A 21-year-old woman presented to the emergency room with palpitations and lightheadedness. Her ECG at presentation showed broad complex tachycardia at 235 beats per minute with right bundle branch morphology, inferior rightward axis, and positive concordance in the precordial leads (Figure 1A). The tachycardia was terminated with electric cardioversion after adenosine had no effect. The subsequent ECG demonstrated sinus rhythm, PR interval 100 ms, a positive delta wave in leads V1, II, III, and aVF, isoelectric in lead I and negative in aVR (Figure 1B). Subsequent echocardiography demonstrated a structurally normal heart. She proceeded to an electrophysiological study.

Editor’s Perspective see p 502

The baseline AH interval was 57 ms and HV was 13 ms. Atrial activation during ventricular pacing featured simultaneous activation of the lateral coronary sinus (CS) and septal (His) electrodes (not pictured). Programmed atrial extrastimuli reproducibly induced broad QRS complex tachycardia (Figure 1A). Attempts to entrain the tachycardia from the atrium induced atrial fibrillation that spontaneously terminated. Attempts to entrain tachycardia from the ventricle terminated tachycardia. Therefore, scanning sensed atrial extrastimuli during tachycardia were delivered from 2 atrial sites: septal right atrium and distal CS. Extra-stimuli that terminated tachycardia from both sites are displayed in Figure 2.

In Figure 2A, an extrastimulus was delivered from the septal right atrium; in Figure 2B, an extrastimulus was delivered from the distal CS. What is the tachycardia mechanism?

Discussion

Broad complex tachycardia in this patient with pre-excitation carries a differential diagnosis of supraventricular tachycardia (SVT) with aberrancy, SVT with bystander pre-excitation, antidromic atrioventricular re-entrant tachycardia or VT.

SVT with aberrancy is most unlikely as the QRS morphology would be unusual for right bundle branch block. A-V node dependant SVT (such as AVNRT) with bystander pre-excitation would be less likely as that tachycardia circuit is close to the His-Purkinje system and therefore a greater contribution of ventricular depolarization from the conduction system (ie, less pre-excited morphology) would be expected. The atrial activation pattern does not support the presence of an atrial tachycardia originating near the lateral accessory pathway and in any case this would not likely terminate repeatedly with ventricular pacing.

Figure 2A shows broad QRS (right bundle branch block) tachycardia, 1:1 VA relationship, with concentric atrial activation (septal right atrium being on time with the His A). The HV is negative thus ruling out bundle branch aberrancy. An extrastimulus from the septal right atrium terminated tachycardia while advancing the septal atrial (proximal CS) activation. The premature atrial extrastimulus (PAC) terminated tachycardia without conducting to the ventricle, thus ruling out ventricular tachycardia. This leaves antidromic atrioventricular re-entrant tachycardia as the most likely diagnosis; however, SVT with bystander pre-excitation has not be conclusively excluded though this is a fairly rare diagnosis.

The orthodromic wavefront generated by the PAC found the accessory pathway refractory and the antidromic wavefront generated by this PAC collided with the tachycardia wavefront before septal atrial activation and after ventricular activation. This maneuver indicates the presence of an accessory pathway but not its participation in the tachycardia. Potentially an AVNRT with bystander accessory pathway conduction could be terminated in this fashion.

On the contrary, Figure 2B shows an atrial extrastimulus with the same prematurity from the distal CS (near the accessory pathway) terminating tachycardia without advancing septal atrial activation. On this occasion, the antidromic wavefront from the PAC collides with the tachycardia wavefront above septal atrial activation. Hence, termination is in the anterograde limb only (namely the accessory pathway) when the septal atrium is refractory. This finding conclusively demonstrates that the left lateral accessory pathway participates in the tachycardia circuit. Because the tachycardia terminated...
by conduction block in the pathway, the pathway is an essential component of the tachycardia circuit and thereby rules out AVNRT with bystander accessory pathway conduction. This maneuver also rules out other forms of AV nodal-dependent tachycardia with bystander pre-excitation (such as nodoventricular or nodofascicular pathway-mediated tachycardia) because these tachycardias would also require a PAC to penetrate the septal atrium for termination. Therefore, pacing at this second site is diagnostic, independent of other maneuvers.

Subsequently, a PAC that again did not alter septal atrial activation, pre-excited the next ventricular activation and also advanced the subsequent atrial activation; proving presence and participation of an accessory pathway. This accessory pathway was successfully ablated with the first application of radiofrequency energy at the lateral mitral annulus, resulting in the loss of pre-excitation and no further inducible tachycardia.

The role of atrial extrastimuli in the diagnosis of antidromic atrioventricular re-entrant tachycardia has been previously described. Single beat termination is regarded as highly suggestive of macro-re-entrant tachycardia. In this case, single beat termination is observed at 2 different sites within the tachycardia circuit with electrograms able to discern 2 different sites of block (ie, the septum and lateral LA). This case highlights the use of multiple sites

Figure 1. Surface ECGs of (A) presenting tachycardia and of (B) sinus rhythm after cardioversion.

Figure 2. Intracardiac electrograms of single atrial premature beats terminating broad complex tachycardia. A, Atrial premature beat delivered at the septal right atrium 190 ms after sensed beat. Tachycardia is terminated without subsequent conduction to the ventricle; however, atrial activation is advanced. B, Atrial premature beat delivered at the lateral coronary sinus (CS) 190 ms after sensed beat. Tachycardia also terminates without conduction to the ventricle; however, the timing of septal atrial activation is unchanged.
of pacing to terminate tachycardia. A PAC delivered close to the site of the accessory pathway succeeded in terminating the tachycardia by blocking exclusively in the pathway. This proximity effect conveys a measure of electrophysiological distance of the pacing sites from the macro-reentrant circuit—in doing so, helping define its location. This technique could be adopted in instances when entrainment maneuvers cannot be applied.

Disclosures

None.

References


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