Atrial tachycardia ablation at the pulmonary outflow tract in a patient with congenitally corrected transposition of the great arteries

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Introduction

Atrial tachycardia (AT) is a common complication and may lead to serious consequences such as heart failure in adult congenital heart disease, and catheter ablation is an effective treatment.[1] Congenitally corrected transposition of the great arteries (ccTGA) is a rare congenital heart disease that contains atrioventricular (AV) discordance accompanied by ventriculoarterial discordance. Previous studies have reported the AT in patients with {S, L, L}-type ccTGA originates from the pulmonary outflow tract (POT) and can be eliminated by ablation in the POT.[2,3] However, the successful ablation of AT in patients with {I, D, D}-type ccTGA has been rarely reported. Here, we report a special case of a patient with {I, D, D}-type ccTGA in whom AT was successfully ablated in POT.

KEY TEACHING POINTS

- Congenitally corrected transposition of the great arteries (ccTGA) is a rare congenital heart disease, and computed tomography (CT) or cardiac magnetic resonance image is very useful to construct the true anatomical position relationship in patients.
- Atrial tachycardia (AT) in patients with ccTGA is relatively rare, and its ablation is difficult because of the reversed anatomical locations. It is very important with comprehensive activated mapping in the entire left and right atria and the adjacent structure (such as the pulmonic outflow tract) to find the earliest activation site for AT.
- Real-time intracardiac echocardiography images combined with CT or cardiac magnetic resonance imaging is very useful to guide the activated mapping during radiofrequency ablation.

Case report

A 50-year-old woman with {I, D, D}-type ccTGA presented with frequent palpitations. The symptoms occurred intermittently over the past 6 years but occurred frequently in the recent 6 months. Palpitations presented as sudden-onset and sudden-offset without chest pain and syncope. The transthoracic echocardiography demonstrated anatomy consistent with {I, D, D}-type ccTGA. The patient also had situs inversus, which was confirmed by computed tomography (CT) scan.

Standard 12-lead electrocardiogram (ECG) showed a regular supraventricular tachycardia (Figure 1). The characteristics of the ECG included the following: (1) it was a narrow QRS tachycardia; (2) the interval of RP′ > the interval of P′R; (3) the P′ was positive in leads V_1, II, III, and aVF and negative in lead I and aVL (Figure 1). Based on the ECG, we speculated that the possible type of this tachycardia was AT, fast-slow or slow-slow AV nodal reentrant tachycardia, or AV reentrant tachycardia. In order to identify the type of tachycardia, the patient underwent electrophysiological examination. In this case, a decapolar catheter (Dynamic

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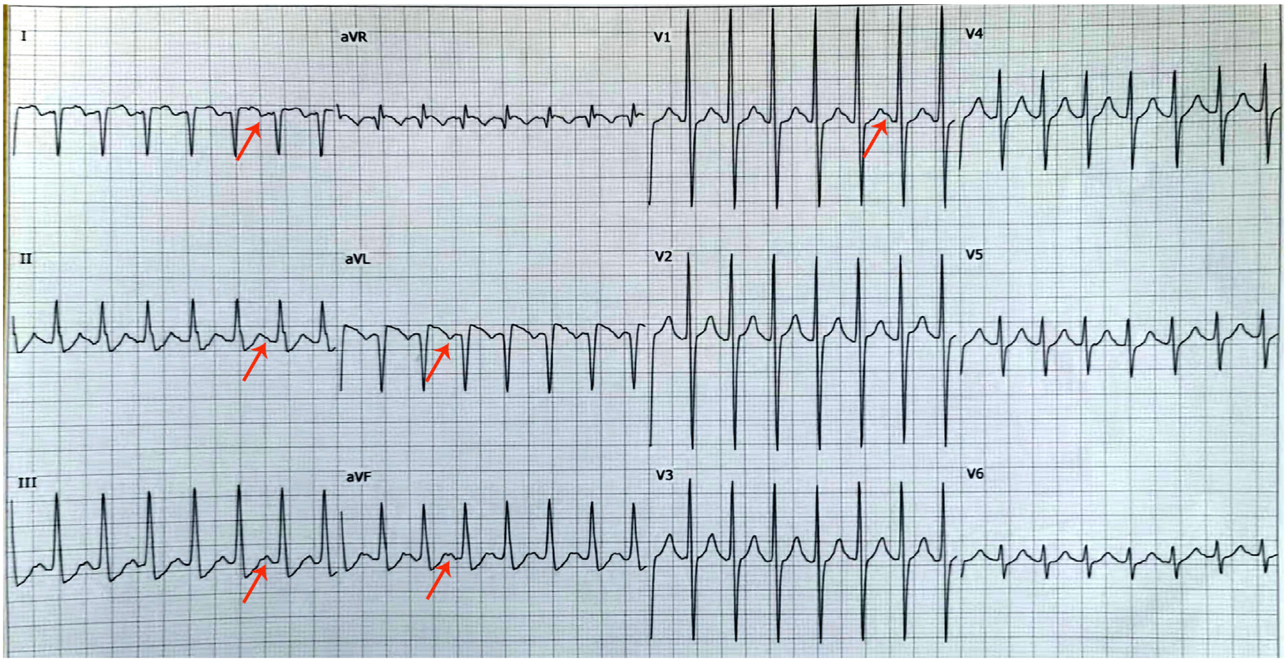

Figure 1 The standard 12-lead electrocardiogram of the supraventricular tachycardia. It was a narrow QRS tachycardia; the interval of RP > the interval of PR; the P wave (*red arrow*) was positive in leads V_1, II, III, and aVF and negative in leads I and aVL.

XT; Boston Scientific, California) was placed into the coronary sinus and a quadripolar catheter (Response CRD; Abbott, California) was advanced into the morphologic left ventricle (LV). An intracardiac echocardiography (ICE) catheter (AcuNav; Biosense Webster, California) was used to identify anatomical landmarks including the morphologic LV and right ventricle (RV), the left and right atria, pulmonic outflow tract, aortic root, and so on. Ventricular pacing at different cycle lengths showed ventriculoatrial dissociation, and thus excluded an accessory pathway that conducted retrogradely, therefore excluding orthodromic AV reentrant tachycardia (Figure 2A). Atrial programmed S1-S2 stimulation promoted a Wenckebach conduction mechanism at the AV nodal site, while no AV jump or AV nodal echo beats were observed. Atrial programmed stimulation could easily induce tachycardia (Figure 2B). Ventricular overdrive pacing still revealed ventriculoatrial dissociation during tachycardia (Figure 2C), which suggested that this was AT. A multielectrode catheter (PentaRay NAV, 2-6-2 mm spacing; Biosense Webster, California) was sent to the morphologic left atrium (functional right atrium) and was used for activation mapping. A stable intracardiac electrogram at the coronary sinus was used as reference for activation mapping (approximately equal timing as the onset of the P wave).

Activation mapping in the morphologic left atrium showed the early region of activation focused on the atrial septum, but there was no notable early site of activation (Figure 2D). So, we speculated that AT might originate from the morphologic right atrium (functional left atrium). In this patient with {I, D, D}-type ccTGA, transseptal puncture was the greatest challenge because of the significant change in the adjacent relationship between the left and right atria. Under the guidance of ICE, we successfully punctured the interatrial septum and performed activation mapping of the morphologic right atrium (functional left atrium). The early region of activation was in the atrial septum, which was similar to the result in the morphologic left atrium (Figure 2D).

A 3.5-mm, deflectable, irrigated catheter (NaviStar ThemoCool Smart Touch; Biosense Webster, Yokneam, Isreal) was used for ablation. Radiofrequency ablation (43°C, 40 W, 20 mL/min) was delivered at the early region of activation of both the right and left atrial septum, but AT did not terminate. The ablation catheter was used for remapping and the early region of activation was still at the atrial septum. Further mapping demonstrated the earliest activation site (EAS) in the POT, which was above the atrial septum (Figure 2F and 2G), and recorded double potentials with complex fractionated electrograms in the EAS. CT also showed that the POT was located just above the atrial septum (Figure 2H). Radiofrequency ablation performed at this site resulted in immediate termination of AT within 3 seconds. Additional lesions were delivered to consolidate the ablation surrounding the target. AT remained noninducible, despite aggressive programmed stimulation, burst pacing, and isoproterenol infusion.

The patient was discharged home on the third day after surgery with no recurrent AT over 6 months.

Discussion

ccTGA is a rare congenital heart disease characterized by discordant AV and ventriculoarterial connections, which contained 4 types: {S, L, L}, {S, L, D}, {I, D, D}, and {I, D, L}. The most common type is {S, L, L}-type ccTGA, and the other types are extremely rare. In the {I, D, D}-

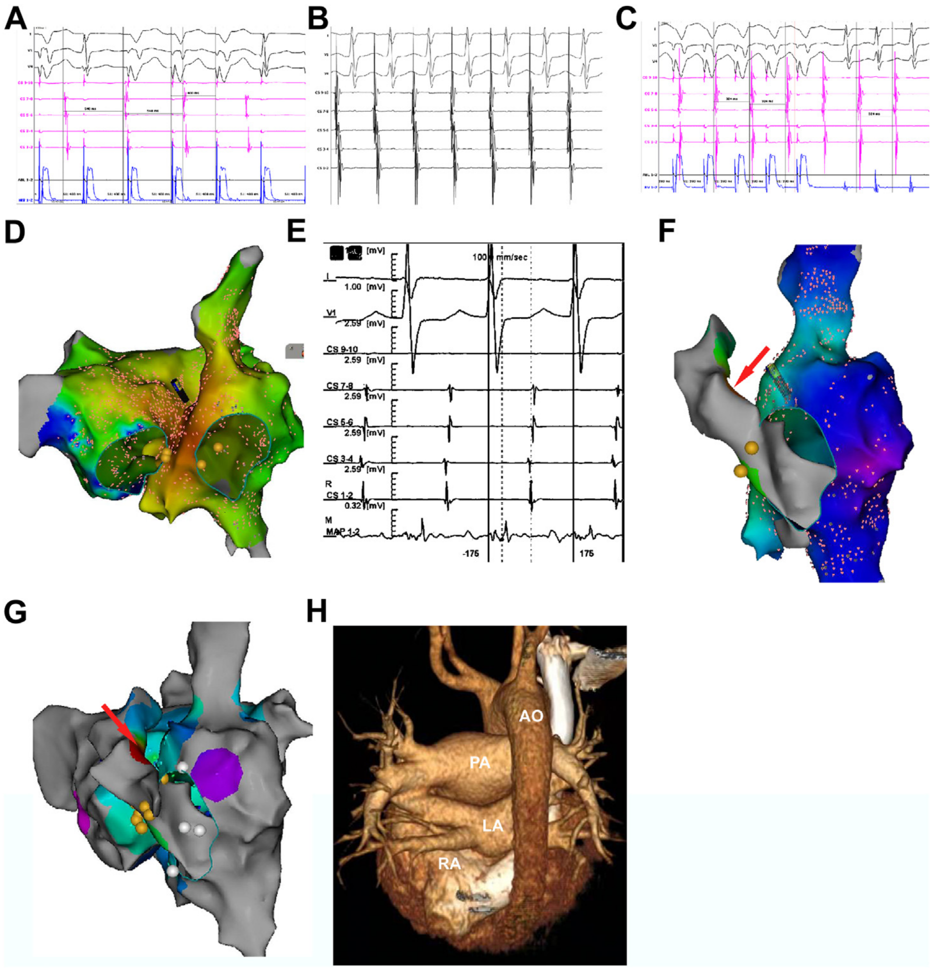

Figure 2 Intracardiac electrograms and activation mapping during atrial tachycardia (AT). **A:** In the sinusoidal state, ventricular pacing (pacing interval 400 ms) showed ventriculoatrial dissociation. **B:** Intracardiac electrograms of AT. **C:** Ventricular overdrive pacing still revealed ventriculoatrial dissociation during tachycardia. **D:** Activation map of both right and left atrium. **E:** The potential in the earliest activation site recorded by ablation catheter. **F:** Activation in the pulmonary outflow tract (*red arrow*) is markedly earlier compared with both right and left atria. **G:** The left and right atrium and pulmonary artery were remapped by the ablation catheter. The earliest activation site (*red arrow*) was consistent with previous mapping result. **H:** {I, D, D}-type ccTGA can be seen in the 3-D reconstructed heart model (posteroanterior position). AO = aorta; LA = left atrium (morphologic right atrium); PA = pulmonary artery; RA = right atrium (morphologic left atrium).

type ccTGA, the right atrium is connected to the morphologic LV across the mitral valve, with the LV connected to the pulmonary artery; the left atrium is connected to the morphologic RV across the tricuspid valve, with the RV connected to the aorta. Previous studies have reported the electrophysiological features and successful ablation of ATs in patients with {S, L, L}-type ccTGA.[3,4] However, the reports about AT from the patients with {I, D, D}-type ccTGA are rare. In this case, using electroanatomical mapping combined with ICE and CT images, the POT is directly adjacent to the interatrial septum. Activation at the POT was significantly earlier than the left and right atrium, and ablation at this site permanently terminated AT.

In normal human hearts, the aortic root occupies a central position in the heart and the noncoronary cusp is immediately adjacent to the atrial myocardium of the interatrial septum. Because of the anatomically adjacent relationship, ablation in the noncoronary cusp becomes a safe and effective strategy to treat para-Hisian accessory pathways or AT.[5,6] Whether {S, L, L}- or {I, D, D}-type ccTGAs, the POT becomes the adjacent vessel between the bilateral atria, which provides the ablation target of AT originated from the anterior septal atria. It remains unclear whether the mechanism for POT AT is arising from atrial tissue or the Bachmann bundle adjacent to the POT, or fibrous tissue (microreentry). Jiang and colleagues[3] found that in 3 cases, double potentials with complex fractionated electrograms were detected, suggesting localized reentry as a possible mechanism, while in the other 2 cases, double potentials were not detected. However, in another case report, multiple ablations were performed to eliminate the AT, suggesting that the POT provides only an access point to ablate the adjacent origin position in the interatrial septum.[7] Noheria and colleagues[8] revealed an unusual AV reentry tachycardia in ccTGAs, and provided much very useful information for differential diagnosis and mapping approaches for tachycardia arrhythmia in patients with ccTGAs. Similarly with the reference images of site ablation provided by Noheria, the earliest activated region in both atria of this case was in the interatrial septum, but it presented a diffuse pattern, which suggested that the real origin site was an adjacent structure rather than the atrial endocardium. Meanwhile, it had no effect of tentative ablation in both sides of the interatrial septum, further verifying our speculation. The EAS located in the POT and the double potentials with complex fractionated electrograms could be seen in this site, which was consistent with previous reports.[3,4] Ablation performed at this site resulted in immediate termination of AT within 3 seconds. Ablation in the POT rapidly terminated AT and no recurrence of AT happened.

In summary, AT in patients with ccTGA is relatively rare, and its ablation is difficult because of the reversed anatomical locations. In addition, care should be taken to avoid ablation injury of the conduction system. Real-time ICE images combined with CT imaging or cardiac magnetic resonance imaging are very useful to construct the true anatomical position relationship and then guide the activated mapping. In order to identify the EAS, it is important to conduct comprehensive activation mapping in the entire left and right atria and in the adjacent structures (such as the POT).

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