Wide complex tachycardia in a patient with pre-excitation: What is the mechanism?

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Introduction

Patients with Wolff-Parkinson-White syndrome are generally evaluated for symptomatic supraventricular tachycardia (SVT). Not infrequently, these patients are also at risk of wide complex tachycardia. Although overt pre-excitation in sinus rhythm is most often present in patients with Wolff-Parkinson-White syndrome, the absence of a delta wave on the resting electrocardiogram (ECG) does not preclude antegrade conduction over the accessory pathway. The deductive analysis required in the interpretation of wide complex tachycardia, and its termination by adenosine, in a patient with intermittent pre-excitation is reviewed.

Case report

A 29-year-old woman was evaluated for a 2-year history of intermittent palpitations. She had previously presented to the emergency room with narrow complex SVT, which was terminated by intravenous adenosine. Her resting 12-lead ECG showed intermittent pre-excitation, consistent with a left free wall accessory pathway. Echocardiography demonstrated a structurally normal heart. In the electrophysiology laboratory, during introduction of the venous sheaths and catheters, the patient had spontaneous sustained wide complex tachycardia. The patient was clinically stable; the catheters were inserted during tachycardia, and programmed atrial and ventricular stimulation was performed.

Discussion

Figure 1 shows the spontaneous tachycardia observed in the electrophysiology laboratory. What are the main findings, and what is the differential diagnosis?

The resting ECG prior to the tachycardia did not show pre-excitation (Supplemental Figure, available online). The differential diagnosis of the wide complex tachycardia includes antidromic tachycardia, ventricular tachycardia (VT), SVT with aberrancy or with bystander conduction over an accessory pathway, or an atrial tachyarrhythmia with antegrade conduction over the accessory pathway. As previously reported by Brugada and colleagues,[1] in the presence of antegrade conduction over an accessory pathway, the 12-lead ECG has typical features of VT, including positive precordial concordance, and the atypical right bundle branch block morphology. The wide complex tachycardia in Figure 1 is consistent with a possible pre-excited tachycardia, showing a right bundle branch block morphology and a normal axis. There is positive concordance of the precordial leads and slurring of the QRS complex.

Thus, a normal ECG without pre-excitation does not exclude the possibility of rapid antegrade conduction over an accessory pathway. The absence of pre-excitation could be due to the distant location of the left lateral accessory pathway in relation to sinus node depolarization and normal AV conduction, or to different properties of conduction and refractoriness between the AV node and the accessory pathway. Catheters were advanced under fluoroscopic guidance to the high right atrium (HRA), the His bundle, the coronary sinus, and the right ventricular apex. The tachycardia cycle length was 380 ms.

What maneuvers should then be performed to accurately diagnose the mechanism of the tachycardia? Should we introduce atrial or ventricular extrastimuli? How could introduction of an atrial premature beat (APB) be useful during wide complex tachycardia? We introduced 1, then 2, late APBs from the HRA during wide complex tachycardia at 360 ms synchronized to the HRA (Figure 2A) without terminating the tachycardia. The first APB did not capture the tachycardia circuit, but the second APB captured and reset a single local atrial electrogram ("A") and a single local ventricular electrogram ("V") for 1 cardiac cycle. In both antidromic atrioventricular reciprocating tachycardia and a pathway-to-pathway tachycardia, an APB, which advances the next "V," would also be expected to advance the next "A" after the advanced "V." This does not appear to happen in this case, and evidence is therefore in favor of atrial tachycardia (AT); a decremental retrograde limb cannot be completely excluded, however. There was also fusion of

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KEY TEACHING POINTS

- Wide complex tachycardia in patients with pre-excitation involves several potential mechanisms, requiring a systematic approach during electrophysiological testing.
- Although a "VAAV" response during tachycardia confirmed the mechanism of tachycardia, the response to introduction of late atrial premature beats during tachycardia essentially excluded re-entry over the accessory pathway.
- Adenosine not only terminates AV re-entry tachycardia by blocking conduction over the AV node or a decremental pathway, but may terminate atrial tachycardia, even with doses as small as 3 mg, as shown in this patient.

the QRS morphology on the surface ECG following the second APB. The response to this maneuver excludes antidromic atrioventricular reciprocating tachycardia (ie, antegrade limb via left accessory pathway and retrograde limb via the AV node), as the APB could not advance and fuse the ventricular beat without terminating the tachycardia. AV node re-entry tachycardia is very unlikely, as it is not possible for a single APB to be able to advance and fuse with the ventricular beat without terminating the wide complex tachycardia, as both the antegrade and retrograde limb of the re-entry circuit of AV node re-entry tachycardia is in the vicinity of the AV node, which is a considerable distance from the pacing site (HRA). VT is very unlikely, both for clinical reasons (young woman with a normal 2D echocardiogram) and electrophysiologically, as we would not be able to advance a single ventricular beat during VT with an APB.

The most likely diagnosis is AT with conduction over a bystander left-sided accessory pathway, or a pathway-to-pathway re-entry tachycardia, as possible mechanisms for this wide complex tachycardia. In both AT and pathway-to-pathway tachycardia, the AV node is not a critical part of the tachycardia; hence we would be able to advance and cause fusion of the ventricular complex with an APB, likely related to sufficient proximity or conduction from the pacing site to the location of the accessory pathway.

Would there be any value in performing ventricular pacing? What would be expected to occur to the tachycardia during ventricular pacing at a rate faster than the tachycardia cycle length? During pacing of the right ventricular apex at 350 msec (Figure 2B), there was local capture of the ventricle and reset of the tachycardia of the last pacing beat. Following overdrive pacing the tachycardia continued, initially with an "A" followed by a "V," demonstrating a "VAAV" response.[2] This confirmed the mechanism of the wide complex tachycardia as AT with a left-sided bystander accessory pathway.

What would be the next maneuver that could provide useful information about the mechanism of tachycardia? If the patient has an AT, adenosine could result in the occurrence of AV block with continued tachycardia, or conversely the pre-excited tachycardia could terminate, which occurred following the administration of 3 mg of intravenous adenosine (Figure 3). This is consistent with adenosine-sensitive AT, which is far more likely than its potential similar effects on the retrograde limb of an accessory pathway. The local electrograms do not suggest that the AT originates from the

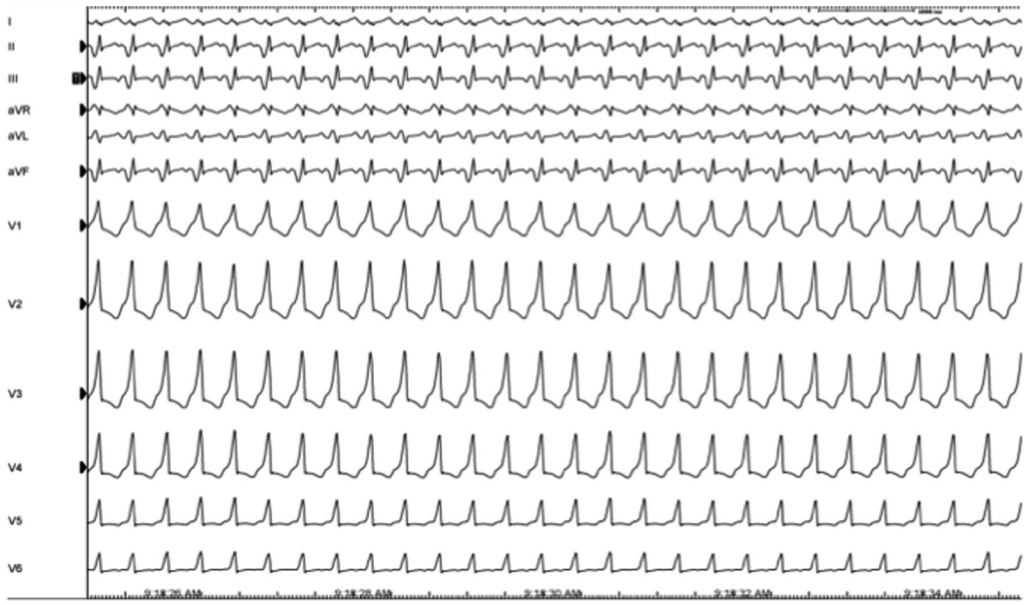

Figure 1 Electrocardiogram recorded during spontaneous tachycardia in the electrophysiology laboratory. See text for details.