Not so innocent bystander(s)

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A patient with atrial flutter, intermittent non-sustained wide complex tachycardia and 1:1 AV conduction is reported. Electrophysiology testing showed counterclockwise isthmus-dependent right atrial flutter with conduction via the AV node and an innocent bystander left lateral accessory pathway. This explained the observed intermittent wide complex tachycardia. After successful bidirectional cavotricuspid isthmus conduction block, a sustained wide complex tachycardia with identical counterclockwise right atrial activation and rate occurred. This was due to antidromic AV re-entrant tachycardia with innocent bystander activation of the right atrium mimicking atrial flutter. Accessory pathway ablation effectively stopped tachycardia.

Case report

A 67-year-old male with history of palpitations had highly symptomatic, paroxysmal tachycardia consistent with counterclockwise atrial flutter (AFL) with and without an associated narrow and wide QRS complex morphology and with 1:1 or variable conduction (Figure 1A). In sinus rhythm, the electrocardiogram showed no pre-excitation (QRS duration = 110 ms).

Ventricular pre-excitation was first noted with fixed right atrial pacing at 490 ms. Delivery of decremental atrial extrastimuli then showed progressive pre-excitation consistent with a left lateral accessory pathway (AP). Antegrade AP effective refractory period was <270 ms. Decremental ventricular extrastimuli resulted in progressive retrograde VA delay until 230 ms when ventricular effective refractory period was attained. There was no retrograde AP conduction. No tachycardia was initiated with single atrial or ventricular premature extrastimuli.

Rapid atrial pacing induced typical counterclockwise AFL with a cycle length (CL) of ~290 ms (Figure 1B). Atrial flutter CL during the study, however, varied and ranged from 270 to 310 ms. Variable and episodic 1:1 AV conduction occurred with a narrow and wide QRS consistent with intermittent bystander AP conduction (Figure 2A). Cavotricuspid isthmus ablation resulted in apparent bidirectional block and initial termination of AFL.

Despite apparent bidirectional block, reduction in isthmus voltage, and double potentials in the isthmus, pre-excited 1:1 tachycardia with identical right atrial activation (and CL) as during AFL occurred spontaneously and was easily induced (Figure 2B). Isthmus conduction was assumed present; yet, despite repeated attempts at ablation, tachycardia with 1:1 activation remained inducible.

However, ventricular activation noted in the coronary sinus catheter had changed (Figure 2B) suggesting, possibly, a second tachycardia with the same CL in which the AP was no longer an innocent bystander but the right atrium was. This wide QRS tachycardia (CL 290 ms), without His activation before QRS, was induced easily with atrial extrastimuli, but not with ventricular pacing.

The shortest AV interval during tachycardia occurred at the distal coronary sinus. This and the absence of antegrade His activation during tachycardia were consistent with left anterolateral AP conduction. During tachycardia, retrograde activation was midline with ventricular pacing and retrograde extrastimuli consistent with retrograde AV nodal conduction (Figure 3A).

A single left atrial premature delivered from a distal coronary sinus site during His refractoriness reset the tachycardia CL for the following beat, pre-excited the ventricle, shortened the VH interval, and prolonged the HA interval, indicating active involvement of AP in the circuit and consistent with antidromic AV re-entrant tachycardia (AVRT) (Figure 3B).

Radiofrequency was delivered to a left lateral site on the mitral ring where AV interval was the shortest, local ventricular activation preceded the pre-excited QRS, and an AP potential was present. This eliminated pre-excitation and tachycardia induction. No tachycardia was evident during follow-up.

Discussion

This case demonstrated a shift in innocent bystander activation from a left lateral AP during typical counterclockwise right AFL to utilization of the AP as part of antidromic AVRT after cavotricuspid isthmus ablation. The latter scenario resulted in innocent bystander right atrial activation mimicking typical AFL. Atrial flutter and antidromic tachycardia CLs were almost the same. Although there was apparent bidirectional isthmus block, it was assumed initially that conduction persisted. The presence of inducible pre-excited tachycardia, now with only 1:1 AV relationship, despite a similar CL and right atrial activation prompted search for a second tachycardia.

The earliest AV interval in the distal coronary sinus along with evidence for 1:1 retrograde AV nodal conduction during tachycardia suggested active involvement of AP in the tachycardia circuit. A left atrial premature delivered from a distal coronary sinus site resulted
in ventricular pre-excitation and resetting of the tachycardia for one beat (CL from 287 to 255 ms), despite lengthening of the HA interval. Shortening of VH interval appeared to precede shortening of the tachycardia CL. This supported the diagnosis of antidromic AVRT and excluded AFL with bystander AP activation.

Perhaps, cavotricuspid isthmus ablation, by creating a line of block, helped facilitate sustained antidromic AVRT. When isthmus activation was present, it may have been possible for AFL to cause concealed depolarization of the AV node to limit retrograde conduction and make a macro-re-entrant AVRT less likely to sustain. Isthmus ablation may have eliminated tonic concealed depolarization of the AV node leaving retrograde AV node activation now unopposed. This hypothesis, to our knowledge, has not been reported before. Thus, the AP and the right atrium were contributors to different tachycardias and were also innocent bystanders.

Conflict of interest: none declared.
Figure 2  (A) Atrial flutter with variable and 1:1 AV conduction. The second and fifth QRS complexes are pre-excited (short arrows) with evidence of antegrade conduction down a left lateral AP with earliest AV interval noted in CS 1,2. The first, third, and fourth QRS complexes show antegrade AV nodal conduction. Third and fourth QRS complexes demonstrate rate-related left bundle branch abnormality (asterisk). Long arrows demonstrate differences in the ventricular activation pattern as AV conduction switches from AV node to AP and back. Note variation in atrial flutter CL which ranges from 270 to 285 ms. Shown are recordings from surface leads I, aVF, V1, and V6, and intracardiac recordings from the distal His Bundle (His d) and coronary sinus (CS 9,10–CS 1,2) catheters. We were unable to obtain a consistent His deflection at the time of this recording. (B) Demonstration of wide complex tachycardia (CL 290 ms) after cavo-tricuspid isthmus ablation and attainment of bidirectional block. Earliest ventricular activation (arrow) is noted in CS 1,2, suggesting antegrade conduction through a left lateral accessory pathway. Shown are recordings from surface leads and intracardiac recordings from the Halo (H10–H1), ablation (ABL, ABL d), and coronary sinus (CS 9,10–CS 1,2) catheters.
Figure 3  (A) Antidromic AV re-entrant tachycardia. Earliest ventricular activation (bottom arrow) is noted in CS 1. Retrograde AV node conduction is demonstrated by the His bundle catheter showing a ventricular spike (V) followed by the His (H) and atrial (A) deflections. Shown are recordings from surface leads and intracardiac recordings from the high right atrium (HRA), his bundle (His d), coronary sinus (CS 9–CS 1), and right ventricular apex (RVa). (B) Resetting of the wide complex tachycardia circuit by a His-refractory premature left atrial beat introduced with a coupling interval of 220 ms (arrow). Tachycardia CL for the beat following the premature shortens from 287 to 255 ms. Ventricular CL as shown is measured at the right ventricular apex. Note an apparent shortening of the ventricular (V)–His (H) interval as well as retrograde delay in the AV node as evidenced by lengthening of the His (H)–atrial (A) interval in the His bundle electrogram. Shown are recordings from surface leads and intracardiac recordings from the high right atrium (HRA), His bundle (His d), coronary sinus (CS 9–CS 1), and right ventricular apex (RVa).
References
