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CASE REPORT

ADVANCED

CLINICAL CASE

Focal Atrial Tachycardia Arising From the Posterior Wall of the Left Atrium

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ABSTRACT

Potential foci for atrial tachycardia have been previously described in various locations including crista terminalis, tricuspid annulus, coronary sinus ostium, pulmonary vein ostia. In this report, we present a case of a focal atrial tachycardia arising from the posterior wall of the left atrium which has not been described before. (**Level of Difficulty: Advanced.**) (J Am Coll Cardiol Case Rep 2022;4:192–197) © 2022 The Authors. Published by Elsevier on behalf of the American College of Cardiology Foundation. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

A 73-year-old woman with paroxysmal supraventricular tachycardia (SVT) presented with worsening palpitations. She arrived at the emergency department with several hours of severe palpitations and chest tightness and was found to have incessant nonsustained SVT. She was admitted to the hospital for management of the SVT after acute coronary syndrome was ruled out.

LEARNING OBJECTIVES

- To understand that surface P-wave vector analysis is useful in predicting the origin of atrial tachycardia before ablation, but it is not without limitations.
- To recognize that focal atrial tachycardia could arise from the posterior wall of the left atrium in patients with a structurally normal left atrium.

MEDICAL HISTORY

She had experienced palpitations for a couple of years before this hospitalization. Initially, these episodes were brief and infrequent, occurring about once a month. However, the frequency and severity of her palpitations worsened. Within the past month, she began to have daily palpitations, associated with chest tightness, headache, and tremulousness. She had also presented 2 weeks prior for severe palpitations to an outside hospital emergency department, where she was found to have SVT and discharged home with metoprolol succinate.

DIFFERENTIAL DIAGNOSIS

The cause of her SVT was likely atrial tachycardia, based on her electrocardiogram. Other possible diagnoses included atrioventricular nodal reentry tachycardia and atrioventricular reentrant tachycardia.

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MANAGEMENT

After her admission, she was administered diltiazem and flecainide. Unfortunately, she continued to have symptomatic incessant episodes of SVT, likely atrial tachycardia. A transthoracic echocardiogram showed normal left ventricular size and function, normal size of the left atrium, and no significant valvular heart disease. The origin of her clinical atrial tachycardia was suspected to be on the right side, based on negative P-wave morphology in lead V_1 (**Figure 1**).[1]

Owing to the refractory nature of her SVT, an electrophysiology study (EPS) was performed while she was under conscious sedation by use of a Carto 3-dimensional mapping system (Biosense Webster). Her clinical atrial tachycardia cycle length was 450 ms, with earliest atrial activation in the distal coronary sinus. Right atrial activation mapping by use of a Pentaray mapping catheter (Biosense Webster) localized an area of earliest activation with fractionated signals at the septum of the superior vena cava (SVC), which was 36 ms earlier than in the distal coronary sinus (CS) 1-2 (**Figures 2 and 3**). A series of radiofrequency ablation lesions at 30 Watts for 30 to 60 seconds were delivered with immediate but brief suppression of the tachycardia. At this time, the earliest activation of the SVC septum was suspected to be passive activation from the left atrium. A transseptal access was obtained with a transseptal needle under intracardiac echocardiography guidance. An activation mapping was performed in the pulmonary veins and the left atrium with the mapping catheter. An area of earliest activation (30 ms earlier than in the SVC septum) with fractionated signals and sharp QS on unipolar was localized to the posterior wall of the left atrium (**Figure 4,** Video 1). A series of radiofrequency lesions at 30 W for 30 to 60 seconds was delivered, resulting in elimination of the tachycardia. Esophageal temperature was closely monitored with an esophageal temperature probe (**Figure 5**). There was no inducible atrial tachycardia or other supraventricular tachycardia with isoproterenol challenge and programmed atrial or ventricular stimulation during the 30-minute waiting period.

ABBREVIATIONS AND ACRONYMS

CS = coronary sinus

EPS = electrophysiology study

SVC = superior vena cava

SVT = supraventricular tachycardia

DISCUSSION

We present the case of a patient with incessant atrial tachycardia despite medical therapy that necessitated an EPS and ablation. Her clinical arrhythmia had a negative P-wave in lead V_1, and positive P waves in inferior leads (II, III, aVF), I, aVL, suggesting a right-sided focus. Activation mapping of the right atrium localized an area of earliest activation with

FIGURE 1 Electrocardiogram of Clinical Atrial Tachycardia at Speed 25 mm/s

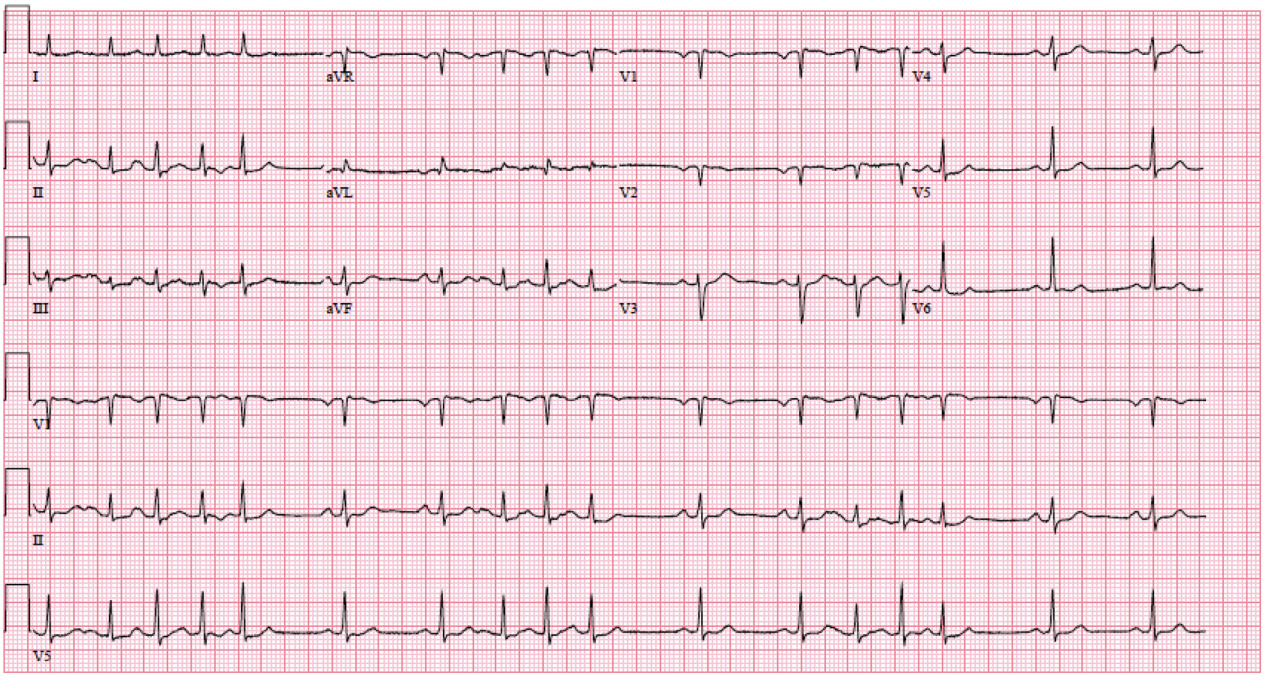

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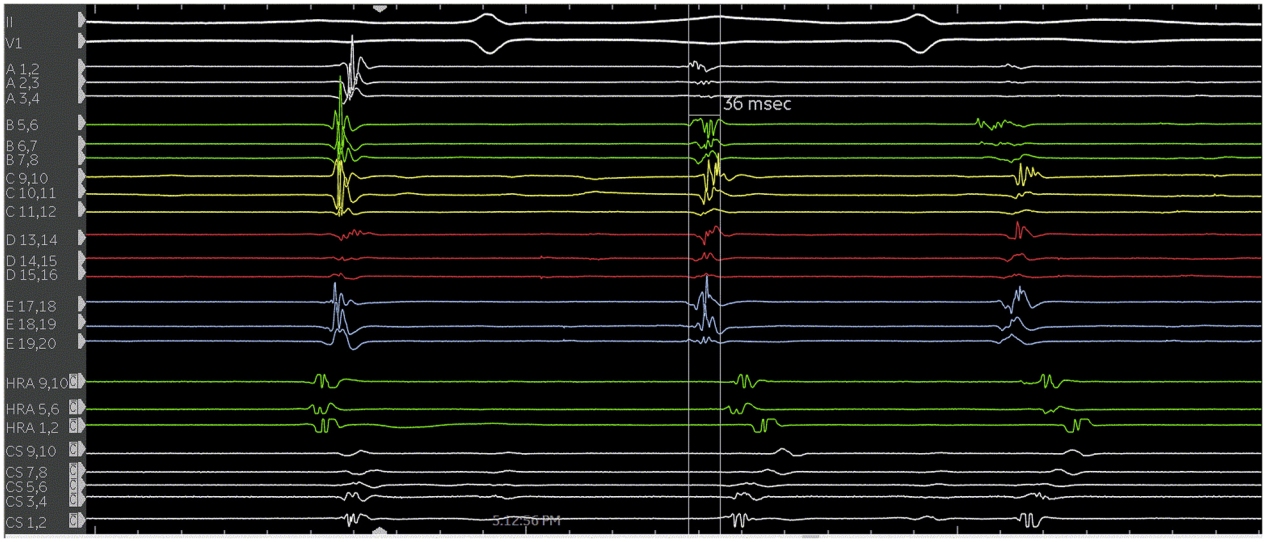
FIGURE 2 **Intracardiac Recording of SVC, CS, and Right Atrium**



Intracardiac recordings of superior vena cava (SVC), right atrium, and coronary sinus (CS) during tachycardia. A1,2 through E19,20 are mapping catheter recordings of SVC. HRA is the recording of the right atrium. CS1,2 and C9,10 are recordings of distal and proximal CS, respectively. During atrial tachycardia, the earliest signal was recorded in A1,2 on the septal SVC, which was 36 ms earlier than the earliest atrial signal (HRA5,6).

fractionated signals at the septum of the SVC. However, ablation within this area only briefly suppressed the tachycardia without eliminating It. This prompted activation mapping of the left atrium, which localized an area of earliest activation (37 ms earlier than in the SVC septum), with fractionated signals in the posterior wall of the left atrium. Ablation within this area resulted in immediate suppression and termination of the tachycardia. This case illustrates 2 important points. First, surface P-wave vector analysis is useful in predicting the origin of atrial tachycardia before ablation but is not without limitation. Second, a focal atrial tachycardia could arise from the posterior wall of the left atrium in patients with a structurally normal left atrium, which has not been previously described.

During the EPS, we found an area of earliest activation in the SVC septum, which was later confirmed as passive activation from the posterior left atrium. This could be explained by 2 main bundles—Bachmann and septopulmonary bundles—connecting the right and left atria electrically. The Bachmann bundle consists of parallel cardiomyocyte fibers, coursing from the right atrium and SVC junction over the anterior wall of the left atrium to the left atrial appendage. The septopulmonary bundle consists of longitudinal cardiomyocyte fibers, arises directly below the Bachmann bundle, and spans over the posterior wall of the left atrium before extending out to pulmonary veins.[2] In our case, we postulate that increased automaticity developed in the cardiomyocytes near or within the septopulmonary bundle on the posterior wall, and the cardiomyocytes became a focus for atrial tachycardia. The activation wavefront then spread along the longitudinal fibers over to the Bachman bundle and then to the junction of the right atrium and the SVC, resulting in early activation in this area. Thus, we believe this is the reason why ablation of the posterior wall resulted in elimination of the tachycardia.

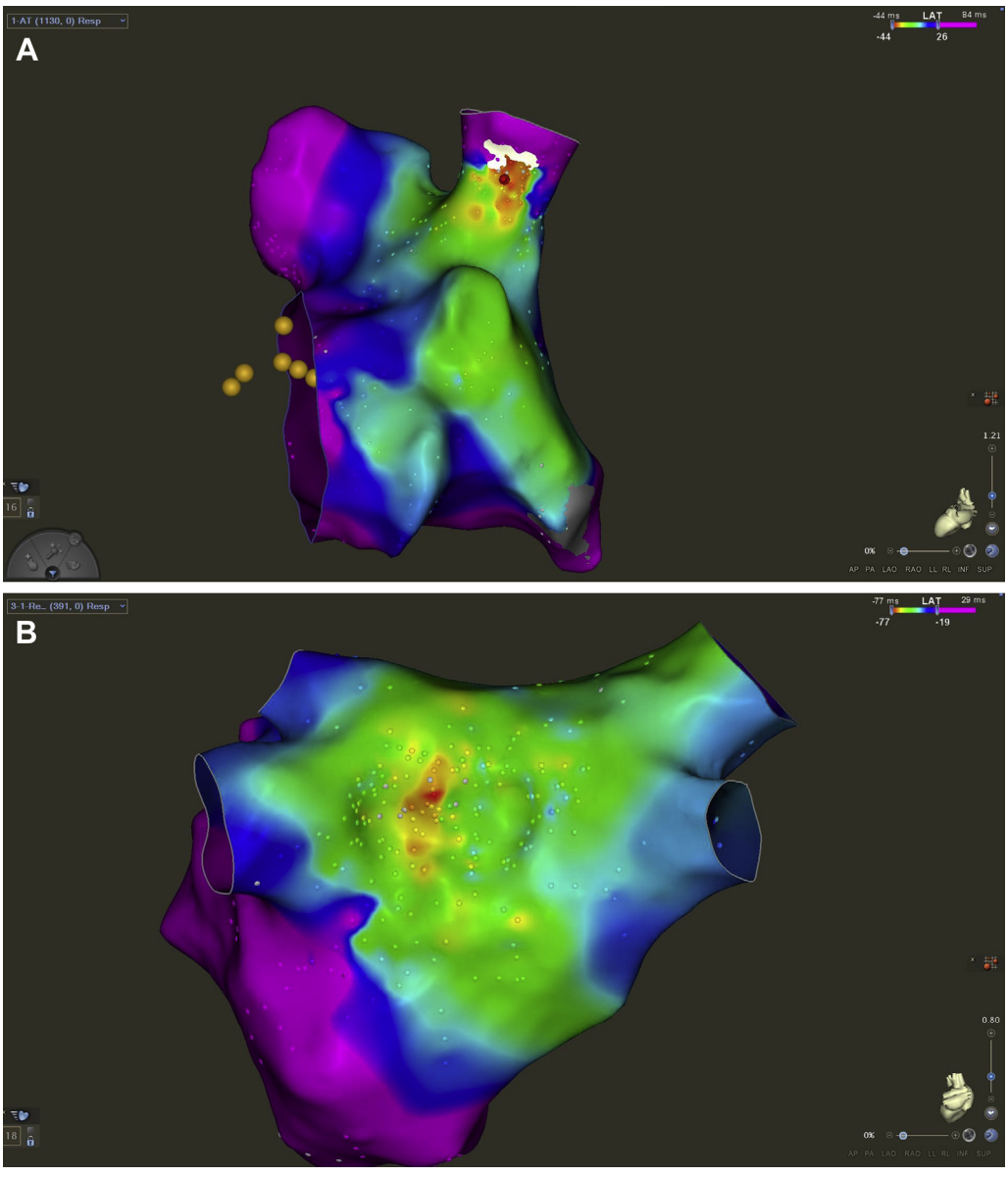
FOLLOW-UP

The patient was discharged home the day after ablation with a 4-week course of apixaban 5 mg twice daily and omeprazole 40 mg twice daily. Metoprolol, diltiazem, and flecainide were discontinued. She was doing well without palpitations at her 6-month follow-up visit.

CONCLUSIONS

Crista terminalis, tricuspid annulus, coronary sinus ostium, pulmonary vein ostia, mitral annulus, left atrial appendage, and left-sided septum have been reported as potential foci for atrial tachycardia.[3-9] In this report, we describe a case in which the posterior

FIGURE 3 **Activation Map of the Right and Left Atria**



(A) Activation map of the right atrium with earliest activation in the septum of the superior vena cava. **(B)** Activation map of the left atrium with earliest activation in posterior wall of the left atrium.

wall of the left atrium could also be a focus for focal atrial tachycardia in a structurally normal heart.

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The authors have reported that they have no relationships relevant to the contents of this paper to disclose.

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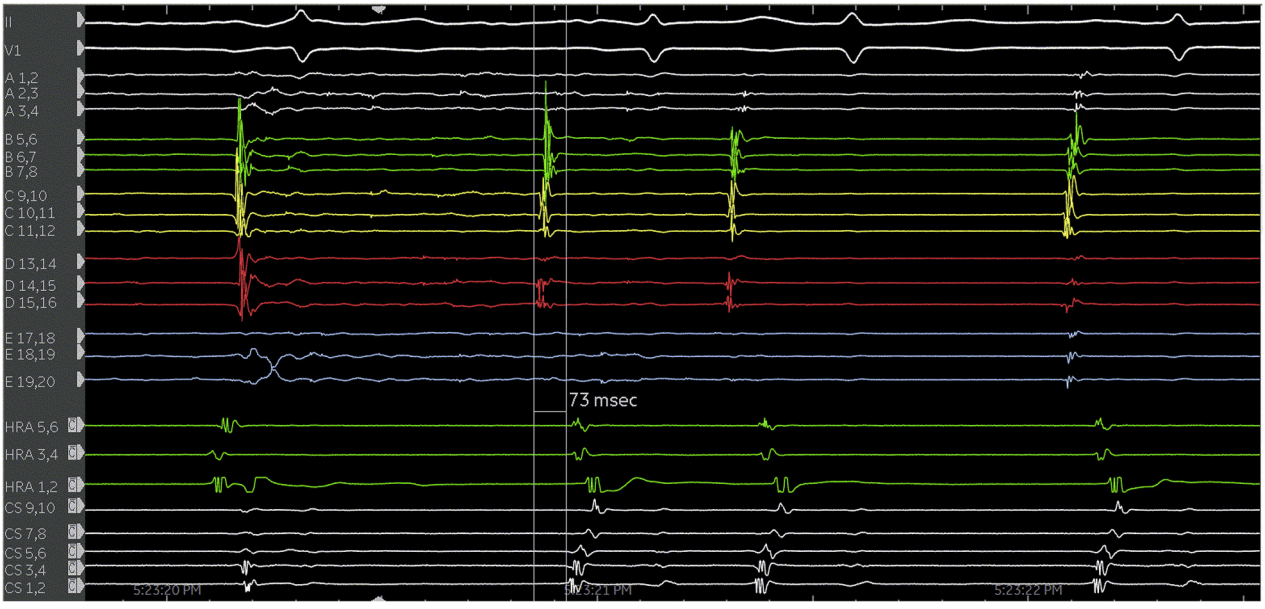
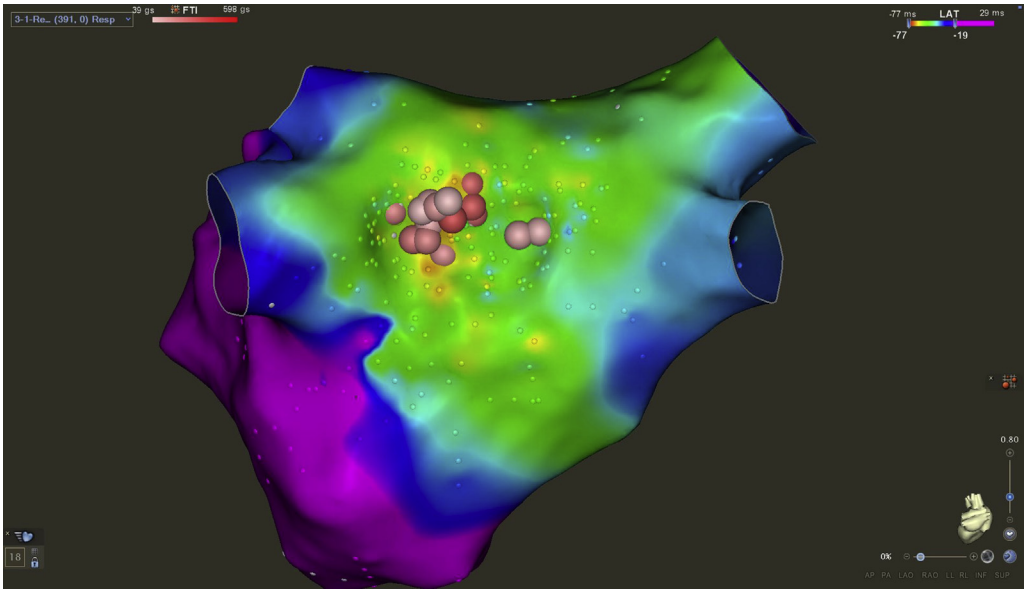
FIGURE 4 **Intracardiac Recording of the Left Atrium, Right Atrium, and CS**



Intracardiac recordings of posterior wall of the left atrium, right atrium, and coronary sinus (CS) during tachycardia. A1,2 through E19,20 are mapping catheter recordings of posterior wall of the left atrium. HRA is the recording of the right atrium. CS1,2 and C9,10 are recordings of distal and proximal CS, respectively. During atrial tachycardia, the earliest signal was recorded in B6,7 on the LA posterior wall, which was 73 ms earlier than the earliest atrial signal (CS1,2). A radiofrequency lesion to this area terminated the tachycardia.

FIGURE 5 **Location of Radiofrequency Lesions on Carto Map in Posteroanterior Configuration**



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KEY WORDS ablation, atrial tachycardia, electroanatomical mapping

APPENDIX For a supplemental video, please see the online version of this article.