Case Report

Catheter Ablation of Left Atrial Posterior Wall Tachycardia Guided by Electroanatomic Mapping in a Young Patient

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Abstract

Focal atrial tachycardias (ATs) arising from the left atrium (LA) most commonly originate from the ostium of the pulmonary vein, the superior mitral annulus, the body of the coronary sinus, the LA septum, and the LA appendage. Focal ATs originating from the posterior wall of the LA are extremely rare. A 34-year-old male patient presented to the cardiology outpatient clinic complaining of palpitation. Electrocardiography showed a tachycardia at a ventricular rate of 150 bpm and a narrow QRS complex. Therefore, an electrophysiological study was performed, which was consistent with an AT. The patient underwent an electrophysiological study in tachycardias with narrow QRS complexes. The diagnostic electrophysiological findings were consistent with an AT. The AT cycle length was found to be 405 ms with variability in the ventriculoatrial interval. Simultaneous LA anatomical and activation mapping was performed during the AT using a 3D electroanatomic mapping system (CARTO) and a quadripolar unidirectional irrigated tip catheter. The activation mapping revealed that the earliest endocardial activation site was at the posterior wall of the LA, where the local electrogram was 72 ms and 35 ms before the coronary sinus reference and the P-wave onset, respectively. The activation mapping also showed centrifugal spreading and mid-diastolic, fractionated signals on the posterior wall. Radiofrequency ablation was successfully performed with 30-watt power at the site of the earliest atrial activation, with a fractionated electrogram terminating the tachycardia. LA posterior ATs are a rare form of AT. The electroanatomic mapping method enables the accurate localization of the LA focal tachycardia, and a high success rate is achieved with ablation therapy.

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Introduction

Atrial tachycardias (ATs) account for nearly 10% of all supraventricular tachycardias. The success rate of the catheter ablation method is high in ATs refractory to medical therapy. ATs may be classified as focal or macroreentrant, depending on the electrophysiological mechanism. Abnormal automaticity, microreentry, and triggered activity may play a role in the development of focal ATs,[1] which most often originate from the crista terminalis, the coronary sinus,

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the tricuspid annulus, or the para-Hisian region of the right atrium. Focal ATs arising from the left atrium (LA) most commonly originate from the ostium of the pulmonary vein, the superior mitral annulus, the body of the coronary sinus, the LA septum, and the LA appendage. Focal ATs originating from the posterior wall of the LA are extremely rare.[2, 3]

Left ATs may occur after circumferential pulmonary vein ablation, with an incidence ranging from 2.5% to 20%. ATs occurring after circumferential pulmonary vein ablation are often macroreentrant but may also be focal.[4] These arrhythmias can be successfully mapped and ablated by using an electroanatomic mapping system.

This manuscript reports the successful radiofrequency ablation of an AT using a 3D electroanatomic mapping method in a young patient who developed tachycardiomyopathy due to a focal AT originating in the posterior wall of the LA.

Case Report

A 34-year-old male patient presented to the cardiology outpatient clinic complaining of palpitation and dizziness. Electrocardiography showed a tachycardia at a ventricular rate of 150 bpm and a narrow QRS complex (Figure 1). Blood pressure was 120/70 mmHg. In his clinical history, the patient had been using amiodarone (200 mg) twice daily for approximately 4 years due to palpitation. The palpitation episodes persisted despite the medical therapy, however. The physical examination was normal (blood pressure=120/70 mmHg), as were serum electrolyte levels and other biochemical and hematological analyses. A transthoracic echocardiographic examination revealed an ejection fraction of 47% with normal left ventricular diameters. A 24-hour rhythm Holter recording showed repetitive tachycardias with narrow QRS complexes starting with a "warm-up" pattern. No atrial fibrillation (AF) was detected. Given the patient's tachycardiomyopathy while receiving medical therapy, an electrophysiological study was performed after obtaining his consent. A transesophageal echocardiographic examination was performed before ablation to exclude LA thrombi.

The patient underwent an electrophysiological study in tachycardias with narrow QRS complexes. Two 6-F quadripolar catheters and one 7-F Duo-Decapolar catheter (HALO XP, Tricuspid mapping catheter, Biosense Webster, USA) were placed into the coronary sinus, the right ventricular apex, and the right atrium, respectively. An examination of right atrial activation with the Halo catheter showed activation spanning of less than 50% of the tachycardia cycle length (Figure 2A). The AT cycle length was found to be 405 ms with variability in the ventriculoatrial interval (Figure 2B). Overdrive ventricular pacing was performed to entrain the tachycardia, and the post-pacing response was noted to be VAAV: the diagnostic electrophysiological findings were consistent with an AT.

The decision to carry out LA activation mapping was made due to a positive P wave in the V1 lead during the tachycardia, with the earliest atrial activation being detected in the coronary sinus 5-6 electrode. After a trans-septal puncture, intravenous heparin was administered to maintain the activated clotting time at 300 to 350. Simultaneous LA anatomical and activation mapping was performed during the tachycardia using a 3D electroanatomic mapping system (CARTO) and a deflectable quadripolar unidirectional irrigated tip catheter (NAVISTAR, Biosense Webster, Diamond Bar, CA, USA). A stable coronary sinus signal was selected as a reference during a tachycardia. The activation mapping revealed that the earliest endocardial activation site was the posterior wall of the LA, where the local electrogram was 72 ms and 35 ms before the coronary sinus reference and the P-wave onset, respectively. Additionally, the activation mapping showed centrifugal spreading and mid-diastolic, fractionated signals on the posterior wall (Figure 3, Panel A).

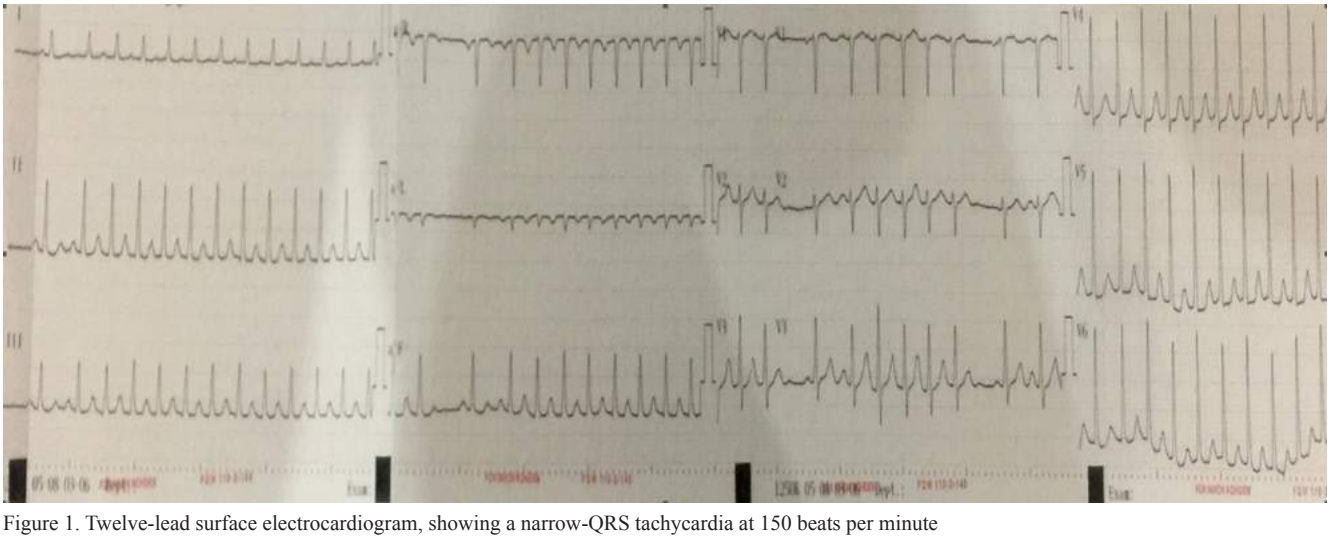

Figure 1. Twelve-lead surface electrocardiogram, showing a narrow-QRS tachycardia at 150 beats per minute

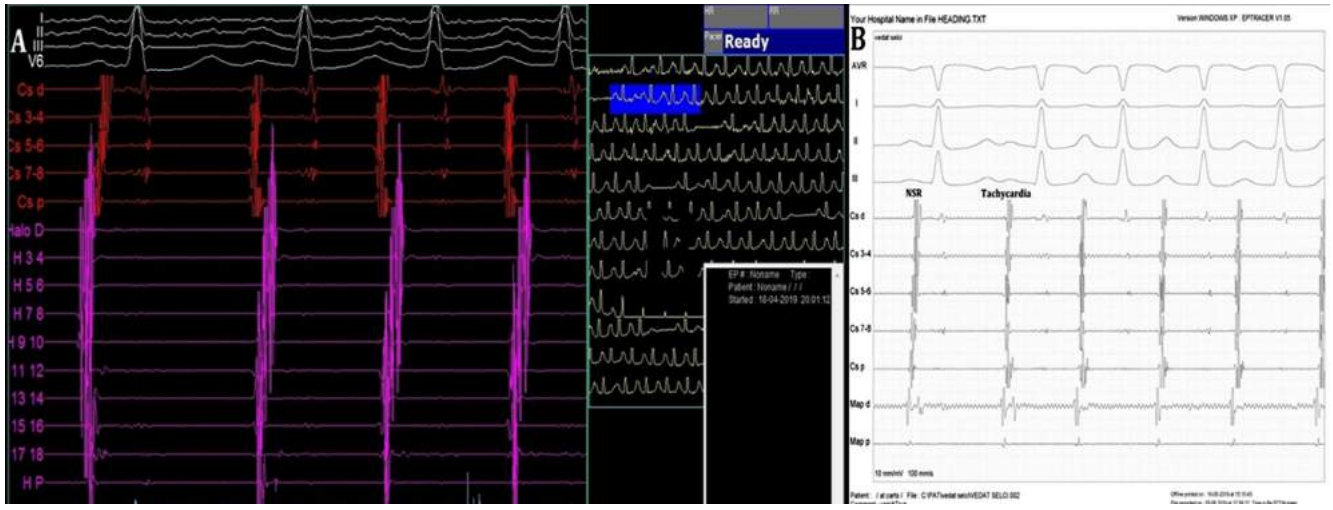

Figure 2.

A) Intracardiac electrograms, showing right and left atrial activation sequences

B) Surface electrogram leads I, II, III, and aVR, together with intracardiac electrograms from the coronary sinus catheter, during left atrial posterior wall tachycardia

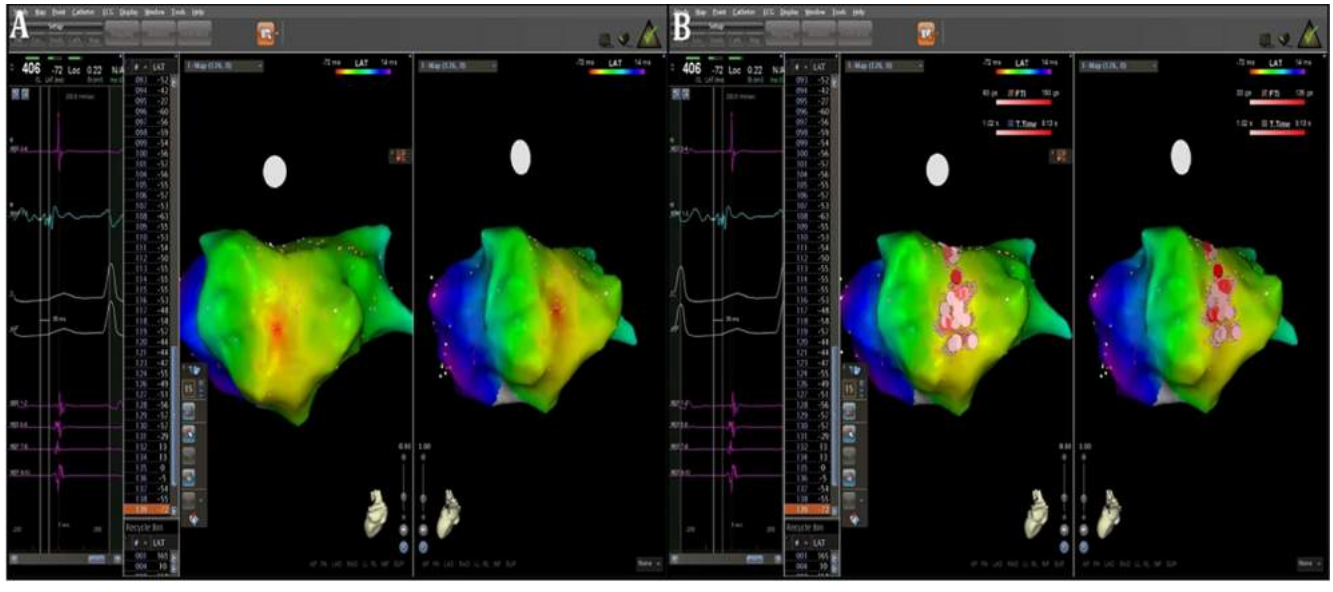

Figure 3.

Panel A: Simultaneous left atrial anatomical and activation mapping by using a 3D electroanatomic mapping system (CARTO) during tachycardia (The earliest endocardial activation site was the left atrial posterior wall, where the local electrogram was 72 ms and 35 ms before the coronary sinus reference and the P-wave onset, respectively. The activation mapping also showed centrifugal spreading and mid-diastolic, fractionated signals on the posterior wall.)

Panel B: Successful ablation site in the left atrial posterior wall

Radiofrequency ablation was performed with 30-watt power (maximum temperature=42˚C and infusion rate=17 mL/min) at the site of the earliest atrial activation, with a fractionated electrogram terminating the tachycardia (Figure 3, Panel B). Following the radiofrequency ablation, tachycardias could not be induced with programmed atrial extrastimulation and burst atrial pacing by isoproterenol infusion in a 30-minute period, and the sinus rhythm persisted (Figure 4).

Discussion

LA focal tachycardias occur less frequently than right atrial focal tachycardias, although there has been an increase in the prevalence of LA tachycardias due to the more frequent use of ablation therapy for AF.[5] In a paroxysmal AF, pulmonary vein isolation is sufficient to treat AF in the vast majority of patients. Very few ATs are observed in such contexts inasmuch as they are usually localized small reentry circuits

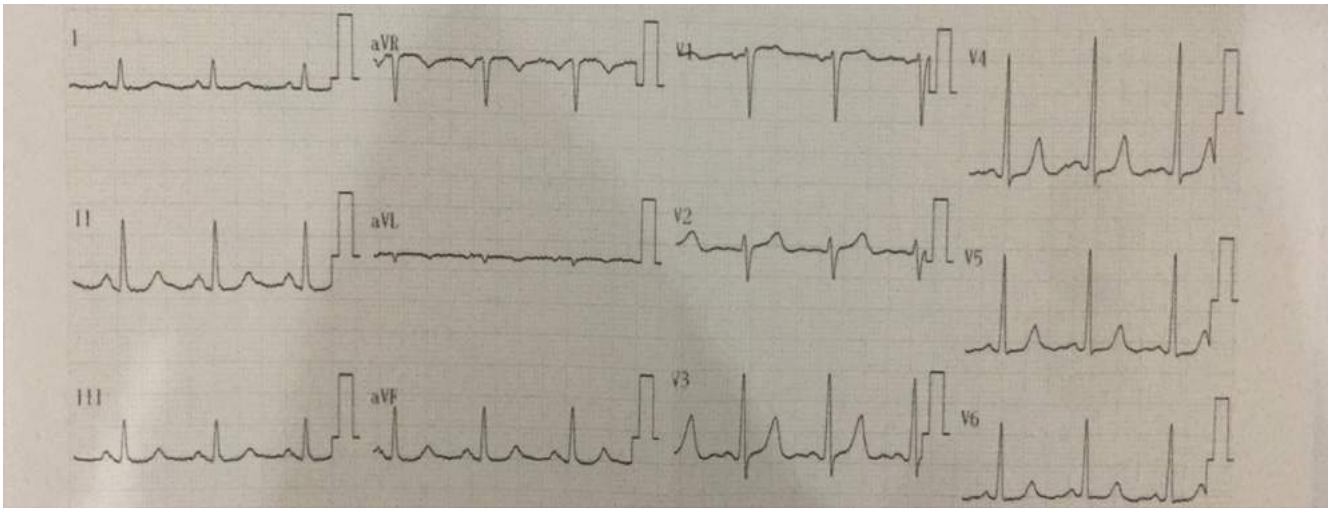

Figure 4. Post-ablation 12-lead surface electrocardiogram, showing the sinus rhythm

at the pulmonary vein ostium associated with the formation of gaps in the previous pulmonary vein isolation lesion. In the era of the circumferential pulmonary vein isolation involving higher volumes of ablated tissue, few patients also develop roof-dependent and/or perimitral circuits.[6] In persistent AF whose termination requires more extensive ablation, all types of ATs may be observed. Notably, patients with ATs after previous linear ablations (roof and mitral isthmus lines) for AF frequently show macroreentrant circuits involving the LA, with perimitral flutters being the most common, followed by roof-dependent ATs. Cavotricuspid isthmus-dependent ATs are other likely macroreentrant right atrial tachycardias, but they are the least common type.[7]

In the literature, rare focal LA tachycardias have been reported originating from the residual gaps at the linear lesions on the anterior aspect of the pulmonary vein and the septal aspect of the right pulmonary vein following circumferential pulmonary vein ablation.[8] LA tachycardias in patients with no history of AF ablation most commonly arise from the pulmonary vein ostium and the mitral annulus. In a study by Dong et al,[9] who examined focal LA tachycardias, the most common localizations were the pulmonary vein ostium and the mitral annulus, whereas focal ATs originating from the posterior wall of the LA were rarely observed. Chen et al[10] evaluated the localization of ATs and substrate characteristics in patients who underwent surgery for rheumatic mitral valve disease and reported a 33% prevalence rate for LA tachycardias, with focal ATs originating from the posterior wall of the LA being observed in only 2 patients. They stated that atrial electrical and structural remodeling due to rheumatic inflammation produced slow conduction, low-voltage areas and the line of conduction block caused the development of LA tachycardias. Thus, Chen and colleagues reported that the ATs in the 2 deceased patients had a focal origin, localizing on the posterior wall at the edge of a scar.

We did not perform LA voltage mapping for our patient due to his young age, the lack of evidence of valvular heart disease on echocardiography, and the absence of a documented AF on Holter rhythm monitoring. However, a fragmented atrial electrogram was noted at the successful ablation site where the earliest atrial activation was observed. We hypothesize that the fractionation at this site indicates an underlying slow conduction, making microreentry possible.

Conclusion

LA posterior atrial tachycardias are a rare form of atrial tachycardias. The electroanatomic mapping method enables the accurate localization of the LA focal tachycardia, and a high success rate is achieved with ablation therapy.

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