Interesting changeover from short VA to long VA tachycardia with single atrial premature depolarisation: What is the mechanism?

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TITLE PAGE

Title: Interesting changeover from short VA to long VA tachycardia with single atrial premature depolarisation: What is the mechanism?

Short title: Concomitant AVNRT and atrial tachycardia: Killing two birds with one stone.

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Case:

A 35 year-old-lady underwent EP study for recurrent narrow QRS tachycardia (Fig 1A) terminating with adenosine. Baseline ECG showed no pre-excitation (Fig 1B). EP study was performed with decapolar catheter in coronary sinus (CS), quadripolar catheters placed in HRA (high right atrium) and His; and one roving catheter in right ventricle (RV). Baseline intervals: AH interval=76 ms, HV interval 34 ms. During antegrade study AH jump, AV node duality and intermittent rate related aberrancy (RRA) with unusual axis (RBBB with right axis deviation, HV 34 ms) was noted. Retrograde study -VA conduction was concentric and decremental (VAERP=270 ms), no VA jump. No sustained tachycardia could be induced by standard protocols at baseline. On isoprenaline, a short VA tachycardia (SVT1) with near simultaneous A and V activation [HV=34 ms, tachycardia cycle length (TCL)= 305 ms, Fig 1C) was induced with atrial premature depolarisation (APD) and ventricular premature depolarisation (VPD) with similar RRA (Fig 1C and 1D). Maneuvers confirmed SVT1 to be slow-fast atrioventricular nodal reentrant tachycardia (AVNRT) (VAV response, SA-VA=160 ms, cPPI-TCL=151 ms). During programmed decremental APDs from CS to differentiate it from junctional tachycardia (JT), faster SVT2 was induced with a longer VA (Fig 2A and 2B). SVT2 had TCL of 260 ms after initial wobble, septal VA = 140 ms. What could be the mechanism of SVT2 ?

Commentary:

The possibilities were:

1. SVT2 could be atypical AVNRT (slow-slow or fast-slow)
2. SVT2 could be atrial tachycardia (AT) or orthodromic atrioventricular reentrant tachycardia (AVRT).

Maneuvers were performed to confirm the diagnosis. This SVT2 with 1:1 VA could not be entrained by RV pacing, during which the atrium got dissociated essentially ruling out AVRT. Attempt to demonstrate VA linking was uninterpretable, as HRA pacing at 20 ms shorter cycle length (CL) repeatedly terminated the tachycardia. At times, post-VOP (ventricular overdrive pacing) the A:V ratio became 2:1 (supra-hisian), with same atrial CL of 255-260ms (Fig 3A) likely due to concealed conduction into the AV node. All these favored AT, although both AVNRT and AT could exhibit 2:1 conduction. To differentiate between an AT and a difficult-to-entrain AVNRT, injection adenosine (12 mg) was used with an intention to demonstrate AV block. However, it terminated the tachycardia repeatedly (Appendix 1), hence it could not be convincingly validated as a subset of AT are also known to be adenosine sensitive. Finally, few crucial observations were recorded during the transition from SVT1 to SVT2. The TCL during SVT2 became stable (TCL= 260ms) after initial wobble (Fig 2B). The AA intervals were noted to have the first change amongst all the intervals (CS-AAHRA-AA HH/VV) during the wobble (Fig 2B and 3B). This further confirmed the diagnosis of AT.

An intriguing question would be when did the AT start and AVNRT terminate during the changeover? Whether it occurred *immediately after the APD or did the AVNRT continue for few more beats?* The initial wobble again rendered important insights to these (Fig 3B). It is noted that when the APD was delivered @ 230 ms coupling interval, the immediate H (H3) was not perturbed as it was committed to previous slow pathway (SP) conduction. Also, the local A-EGM (A3) in proximal His was not altered. In fact, the proximal His-A (A3) occurred after His EGM (H3) and thus could enter the fast pathway. This also explains the fact that even the next H4 was not pulled. Hence, we speculate that A4 taking place before H4 entered the fast pathway (FP) and terminated AVNRT at this beat. The next AT beat (A5) conducted antegradely by FP pathway to the ventricle and thenceforward the RR interval equated to the AA/HH interval of 260 ms. This initial AA wobble is often a feature of AT.

Another unique finding of this case was reproducible induction of AT directly from AVNRT with APD from CS, but not otherwise. This probably happened as the FP-ERP was reached earlier and the conduction over SP induced typical slow-fast AVNRT. The coupling interval (APD@ 230 ms, Fig 2) required to induce her AT (likely micro-reentrant) could not be reached as AVNRT was reproducibly induced at much longer

coupling interval APDs. Interestingly, ventricular entrainment of SVT2 from RV apex was difficult despite good VA conduction (upto 270 ms) in sinus rhythm and easily entrainable SVT1. We hypothesize that ERP of FP was reached during faster PCL @ 240 ms. Moreover, as the FP was already conducting antegradely during AT, it was possibly not available for retrograde VA conduction.

For AT ablation, the earliest A was mapped and found to be near CS ostium/SP region. Although this AT could have been ablated during tachycardia, we had an apprehension of catheter drift at termination of tachycardia which can lead to AV nodal injury. Hence, it was decided to perform slow pathway modification in sinus rhythm (SR) which might cure both, followed by attempt of AT induction. RF energy (40 watts, 60-degree, non-irrigation tip medium curve catheter) was delivered in SR which promptly resulted in accelerated junctional beats with intact VA conduction. After 90 sec lesion, reinduction was attempted. No tachycardia could be induced with and without isoprenaline after this.

There are reported associations of AVNRT with other tachycardia substrates. In a retrospective study, Schernthaner et al reported focal AT in 8% patients in a cohort of 493 patients after ablation for AVNRT [1]. An earlier study had reported 15% subjects had inducible AT recorded during AVNRT ablation. Majority of them were however isoprenaline dependent. Moreover, only a minority (7%) with inducible AT, actually developed clinical AT on follow up after only SP modification [2]. When AT is associated with AVNRT, the site of origin can be at the CS ostium. P wave morphology and earliest EGM can localize the same [3]. The exact mechanism of the coexistence of AVNRT with AT especially originating from Koch triangle remains indeterminable, although an interaction between AV nodal tissue and perinodal tissue has been postulated [4]. There is only one similar report of AT and AVNRT ablated successfully with SP modification [5].

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Figure legends:

Fig 1 :

A: Clinical narrow QRS tachycardia (SVT1).

B: Baseline EGG showing absence of pre-excitation.

C: Intermittent wide QRS morphology with RBBB and right axis deviation. TCL and HV interval was same as SVT 1.

D: 12 lead ECG morphology of wide QRS tachycardia. Maneuvers proved it as AVNRT with aberrancy.

Fig 2

A: Critically timed APD reproducibly initiated SVT2 (longer VA, shorter TCL) from SVT1

B: Continued SVT2 with longer VA.

Fig 3

A: A:V ratio of 2:1 during SVT2 after a an attempt of ventricular entrainment (atrial CL- 255-260 ms)

B: Annotated Fig 2A showing that the APD did not engage the slow pathway as it was refractory. The H3 and next RR was unperturbed. There was a retrograde conduction *locally (A3)* via FP which could not produce any evident A-EGM in CS as it was prematurely captured by the APD. The same produced another beat of AVNRT (H4) beat via antegrade SP conduction. Hence the timing of next H (H4) was unperturbed. The second AT beat (A4) could capture fast pathway and terminated the AVNRT after which AT completely took over. Over next few beats the wobble got stabilised.

Appendix 1: Adenosine terminating the tachycardia.