Symptomatic Repetitive Right Ventricular Outflow Tract Ventricular Tachycardia in Pregnancy and Postpartum

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Idiopathic ventricular tachycardias, which occur in patients without structural heart disease, are a common entity, representing up to 10% of all ventricular tachycardias evaluated by cardiac electrophysiology services. Pregnancy can increase the incidence of various cardiac arrhythmias. Factors that can potentially promote arrhythmias in pregnancy include the effects of hormones, changes in autonomic tone, hemodynamic perturbations, hypokalemia, and underlying heart disease. Ventricular arrhythmias in pregnancy are repetitive monomorphic ventricular premature complexes and couplets that frequently originate at the right ventricular outflow tract. New onset symptomatic repetitive right ventricular outflow tract ventricular tachycardia during pregnancy has been inadequately reported in the literature. We present a case of symptomatic repetitive right ventricular outflow tract tachycardia that started during pregnancy and continued in the postpartum period, requiring curative treatment with electrophysiology study and radiofrequency ablation.
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Pregnancy can precipitate various cardiac arrhythmias, complicate the invasive evaluation, and necessitate special consideration for treatment.[1-3] Factors that can potentially promote arrhythmias in pregnancy include the effects of hormones, changes in autonomic tone, hemodynamic perturbations, hypokalemia, and underlying heart disease.[4,5] The most common arrhythmias during pregnancy are atrial premature complexes, ventricular premature complexes, sinus tachycardia, and supraventricular tachycardia.[5]

New-onset, nonsustained and sustained ventricular tachycardia (VT) during pregnancy has been reported. In most patients, the condition responds to β-blocker therapy and resolves after delivery.[6] This case review describes a patient with symptomatic repetitive right ventricular outflow tract (RVOT) tachycardia that started during pregnancy and continued in the postpartum period.

Case Description

The 28-year-old primiparous patient presented to the cardiology office at 33 weeks of gestation for evaluation of new onset daily palpitations that had been occurring for the previous few weeks. The palpitations were spontaneous in onset without specific triggers, lasted for a few minutes, and resolved spontaneously. There was no syncope or presyncope associated with the palpitations. The patient did not have any other significant medical history. Her medication included prenatal vitamins. The physical examination was unremarkable except for a systolic murmur, which disappeared during application of pressure by the stethoscope and when the patient sat upright. Her serum electrolytes and thyroid function study results were in the normal range.

The baseline 12-lead electrocardiogram demonstrated frequent monomorphic premature ventricular complexes with left bundle branch block and inferior axis suggestive of RVOT origin (Figure 1). A 2-dimensional echocardiogram revealed preserved left ventricular systolic function, with an estimated left ventricular ejection fraction of 60%. There were no other significant valvular abnormalities or pericardial effusion. A 48-hour Holter monitor recording revealed frequent monomorphic premature ventricular complexes (24,480 per 24 hours) with couplets (3834 per 24 hours) and repetitive nonsustained right ventricular outflow tachycardia (809 per 24 hours) (Figure 2). (Nonsustained VT was defined as 3 or more premature ventricular complexes occurring at a rate of 100 beats per minute and lasting less than 30 seconds.) There were no sustained episodes of VT recorded by Holter monitor.

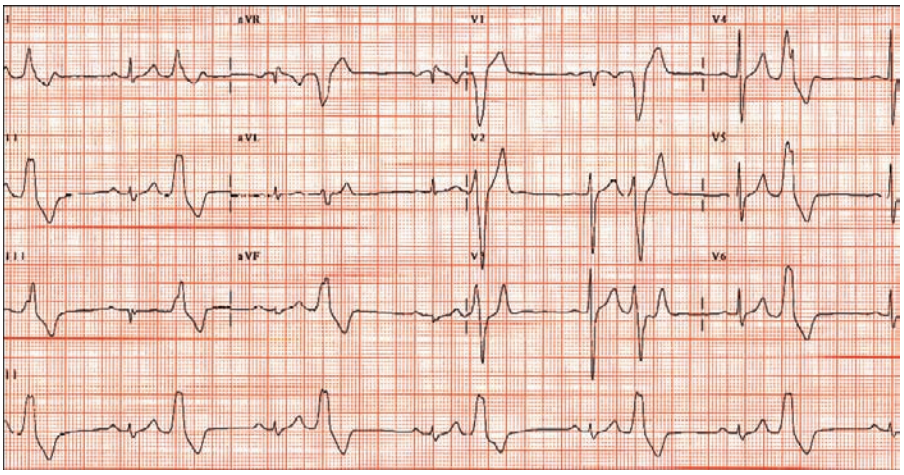

Figure 1. *Findings on this 12-lead electrocardiogram with monomorphic premature ventricular complexes are consistent with right ventricular outflow tract origin (left bundle branch block and inferior axis).*

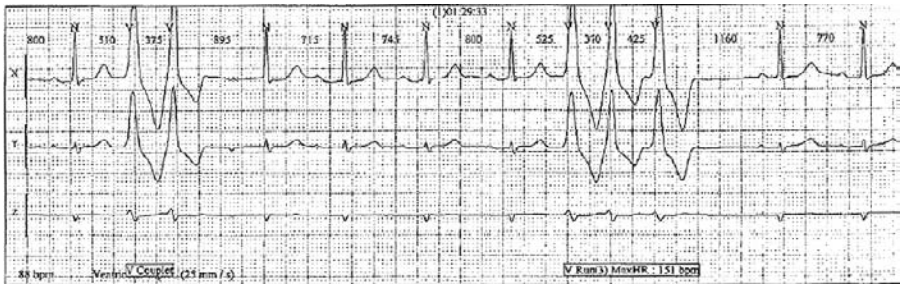

Figure 2. *This Holter monitor recording revealed frequent monomorphic premature ventricular complexes in couplets and repetitive, nonsustained, ventricular tachycardia.*

The symptoms resolved with treatment of metoprolol 50 mg twice daily. The patient's pregnancy and delivery were uncomplicated.

The Peripartum Period

During the peripartum period, the patient continued to have palpitations. Her symptoms were more pronounced during the second and third days of her menstrual period. Increasing the dose of metoprolol resulted in fatigue. The patient declined treatment with antiarrhythmic drugs and elected to undergo curative treatment with electrophysiology study and ablation.

In the electrophysiology study, the baseline intracardiac intervals were within normal limits. There were no echo beats or evidence of dual atrioventricular node physiology. No atrial arrhythmias were induced by burst pacing or programmed electrical stimulation. RVOT VT was easily induced by burst pacing on 5 μ/min of isoproterenol. Pace mapping and electroanatomical mapping were performed, localizing the area to the posterior-septal region of the RVOT

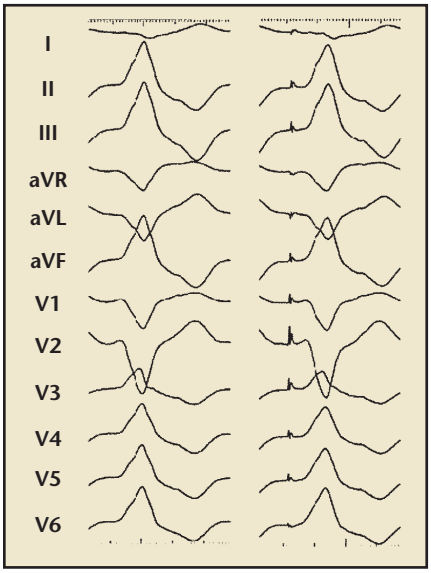

Figure 3. *12/12 match of pace map.*

(Figure 3). The arrhythmia was not entrainable. Radiofrequency ablation at the target site resulted in termination of the arrhythmia. Thirty minutes after radiofrequency ablation on 20 μ/min isoproterenol, the arrhythmia was not inducible. At 1-year follow-up, the patient continued to be symptom-free.

Case Discussion

VTs are usually observed in the setting of structural heart disease. Idiopathic ventricular tachycardias (IVTs), which occur in patients without structural heart disease, are a common entity, representing up to 10% of all VTs evaluated by cardiac electrophysiology services. IVTs usually occur in specific locations and have specific QRS morphologies, whereas VTs that are associated with structural heart disease have a QRS morphology that tends to indicate the location of the scar. IVTs consist of various subtypes that have been defined by their mechanism, QRS morphology, and site of origin. The most common idiopathic VT originates from a focus in the outflow tract of the right ventricle and is observed in 60% to 80% of patients.

The outflow tract tachycardias comprise a subgroup of IVTs that are predominantly localized in and around the RVOT and the left ventricular outflow tracts (LVOT). IVT originating at the RVOT occurs more frequently in women than in men.[7] It is the most common VT in people who are younger than 40 years and who have a structurally normal heart. Idiopathic RVOT tachycardia usually exhibits 1 of 2 clinical presentations: nonsustained, repetitive monomorphic VT or paroxysmal, exercise-induced, sustained ventricular tachycardia. RVOT and LVOT tachycardias share a similar tachycardia morphology (left bundle branch block and inferior axis).[8] Lerman and colleagues[9] demonstrated that the mechanism of the RVOT VTs appears to be triggered activity caused by delayed afterdepolarizations that are determined by intracellular calcium release. The release of calcium is negatively affected by adenosine, which inhibits the afterdepolarizations and terminates the tachycardia, and these arrhythmias are typically "adenosine sensitive." Common triggers of these arrhythmias include exercise, stress, caffeine, and hormonal fluctuation (Table 1).

Ventricular arrhythmias in pregnancy are reportedly benign; however, little is known about their characteristics and underlying mechanism.[10] The onset of the first episode of ventricular arrhythmia is distributed equally throughout the 3 trimesters. These ventricular arrhythmias are repetitive monomorphic ventricular premature complexes and couplets that frequently originate at the RVOT. Sex differences in ventricular repolarization or autonomic balance, particularly depressed parasympathetic tone, may be associated with the occurrence of these arrhythmias. These arrhythmic episodes are not associated with abnormalities in the dynamics of ventricular repolarization. The ventricular arrhythmias disappear completely after delivery, suggesting that the cardiovascular changes that occur during pregnancy may lead to or uncover arrhythmogenic foci in ventricular tissue.

The RVOT VT occurs more frequently in women than in men, and most female patients are sexually mature (ages 20 to 50 years). Estrogen is known to affect the cardiac ion channels and autonomic nervous system. Hormonal effects from elevated estrogen, progesterone, or other pregnancy-related hormones may be arrhythmogenic.[11] Marchlinski and colleagues[12] reported that the hormonal flux that occurs during the premenstrual, perimenopausal, and pregnancy periods can lead to VT initiation in women with RVOT

Table 1
Common Triggers for Right Ventricular Outflow Tract Ventricular Tachycardia

Specific Triggers	Hormonal Fluxes
Physical activity/exercise	Premenstrual/onset of menses
Stress	Perimenopausal
Caffeine exposure	Pregnancy
Extreme fatigue	Oral contraceptive administration

VT. The most common state of hormonal flux associated with the triggering of symptomatic ventricular arrhythmia appears to be the premenstrual state, with symptoms occurring predictably within 2 days of the onset and during the first 1 or 2 days of menses. In addition, arrhythmic symptoms develop coincident with the onset of marked irregularity of the menstrual cycle.[12] The pregnancy-associated alterations in cardiac autonomic modulation may play an important role in RVOT VT. Nakagawa and colleagues[13] found that heart rate variability and baroreflex sensitivity were significantly suppressed during pregnancy as compared with the postpartum period, suggesting a reduction in the cardiac vagal tone during pregnancy. Moreover, the catecholamine-sensitive nature of this type of VT is suggested by its sensitivity to β-blockers. The pregnancy-associated alteration in the autonomic nervous system, particularly depressed parasympathetic tone and sensitivity to circulating catecholamines, plays a central role in the adaptation of the cardiovascular system to various hemodynamic changes in pregnancy.

The mechanism for the arrhythmia initiation associated with hormonal flux has not been determined. The RVOT VT is associated with both high-estrogen conditions (pregnancy/birth control administration) and low-estrogen conditions (menopause/premenstrual period). Electrolyte shifts, such as hypokalemia and hypomagnesemia, may also play a role. Estrogen is known to affect the cardiac ion channels and autonomic nervous system.[14] The direct effect of estradiol inhibiting calcium uptake in myocytes has been demonstrated.[15] In patients who have arrhythmia during low-estrogen states, a lack of this effect may unmask a predisposition to these arrhythmias that may have a triggered mechanism. The triggering of RVOT VT may also be caused by increased levels of circulating catecholamines or an increased sensitivity to catecholamines.[16] Serum norepinephrine levels are increased during the premenstrual period,[17] and sensitivity to circulating catecholamines exists during pregnancy.[5] Because many of these patients do not have arrhythmias precipitated by exercise, the exact mechanism by which hormonal fluxes and endogenous catecholamines precipitate RVOT VT warrants more investigation.

This case report describes a rare example of RVOT VT in a woman that occurred in both high and low estrogen states. Unfortunately, levels of estrogen, other pregnancy-related hormones, and catecholamine were not obtained, and thus hormonal flux could not be established as a mechanism of the arrhythmia. In addition, this case emphasizes the potential curative role of radiofrequency ablation in RVOT VT, which has a very high success rate.

Conclusion

In this case of RVOT VT that started in pregnancy and continued in the postpartum, β-blockers controlled symptoms during pregnancy, which suggests the catecholamine-sensitive nature of this type of VT. It also suggests that automaticity or triggered activity was the underlying mechanism of the arrhythmias. Because the patient desired to have more children and symptoms of palpitations continued on β-blocker therapy, an electrophysiology study was performed. Radiofrequency ablation achieved successful results. ■

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Main Points

- Factors that can potentially promote arrhythmias in pregnancy include the effects of hormones, changes in autonomic tone, hemodynamic perturbations, hypokalemia, and underlying heart disease.
- Idiopathic ventricular tachycardias, which occur in patients without structural heart disease, are a common entity, representing up to 10% of all ventricular tachycardias evaluated by cardiac electrophysiology services.
- Idiopathic right ventricular outflow tract ventricular tachycardia (RVOT VT) usually exhibits 1 of 2 clinical presentations: nonsustained, repetitive monomorphic ventricular tachycardia or paroxysmal, exercise-induced, sustained ventricular tachycardia.
- Ventricular arrhythmias in pregnancy are repetitive monomorphic ventricular premature complexes and couplets that frequently originate at the right ventricular outflow tract.
- RVOT VT occurs more frequently in women than in men, and most female patients are sexually mature (ages 20 to 50 years).
- Radiofrequency ablation in RVOT VT has a very high success rate.

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