

Atrial tachycardia ablation from the pulmonic valve in a patient with congenitally corrected transposition of great arteries



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Introduction

Atrial tachycardia (AT) originating from para-Hisian areas in patients with {S, L, L}-type congenitally corrected transposition of the great arteries (ccTGA) has been previously reported. However, limited data are available for the {I, D, D}-type ccTGA. We present a unique case of a patient with {I, D, D}-type ccTGA and AT originating from the atrial septum and ablation from the pulmonic valve.

Case report

A 21-year-old man with {I, D, D}-type ccTGA presented with frequent palpitations. Palpitations presented as episodes of sudden-onset and sudden-offset palpitations without syncope or presyncope, and could be terminated by vomiting. Electrocardiogram showed a regular supraventricular tachycardia. Symptoms had become nearly incessant over the past month, and he was admitted to the electrophysiology laboratory for evaluation. Preprocedural echocardiography and cardiac computed tomography scan demonstrated anatomy was consistent with {I, D, D}-type ccTGA. The patient also had an atrial septal defect and inflow tract ventricular septal defect.

During the electrophysiological study, a decapolar catheter (DecaNAV; Biosense Webster, Irvine, CA) was advanced into the coronary sinus, and a quadripolar catheter (Biosense Webster) was advanced into the morphological left ventricle. Ventricular pacing at different cycle lengths revealed ventriculoatrial dissociation, which could exclude the existence of a retrograde accessory pathway. Supraventricular tachycardia with a cycle length of 338 ms was easily induced by atrial programmed stimulation. Ventricular

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KEY TEACHING POINTS

- Mapping and ablation in the pulmonic valve are important for atrial tachycardia in patients with congenitally corrected transposition of the great arteries.
- The mechanism of atrial tachycardia is likely to be trigger activity rather than atrial reentry or automaticity.
- That multiple ablations are needed to eliminate atrial tachycardia might illustrate that the pulmonic valve provides only an access point to ablate the adjacent focus in the interatrial septum.

overdrive pacing demonstrated ventriculoatrial dissociation during tachycardia. Therefore, AT was diagnosed. The P wave during AT was narrower than that in sinus rhythm and positive in inferior leads (Figure 1A). Overdrive entrainment of AT twice demonstrated the postpacing interval was variant (Figure 1C and 1D).

A 3.5 mm, deflectable, irrigated catheter (NaviStar ThermoCool; Biosense Webster) was used for mapping and ablation. Early site of activation was first identified along the morphological left atrial septum but was away from the Hisian region (Figure 1G). Radiofrequency was not delivered at this site. Further mapping of the pulmonic valve demonstrated a site of very early activation, at the position directly opposite the atrial septum (Figure 1E–1G). Besides, a much earlier double potential (Figure 1B) was present, with the earliest activation preceding the surface P wave by 64 ms.

Radiofrequency ablation performed at this site resulted in immediate termination of the AT within 3 seconds, but the tachycardia could be reinduced several times. Radiofrequency energy was delivered in a power control mode and titrated from 25 to 30 W, 43°C maximum temperature with

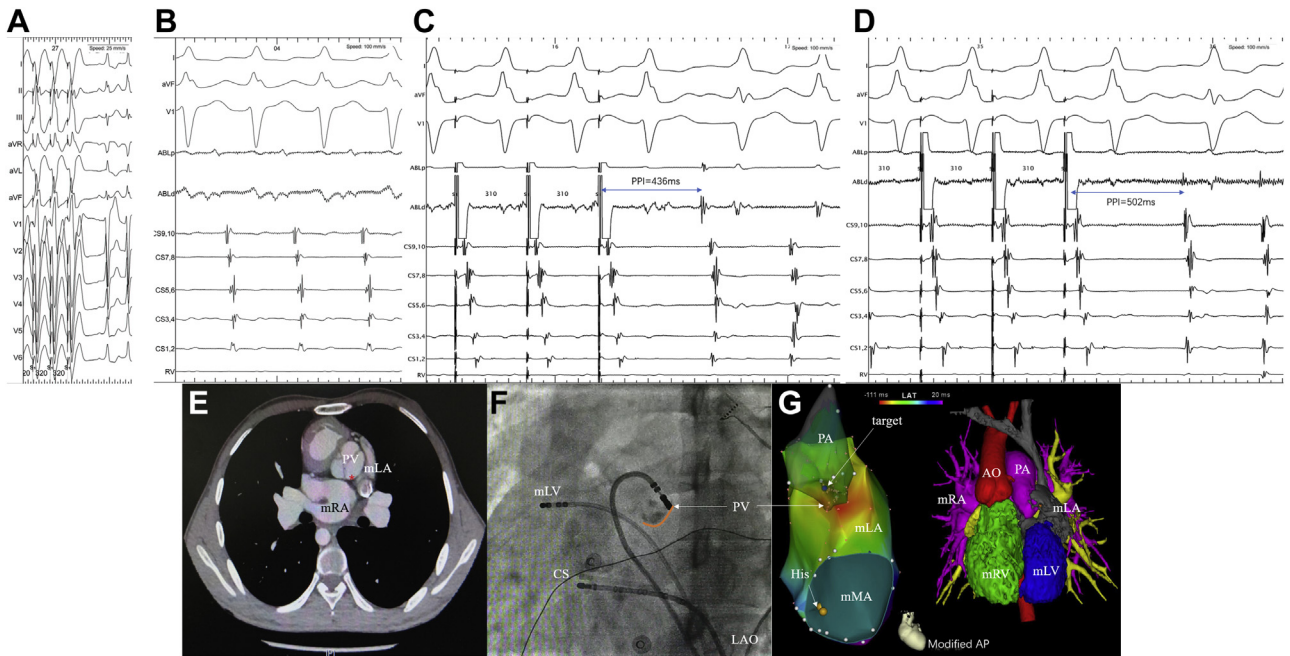


Figure 1 **A:** Electrocardiogram during atrial tachycardia (AT). Limb leads and chest leads were connected in reverse position owing to the mirror-image heart. **B:** Electrogram recorded at the successful ablation site during AT. **C, D:** Various postpacing intervals of overdrive entrainment of the atrial tachycardia at the same site. **E:** Computed tomography slice at the level of the pulmonic valve. The pulmonic valve close to the atrial septum is marked by a red asterisk. **F:** Catheter position via reverse “U” approach at the successful ablation site. **G:** Left panel: Activation map of the morphological right atrium and pulmonic valve. The blue tag denotes the successful ablation target inside the pulmonic valve. Right panel: {I, D, D}-type ccTGA can be seen in the 3-D reconstructed heart model. ABL = ablation catheter; AO = aorta; AP = anteroposterior view; CS = coronary sinus catheter; LAO = left anterior oblique projection; mL A = morphological left atrium; mLV = morphological left ventricle; mMA = morphological mitral annulus; mRA = morphological right atrium; mRV = morphological right ventricle; PA = pulmonary artery; PPI = postpacing interval; PV = pulmonic valve.

an irrigation rate of 17 mL/min. Additional lesions were delivered to consolidate the ablation surrounding the target site and AT became noninducible, despite aggressive programmed stimulation, burst pacing, and isoproterenol infusion. A total of 7 radiofrequency applications were performed and the overall radiofrequency time was 420 seconds. No atrioventricular block or other complications occurred. The patient was discharged home the following day, and was regularly followed up with no recurrent arrhythmias over 3 months.

Discussion

Several studies have previously reported the electrocardiogram and electrophysiological features as well as successful ablation of focal ATs only in patients with {S, L, L}-type ccTGA.¹⁻⁴ The majority of ATs shared the common features of anteroseptal ATs, eg, comparatively narrow P wave, positive in leads I and aVL, negative/positive in leads V₁-V₂, and negative/positive or isoelectric in inferior leads.¹ The P wave during AT was relatively narrower in the present case. However, the inferior leads of the P wave during AT were positive, which was different from the reported cases with {S, L, L}-type ccTGA. It could be explained by its anatomy that the junction between the pulmonary artery and atrium was located at the superior septal atrium (Figure 1G).

Previous studies^{1,3} speculated that the tachycardia mechanism was microreentry, by observing the complex fractionated electrograms or the conduction delay between the double potentials. During the electrophysiological study in the present case, the clinical tachycardia could be reproducibly induced and terminated by atrial stimulation. In addition, overdrive entrainment of the AT twice at the same place exhibited various postpacing intervals. Therefore, we supposed that the tachycardia mechanism is likely to be trigger activity rather than atrial reentry or automaticity.

It is to be noted that previous studies¹⁻⁴ emphasized the importance of mapping and ablation in the pulmonic valve and beyond. For one reason, in patients with ccTGA, the anatomical structure is unique, with the pulmonary artery instead of the aorta in close anatomical relation to the paraseptal region. For another reason, as ablation in the noncoronary cusp eliminates anteroseptal AT,⁵ ablation in the pulmonary valve was effective without damaging the conduction system. A notable feature first demonstrated in our previous study is that the earliest activation site along with a double potential was observed in the pulmonic valve in some cases.¹ In the present type of malformation, the pulmonary artery instead of the aorta overlies the paraseptal region as well, which provided the underlying anatomy to eliminate AT. During mapping, a much earlier double potential was recorded in the pulmonic valve, which was consistent

with those cases with {S, L, L}-type ccTGA. Whether atrial myocardium extends into the pulmonic valve serving as tachycardia origin remains a matter of debate. However, in this case, multiple ablations were applied to eliminate the AT. Therefore, we suppose that the pulmonic valve provides only an access point to ablate the adjacent focus in the interatrial septum.

To the best of our knowledge, this is the first case described in the literature of ablation focal AT in patients with {I, D, D}-type ccTGA in the pulmonic valve. This finding in {I, D, D}-type ccTGA is consistent with findings in {S, L, L}-type ccTGA that ablation from the pulmonic valve could successfully eliminate the arrhythmia.

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