

Case Reports

Elucidation of the Mechanism of Supraventricular Tachycardia with QRS Alternans and Long RP Interval

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We refer to a patient with QRS alternans and RP/PR <1 ratio during a supraventricular tachycardia. Although the presence of these ECG characteristics suggest an atrioventricular reentrant tachycardia involving a retrogradely conducting accessory pathway the electrophysiological study followed by a successful ablation demonstrated an atrial origin of the tachycardia.

The diagnostic value of QRS alternans during a narrow QRS tachycardia as indicative of accessory pathway participation in tachycardia mechanism remains controversial. Green et al suggested that QRS alternans is indicative of orthodromic atrioventricular reciprocating tachycardia involving an accessory pathway with a specificity of 96% and positive predictive value of 92%¹. In contrast, other groups of investigators found that the incidence of QRS alternans was not significantly different among the various types of supraventricular tachycardias².

Case description

A 37-year old patient with recurrent episodes of palpitations despite the use of various antiarrhythmic agents (sotalol, flecainide, verapamil) was referred for evaluation to our centre. Clinical examination, resting ECG, echocardiogram and exercise test were unremarkable.

Quadripolar electrodes were placed in high right atrium, right ventricular apex. His electrogram position and within the coronary sinus. The basic intervals were measured within normal limits. Incremental atrial pacing and the introduction of programmed atrial extrastimuli revealed decremental conduction through

the atrioventricular (AV) node without evidence of preexcitation or dual physiology of AV node. A narrow complex tachycardia with QRS alternans and P wave separate from QRS (RP/PR <1) was easily induced during programmed atrial pacing (Figure 1). The earliest atrial activation during tachycardia was recorded in the HRA dipole with VA interval >90 msec. The tachycardia was terminated spontaneously with a QRS complex. What is the mechanism of the tachycardia?

Discussion

The differential diagnosis of a narrow complex tachycardia with long RP interval includes the following cases: 1) orthodromic atrioventricular reciprocating tachycardia (AVRT), 2) uncommon form (fast-slow) of AV nodal reentry tachycardia (AVNRT) and 3) atrial tachycardia (AT)³.

The observation that the earliest atrial activation is recorded on the electrode placed in the high right atrium, makes the diagnosis of uncommon AVNRT improbable. AVRT utilizing a right sided concealed pathway and AT remain on the list of possible diagnosis. The variable VA interval while HV remains fixed (Figure 2a) makes unlikely the presence of a concealed pathway although cannot be excluded the case of multiple concealed



Figure 1. Twelve-lead surface ECG (paper speed 50 mm/sec) 15 seconds after the induction of the supraventricular tachycardia with a cycle length of 290 msec. Marked QRS alternans is present in all twelve leads. Possible P waves buried in the ascending limb of T waves are present in inferior leads.

pathways or a pathway with decremental retrograde conduction. The inability however to reset the tachycardia with ventricular extrastimulus when His–bundle is refractory rules out the possibility of an accessory pathway (Figure 2b).

Atrial tachycardia remains thus the only potential diagnosis. This is also compatible with termina-

tion of tachycardia spontaneously with a QRS complex rather than an atrial beat. The diagnosis was further confirmed with tachycardia persistence, despite complete AV block induction after the administration of 12 mg adenosine (Figure 3). Termination of tachycardia a few seconds later suggested also a different sensitivity to adenosine of AV node in rela-



Figure 2. (a) A variable VA interval with fixed HV interval is recorded during the tachycardia. (b) A His-synchronous premature stimulus is delivered from the right ventricular apex during the tachycardia which does not reset (advance) the atrium. From top to bottom are leads I, II, aVF, V₁ and V₆, HRA px=high right atrium catheter recording, His px and His dx=bipolar recordings from proximal and distal His catheter, CS_{1,2} and CS_{3,4}=bipolar recordings from proximal and distal coronary sinus catheter, RV px=right ventricular apex catheter recording.



Figure 3. Response of the tachycardia to adenosine administration. Note persistence of the tachycardia despite the induction of high grade complete AV block. Slowing and finally termination of the atrial tachycardia is also observed.

tion to the atrial source of tachycardia. The progressive deceleration of the atrial rate during the same pharmacological trial disclosed a positive P wave in limb leads, slightly different from those in sinus rhythm (Figure 3). This was suggestive of high right atrial origin of the tachycardia.

Tachycardia mapping indicated the earliest atrial activation in the antero-lateral wall of the right atrium towards superior vena cava junction. Energy application at this site where the A electrogram was 13 msec before the onset of the surface P wave terminated the tachycardia.

The presence of QRS alternans during a narrow complex tachycardia has been reported as highly predictive of AVRT whereas other studies did not confirm this correlation. Green et al reported a 96% specificity of QRS alternans for AVRT while the incidence of QRS alternans among AT was only 4% to 12%.^{1,4} Kalbfleisch et al found that QRS alternans during tachycardia was an independent predictor of AVRT with a positive predictive value of 76%. In the same study the RP/PR ratio ≤ 1 was more common with AVRT (87%) than AT (11%) with a positive predictive value of 88% for AVRT.⁵

In our case, the diagnosis of the tachycardia proved to be atrial, despite the coexisting presence of QRS alternans and RP/PR ratio < 1 . This is in agreement with Morady's view according to which QRS alternans is a rate-related phenomenon, independent of the tachycardia mechanism.^{2,6} In rates exceeding 180 bpm, as in our case, the incidence of QRS alternans increases in all supraventricular

tachycardias. The relatively prolonged conduction time at the level of AV node (AH=115 msec) explains the extended PR interval.

Termination of tachycardia as the final response to adenosine administration in combination with tachycardia initiation by programmed stimulation indicate triggered activity as the mechanism of the arrhythmia.⁷ However, this has not been further investigated using additional pacing or pharmacological tests.

Our case illustrates that the mechanism of supraventricular tachycardia can not be determined accurately based only on surface ECG analysis and an EP study may be required in several cases.

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