Pre-excited tachycardia: Atrial tachycardia with a bystander left lateral accessory pathway

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Introduction

Wide complex tachycardias have a broad differential diagnosis including ventricular tachycardia (VT), antidromic reciprocating tachycardia (ART), supraventricular tachycardia (SVT) with aberrancy, atrial tachycardia (AT) or atrioventricular nodal reentry tachycardia (AVNRT) with a bystander accessory pathway, or unusual tachycardias involving nodoventricular or atriofascicular pathways. Diagnostic criteria for differentiating VT from SVT with aberrancy have been developed, all having similar diagnostic accuracy, but are limited in the ability to distinguish VT from pre-excited tachycardias because morphology on surface electrocardiogram (ECG) is identical to VT originating from the basilar segment of the ventricle.[1,2] In this case of wide complex tachycardia, we use observations of intracardiac electrograms during tachycardia as well as a single atrial extrastimulus to make a diagnosis in our patient.

Case presentation

A 64-year-old woman with a long-standing history of palpitations was referred to the electrophysiology service after a recurrent episode that required an emergency department visit. This episode persisted despite her attempts to stop the palpitations by breath holding or splashing cold water on her face. In the emergency department a 12-lead surface ECG was performed showing a wide complex tachycardia (Figure 1, *top*). Adenosine 6 mg intravenous rapid bolus was administered, followed by 2 separate additional boluses of 12 mg each without termination of the tachycardia. External synchronized DC cardioversion was then performed with successful termination of the tachycardia and restoration of normal sinus rhythm.

Prior cardiac evaluation had shown a structurally normal heart. A 12-lead ECG in sinus rhythm (Figure 1, *bottom*) revealed ventricular pre-excitation having a negative delta wave in lead I and positive delta wave with R > S in lead V1, consistent with a left lateral accessory pathway.[3]

The patient underwent an electrophysiology study after withholding cardioactive medications for 5 half-lives. Catheters were placed in the high right atrium, coronary sinus (CS), His bundle region, and right ventricular apex.

Differential atrial pacing confirmed the presence of a left lateral accessory atrioventricular (AV) pathway with progressive ventricular pre-excitation when pacing from proximal to more distal CS catheter poles and a negative HV interval. Antegrade conduction block of the accessory pathway occurred at 220 ms during decremental atrial pacing. The antegrade effective refractory period of the accessory pathway was 300 ms for a drive cycle length of 500 ms and 260 ms for a drive cycle length of 400 ms.

Right ventricular pacing revealed eccentric retrograde atrial activation with the earliest atrial activation in the mid to distal CS. Ventricular extrastimuli showed no evidence of retrograde dual AV node physiology.

Programmed stimulation with atrial extrastimuli did not induce any arrhythmias. Atrial burst pacing from distal CS catheter poles induced a wide complex tachycardia with a 1:1 AV association (Figure 2, *top*). The QRS, with a right bundle branch block–like morphology, during the tachycardia was identical to that during maximal ventricular pre-excitation when pacing at the distal CS. Single atrial extrastimuli were introduced during tachycardia from the distal CS and the same finding was repeatedly observed (Figure 2, *bottom*) and was diagnostic of AT.

A transseptal approach was used for mapping and successful ablation of the left lateral accessory pathway owing to rapid conduction (conduction block 220 ms). Localization of the accessory pathway was achieved by mapping in the left atrium along the lateral mitral annulus in the area adjacent to the CS catheter poles that achieved maximum pre-excitation with pacing. The specific location of the accessory pathway was identified by the presence of an accessory pathway potential (Figure 3, *top*), representing the mid-body portion of the accessory pathway.[4,5] Atrial burst pacing induced tachycardia once again at the same cycle length but now with a narrow QRS complex, and the AT was mapped along the anterior interatrial septum with the earliest

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KEY TEACHING POINTS

- Algorithms to differentiate the mechanism of wide complex tachycardias cannot be applied in cases of pre-excited tachycardias. They are typically applicable in diagnosing ventricular tachycardia vs supraventricular tachycardia with aberrancy.
- Atrial extrastimuli introduced during a wide complex tachycardia is a useful maneuver for eliciting the mechanism of tachycardia.
- Accessory pathways that have rapid antegrade conducting characteristics may be ablated despite not being integral to the mechanism of the clinical arrhythmia, since they may be associated with an increased risk of sudden death.

local atrial electrogram found at the proximal His catheter, which was 31 ms earlier than the onset of the surface P wave. The mapping catheter was then advanced retrograde to the aortic root and additional mapping was performed in the non-coronary cusp, and the earliest local atrial electrogram there was 35 ms earlier than the onset of the surface P wave. Ablation was successful within the non-coronary cusp at this earliest activation point with fluoroscopy and intracardiac echo guidance (Figure 3, *bottom*). The tachycardia terminated within 5 seconds of initiation of delivery of radiofrequency energy, which was continued for a total of 60 seconds. Repeat atrial burst pacing was performed post-ablation with and without isoproterenol infusion, demonstrating noninducibility of the tachycardia post-ablation, in contrast to pre-ablation, when the tachycardia was easily inducible. The patient remains asymptomatic 6 months post procedure.

Discussion

Wide QRS tachycardia with 1:1 AV association has the following differential diagnosis:

1. VT with retrograde conduction.
2. ART.
3. AVNRT or AT with bystander pathway antegrade conduction, either AV or fasciculoventricular.
4. Orthodromic reciprocating tachycardia, AVNRT, or AT with aberrancy.
5. Tachycardia involving a manifest nodoventricular or nodofascicular pathway.

Tachycardia was persistent after a blocked atrial extrastimulus and subsequent narrow QRS, as well as variations in AA intervals dictating the change in ventricular cycle length, suggested that the origin of tachycardia was supraventricular. Additionally, using commonly employed criteria including Brugada, Vereckie, R wave to peak time, and Griffith algorithm would have misclassified the rhythm as VT. Employing likelihood ratios with the Bayesian methodology would have resulted in a very high likelihood ratio.[1] The clear limitation of these algorithms is when a wide complex tachycardia is the result of SVC with pre-excitation, which has an identical QRS morphology to a VT originating from the base of the ventricle.[2] Aberrancy was excluded, since the right bundle branch–like morphology in V1 was monophasic and lead V6 had an rS pattern. A fasciculoventricular pathway was excluded because differential atrial pacing produced progressive pre-excitation.[6] The QRS morphology during tachycardia was the same as pre-excitation during normal sinus rhythm, suggesting either ART or bystander pre-excitation with AVNRT or AT. A diagnosis of ART was excluded because introduction of an atrial extrastimulus when the septal A was committed did not conduct to the ventricle and did not terminate the tachycardia.

If the tachycardia was AVNRT, it was likely atypical AVNRT because of the VA time. The following features made atypical AVNRT extremely unlikely: (1) lack of dual AV node physiology; (2) tachycardia always terminated with a ventricular electrogram; (3) only atrial burst pacing and not atrial extrastimuli from any site and at any cycle length induced tachycardia; (4) the atrial extrastimulus that blocks at the level of the AV node during tachycardia, which does not terminate the tachycardia, should not delay the subsequent V in the case of AVNRT. If in fact the atrial extrastimulus blocked antegrade in the bystander accessory pathway and conducted down the antegrade limb of AVNRT in a decremental manner, there would be maintenance of a 1:1 AV association of block at the level of the His. Certainly the atrial extrastimulus could block in a lower common pathway, which could not be excluded here.

Tachycardia using an atriofascicular or nodoventricular pathway was excluded because the first QRS after the blocked atrial extrastimulus was narrow as a result of antegrade conduction down the His-Purkinje system, which would have rendered the retrograde limb of the reentry circuit refractory, terminating the tachycardia. In the case of an atriofascicular pathway, an early coupled atrial extrastimulus would result in a delay in the subsequent ventricular activation and a fixed ventriculoatrial (VA) and His-atrial time.[7]

Atrial extrastimulus testing was diagnostic in this case. Premature atrial extrastimuli with AV block and continuation of the tachycardia without constant VA association on resumption of the tachycardia is diagnostic of AT. Therefore, AT with ventricular pre-excitation over a bystander left lateral accessory pathway was diagnosed. Atrial extrastimuli have been used to elucidate the mechanism of a wide complex tachycardia in the case of ART. Advancement of the subsequent QRS and atrial activation after introduction of an atrial extrastimulus timed to atrial septal activation is diagnostic of ART.[8] Differential atrial overdrive pacing during tachycardia can also be used for differentiation of a septal AT from AVNRT by calculating the delta VA.[9]

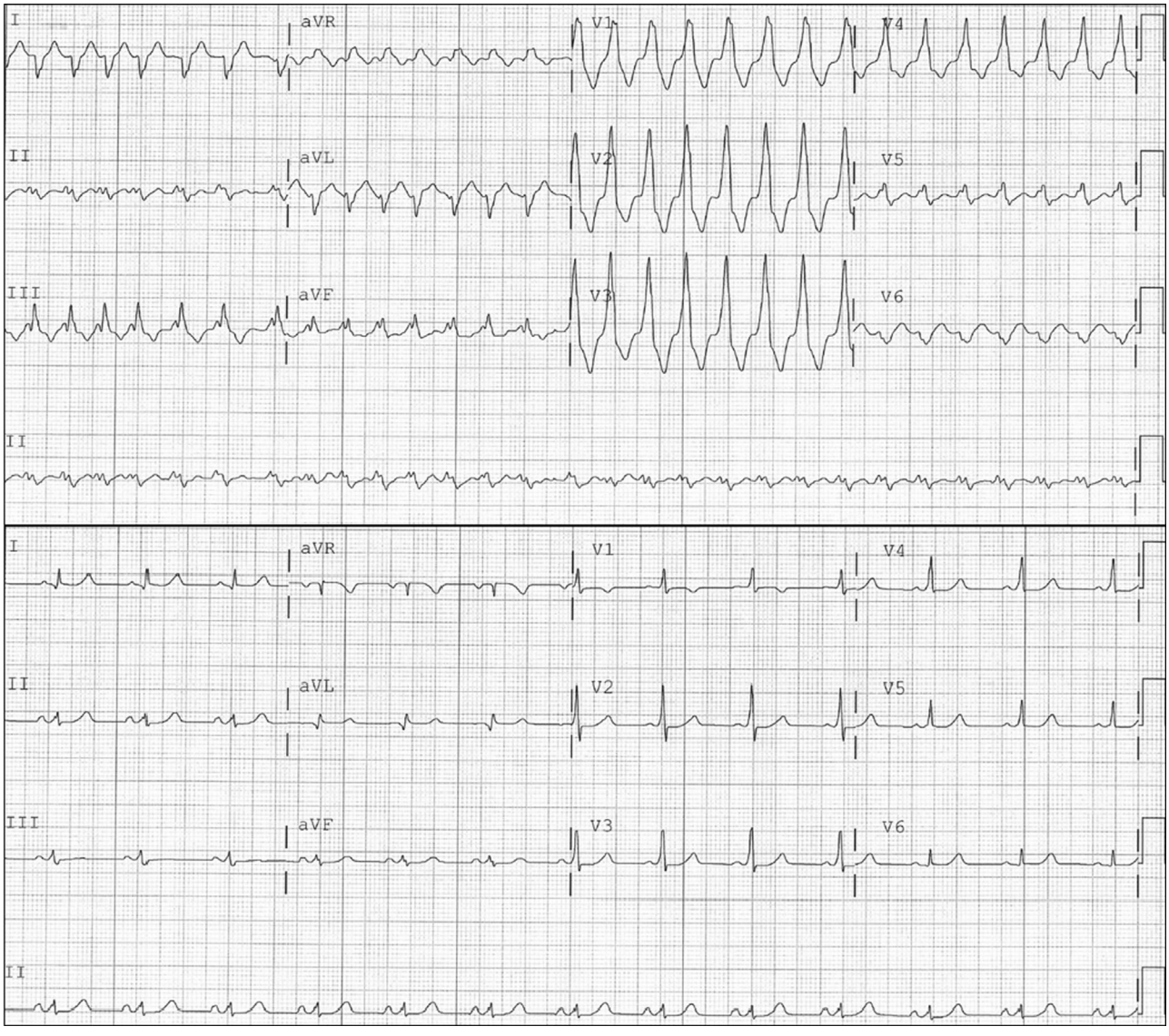

Figure 1 Surface electrocardiograms of the wide complex tachycardia (*top*) and in normal sinus rhythm (*bottom*).

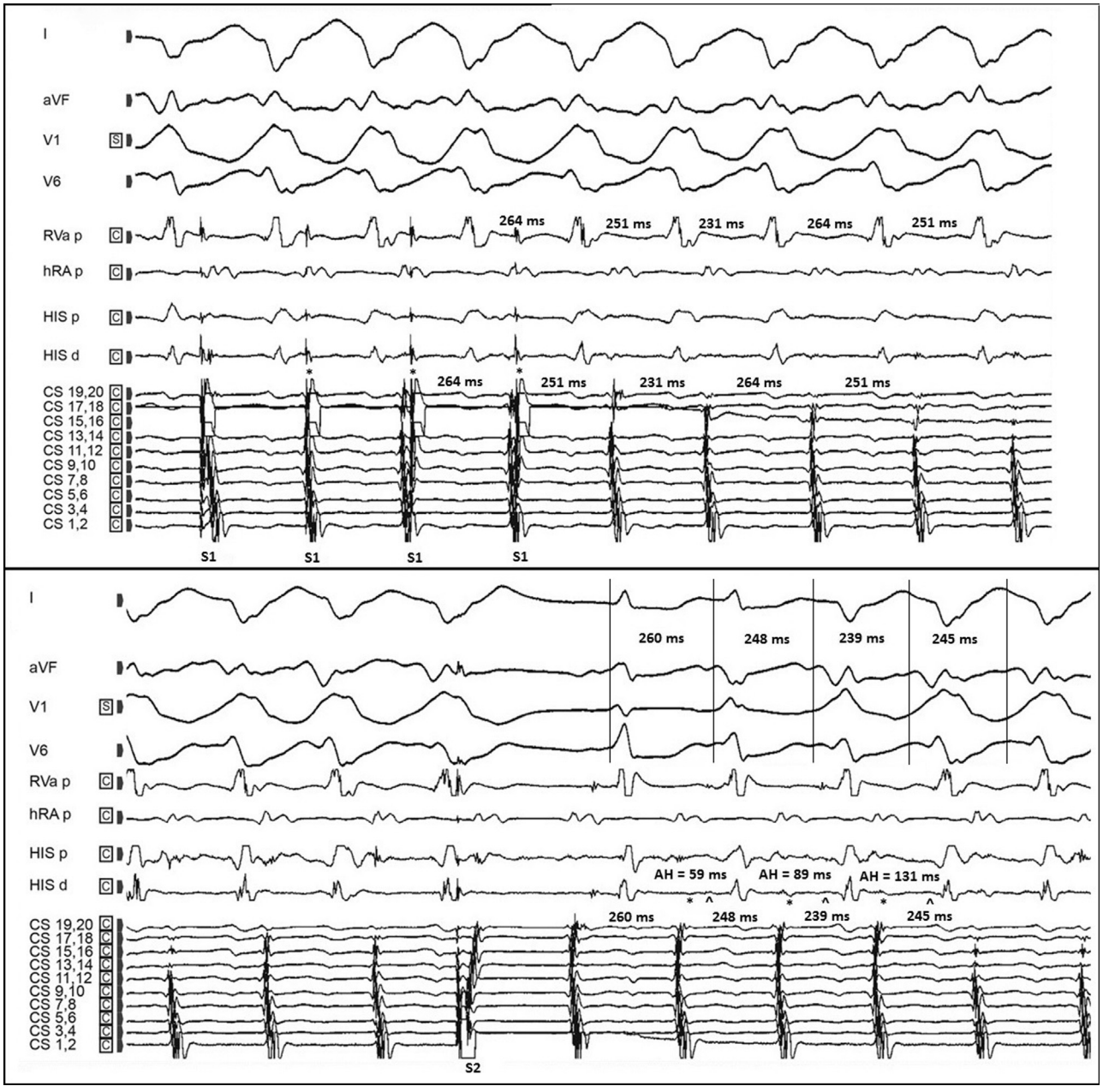

Figure 2 Induction of a wide complex tachycardia with atrial burst pacing (S1) (*top*). Changes in the VV interval follow changes in the AA interval. The last 3 pacing stimuli (*) did not capture the atrium since the local electrogram precedes the pacing artifact. Introduction of an atrial extrastimulus (S2) blocks in the left lateral accessory pathway and atrioventricular node without interrupting the tachycardia and is diagnostic of atrial tachycardia with bystander accessory pathway (*bottom*) (see text for details). Of note, the RVa catheter is in a septal location rather than apical because of the right bundle potential observed. (*) atrial electrogram and (^) His electrogram on His catheter.

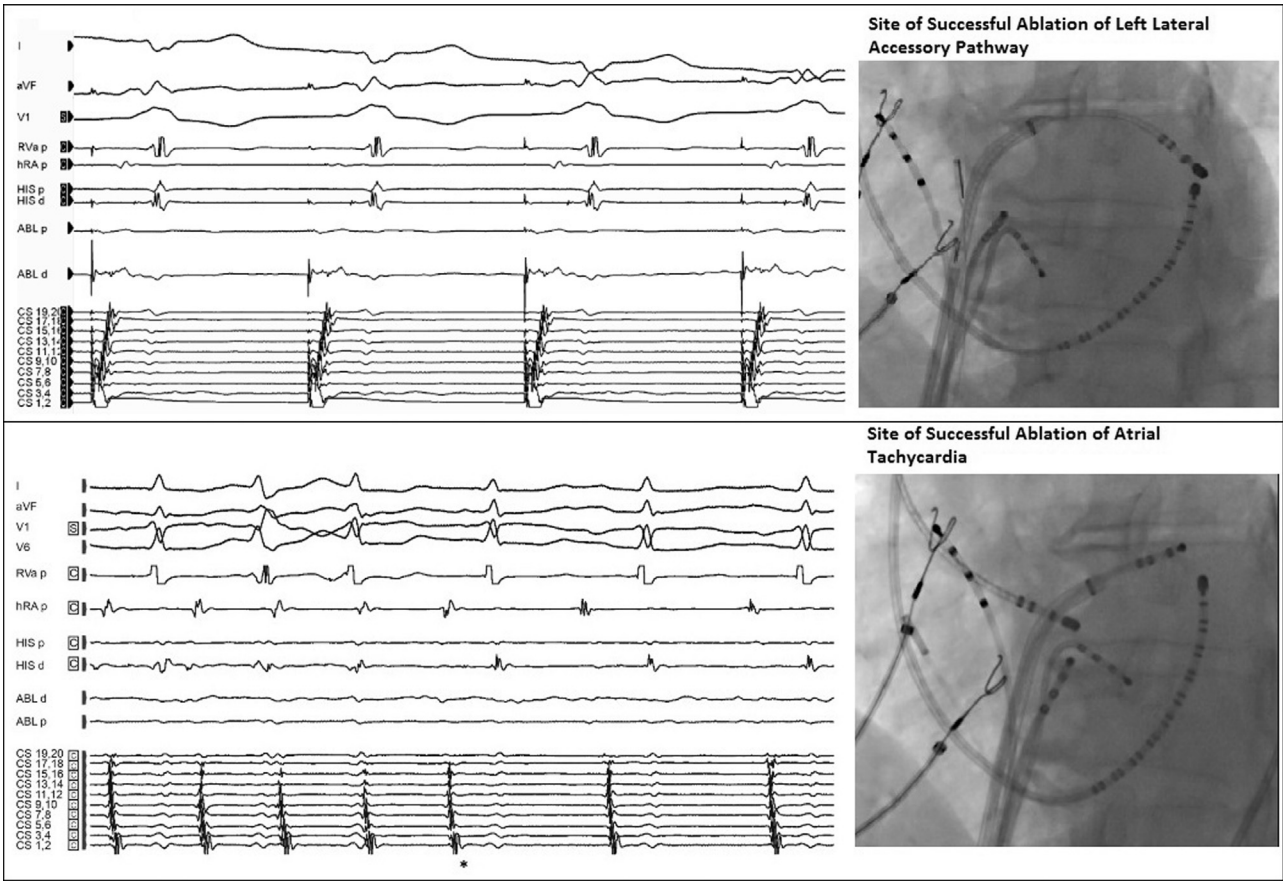

Figure 3 Site of successful ablation of the left lateral accessory pathway (*top*). The *arrows* denote the accessory pathway potential. The atrial tachycardia was successfully ablated using a retrograde aortic approach, mapping the earliest local atrial electrogram (35 ms earlier than the surface P wave) in the non-coronary cusp of the aortic root (*bottom*). Termination occurred 5 seconds after the start of the ablation lesion. The last beat of atrial tachycardia is denoted by the asterisks.

Conclusion

Introduction of single atrial extrastimulus facilitates precise diagnosis and therapeutic choice in wide QRS tachycardia with 1:1 AV association. AT with ventricular pre-excitation is an uncommon presentation but must be considered in the differential diagnosis of wide complex tachycardias.

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