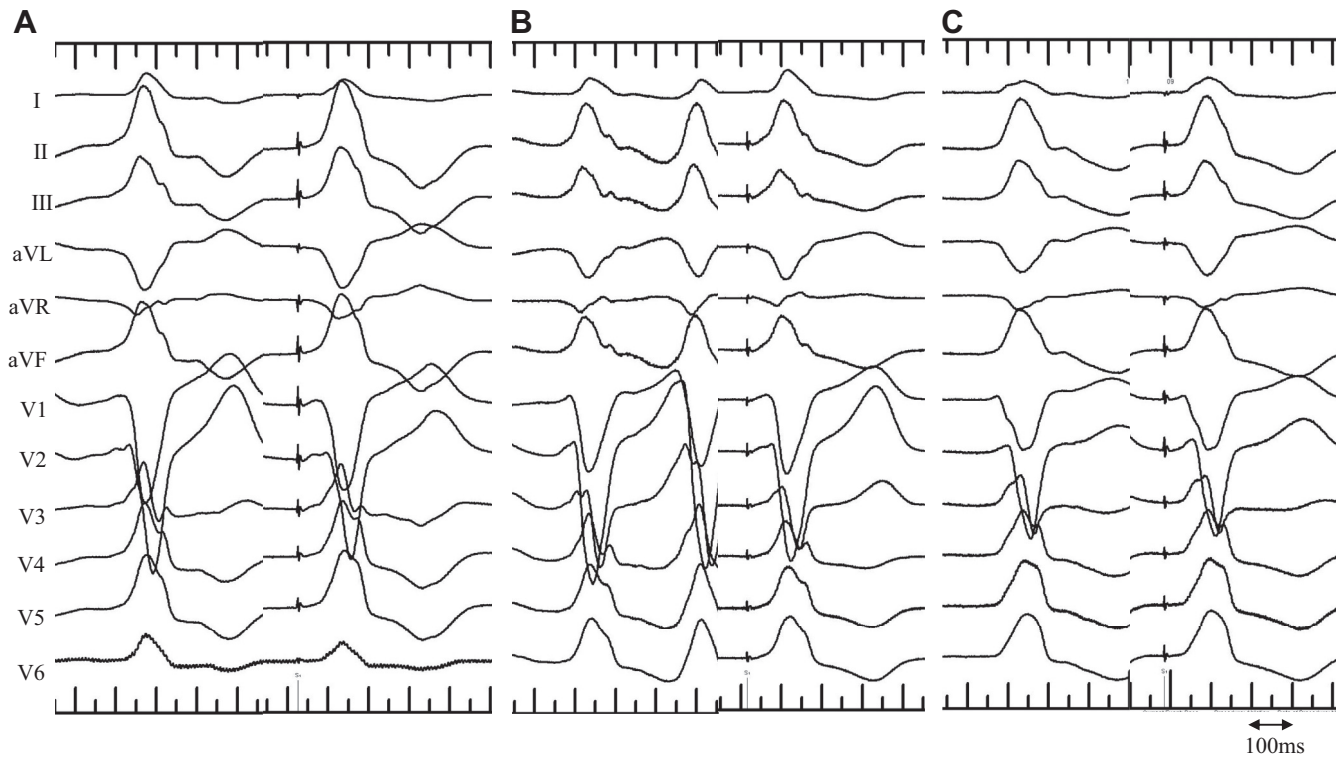

Figure 2 The QRS morphology of targeted trigger premature ventricular contraction of ventricular tachycardia (left) and pace map (right) in **A:** the first session and **B:** the second session. **C:** The QRS morphology changed slightly during the second session after radiofrequency application. PVC = premature ventricular contraction; VT = ventricular tachycardia.

slightly caudal and leftward to the site where we had performed RF application in the first session. Accelerated responses were obtained by RF applications at that site and VT was eliminated briefly. However, her VT recurred readily after intravenous isoproterenol infusion with slight morphological changes. Therefore, we performed activation mapping in more detail, and the earliest ventricular activation preceded the onset of QRS complex by 50 ms at a slightly more inferior and rightward location where a pace map matched the trigger PVC (Figures 2 and 3). Finally, we successfully eliminated VT after RF application at the earliest activation site and finished the session after confirming noninducibility of VT. She has been free from any palpitations and presyncope for 6 months after discharge. A 24-hour Holter ECG performed during her menstruation did not show any ventricular arrhythmia.

Discussion

This report showed 2 important clinical issues. First, outflow tract VT can be incessant during certain parts of the menstrual cycle; and second, RF catheter ablation performed at the most inducible period of the menstrual cycle is an effective treatment for menstruation-dependent incessant VT.

VT originating from the RVOT can be incessant during menstruation. It was reported that there were lower levels of estradiol in cases with idiopathic ventricular outflow tract arrhythmia compared to controls,[9] and that there were fewer counts of PVCs in the ovulation period than in the menstruation period.[10] Female sex hormones, especially estrogen, were suggested to play an important role in these clinical results. For premenopausal women, estrogen levels remain high during the ovulation phase and abruptly decrease at the end of the ovulation phase and become low at the beginning of the menstruation phase. Menstruation-dependent arrhythmia is believed to be associated with the cyclic adenosine monophosphate–dependent focal firing caused by the abrupt reduction of the estradiol level just before menstruation.[8] In this case, we diagnosed our patient's intermittent arrhythmia as menstruation-dependent incessant VT, because her symptoms reproducibly occurred only just before or at the beginning of menstruation. Although we could not measure actual hormone levels, we speculated that decreasing estrogen levels at the end of the ovulation phase caused the incessant VT in this patient.

RF catheter ablation is an effective treatment for this arrhythmia, and the appropriate timing to perform ablation during the menstrual cycle is important for success. In general, ventricular arrhythmia originating from the RVOT without structural heart disease is considered to be benign[11] and RF catheter ablation is widely used with a high success rate. However, there are some refractory cases and the optimal treatment in such patients has not been elucidated. Previous reports revealed that taking combined estrogen-progesterone oral contraceptives was an alternative treatment

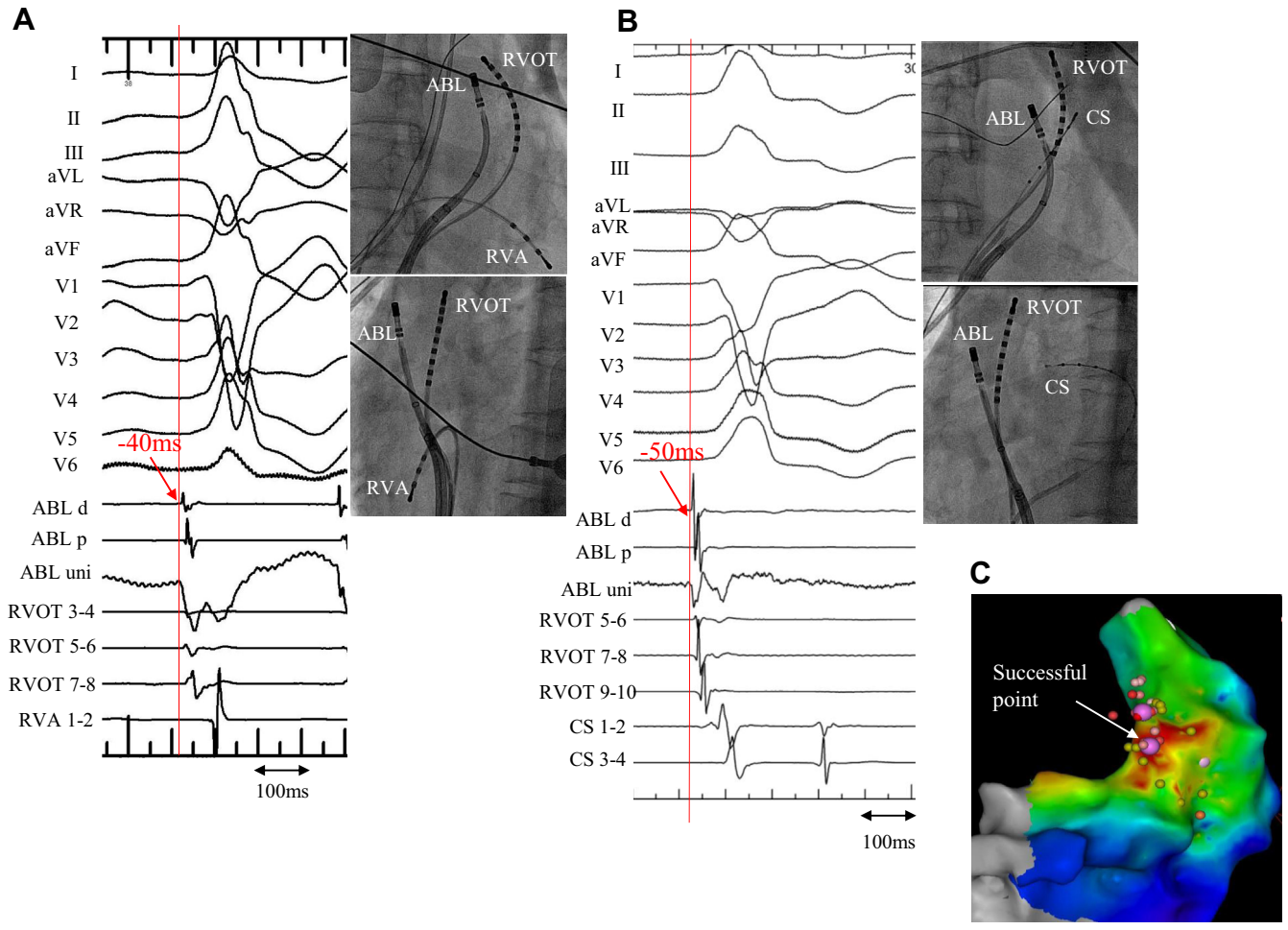

Figure 3 Intracardiac electrogram and fluoroscopic images of successful ablation point during first session (**A**) and second session (**B**) (left, intracardiac electrogram; right upper, right anterior oblique view; right lower, left anterior oblique view). **C:** Carto (Biosense Webster Inc., Diamond Bar, CA) activation mapping of right anterior oblique view; white arrow shows successful ablation point. ABL = ablation catheter electrode; CS = coronary sinus electrode; RVA = right ventricular apex electrode; RVOT = right ventricular outflow tract electrode.

for drug-refractory ventricular arrhythmias in premenopausal women.[12,13] We did not use oral contraceptive pills for the present case; however, this could be another treatment strategy if VT is ablation refractory. Periodic administration of antiarrhythmic agents was also reported to be an effective strategy to reduce symptoms of menstruation-dependent arrhythmia.[1,8] There are no reports on treatment of menstruation-dependent ventricular arrhythmia by RF catheter ablation.

In the present case, we performed the first session of catheter ablation when VT worsened. The session was planned in the middle of the menstrual cycle to avoid heavy menstrual bleeding. Although VT was not incessant during that session, we could induce VT easily with isoproterenol infusion and perform activation mapping. We judged that the procedure succeeded in view of certain criteria, including favorable responses during RF application and no induction of VT after the procedure; however, VT recurred during the next menstruation. Therefore, we scheduled the second session during her menstruation cycle when the arrhythmia was most incessant. Finally, we eliminated VT by performing RF application at a slightly different site from the first session. These results indicated that RF applications during the first session of ablation resulted in interference with an exit site; however, they incompletely abolished the arrhythmogenic origin. Higher inducibility just before the next menstrual period might facilitate the opening of another exit site. We could interfere with another exit site and eliminate the origin of the VT during the second session. These findings suggested that some menstruation-dependent outflow tract VTs could be difficult to eliminate by ablation and we should schedule to perform ablation at the most inducible time during the menstruation cycle for success.

In conclusion, this is the first report of menstruation-dependent incessant VT that was efficiently treated by catheter ablation. Outflow tract VT can be incessant just before and at the beginning of menstruation. Therefore, catheter ablation may be an effective treatment strategy for incessant VT. We should be aware that some idiopathic ventricular arrhythmias in premenopausal women can be influenced by their menstrual cycle.

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