Paroxysmal Supraventricular Tachycardia Caused by 1:2 Atrioventricular Conduction in the Presence Of Dual Atrioventricular Nodal Pathways

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Abstract: One-to-two atrioventricular conduction, ie, the double response to a single sinus or atrial impulse, resulting in two QRS complexes for one P wave, is a rare manifestation of dual atrioventricular (AV) nodal pathways. This report describes the case of a 61-year-old woman with continuous episodes of supraventricular tachycardia caused by independent conduction to the ventricles of sinus impulses over both the fast and the slow AV nodal pathway, giving rise to a ventricular rate that was twice the sinus rate. A wide spectrum of electrocardiographic manifestations of 1:2 AV conduction was observed on the surface electrocardiogram. The diagnosis was suggested by several elements including evidence of dual AV nodal pathways during sinus rhythm and cycle length alternans during tachycardia. The patient underwent successful slow pathway ablation with complete disappearance of symptoms and electrocardiographic manifestations of 1:2 AV conduction. Key words: catheter ablation, dual atrioventricular nodal pathways, electrocardiogram, supraventricular tachycardia.

Case Report

Clinical Presentation

A 61-year-old woman was referred because of drug-resistant tachycardia. The patient had been suffering several daily episodes of palpitations lasting from a few minutes to several hours, often associated with flushing and dizziness, occurring both at rest and during physical activity. Based on
resting electrocardiogram (ECG) and Holter monitoring several different supraventricular (atrial fibrillation and flutter, AV junctional extrasystoles and tachycardia) and ventricular arrhythmias (extrasystoles and nonsustained tachycardia) had been suspected. Since the age of 35, she had been receiving drug treatment with digoxin, quinidine, verapamil, amiodarone, propafenone, sotalol, or combinations of these, with either no improvement or only temporary improvement. Pertinent physical findings were limited to irregular pulse and moderate hypertension. The echocardiogram and coronary angiogram were normal.

**Holter Monitoring**

A 24-hour ambulatory recording showed an extremely variable heart rate, ranging from 45 to 200 beats/min. Normal sinus rhythm was limited to a few minutes of the recording. The most common rhythm abnormality consisted of paroxysmal episodes of irregular, narrow QRS complex tachycardia at ventricular rates ranging from 100 to 200 beats/min (Fig. 1a, b). Sustained RR cycle alternans was often observed during tachycardia (Fig. 1c). Atrial activity was often difficult to analyze (Fig. 1a) in such a way that the pattern resembled atrial fibrillation. On other occasions the P waves were clearly detectable (asterisks in Fig. 1b) and expressed an AV relationship that was either variable (Fig. 1b) or with a fixed 1:2 ratio (Fig. 1c). Sinus rhythm was frequently altered by narrow premature QRS complexes apparently not preceded by P waves (Fig. 2a). At times, some short rhythmic sequences occurred with normal rate, 1:1 AV relationship, and very prolonged PR intervals (about 0.60 s), in such a way that the P wave was almost simultaneous to, and partially obscured from, the preceding QRS complex, thereby simulating an AV junctional rhythm. In one occasion the termination of prolonged PR interval occurred in coincidence with a second-degree sinoatrial block, followed by sinus rhythm with normal PR interval (Fig. 2b). Isolated or repetitive wide QRS complexes were also observed (Fig. 2c). Finally, occasional episodes of second-degree AV block were detected (Fig. 2d).

All of these ECG patterns are explained by the presence of dual AV nodal pathways capable of single (Fig. 2a), repetitive (Fig. 1a, b), or sustained (Fig. 1c and related ladder diagram) 1:2 conduction,

![Fig. 1. Noncontinuous ECG strips recorded during Holter monitoring (lead CM 5), showing the patient's paroxysmal episodes of irregular, narrow QRS complex tachycardia (strips a and b). The asterisks below strip b indicate manifest sinus P waves. Sustained RR cycle alternans can be seen in strip c. In the ladder diagram solid lines in the AV section represent conduction over the fast pathway, and the broken lines represent conduction over the slow pathway.](image-url)
with occasional exclusive slow pathway conduction (Fig. 2b). Aberrant conduction (Fig. 2c), Wenckebach periodicity, and AV block (Fig. 2d) contributed to the variety of ECG manifestations.

**Electrophysiologic Study**

After informed written consent, an electrophysiologic study was performed, with the patient in a fasting, unmedicated state. Antiarrhythmic drugs had been discontinued for at least 5 half-lives, and the patient had not been taking amiodarone in the previous 2 years. Quadripolar 6F catheters inserted through the right femoral and left subclavian veins were placed in the right atrium, coronary sinus, His bundle region, and right ventricular apex. A steerable 7F catheter was used for mapping and radiofrequency delivery.

During uncomplicated sinus rhythm (not illustrated) the intracardiac ECG was normal, except for a slight prolongation of the HV interval to 70 ms. At a sinus cycle length of 660 ms the AH interval was 75 ms. An example of spontaneous tachycardia is shown in Figure 3. Both the surface and the intracardiac ECG indicated sinus rhythm with normal atrial activation and a stable cycle of 580 ms. There was a regular 1:2 AV relationship. Each ventriculogram was preceded by a His deflection with a constant HV interval. The interval between the atrial wave and the first His deflection was 115 ms, whereas the interval between the atrial wave and the second His deflection was slightly variable, ranging from 380 to 400 ms.

Figure 4 shows sustained exclusive anterograde conduction over the slow pathway: a 1:1 AV relationship is shown, with constant AH intervals of 380 ms. Figure 5 shows a premature atrial beat that resets the modality of AV conduction: a sequence of 1:1 conduction over the slow pathway was interrupted by an atrial extrasystole (arrow) that "switches" to normal exclusive conduction over the fast pathway. A standard study of AV nodal function with premature atrial extrastimuli could not be performed because of disturbing double responses.
Fig. 3. Simultaneous recording of four surface ECG leads (I, III, aVF, and V1) and intracardiac electrograms from the high right atrium, distal (HRAp) and proximal (HRAp), the His bundle region, proximal (HBEp) and distal (HBEp), the coronary sinus, proximal (CSp) and distal (CSd), and the right ventricular apex (RVAp).

Ventricular pacing resulted in complete retrograde VA block (not illustrated).

After obtaining intracardiac recordings, radiofrequency ablation of the slow pathway was performed. Two radiofrequency pulses (55°C, 60 s each) delivered in the right postero-septal region next to the coronary sinus os, in correspondence with a typical "slow pathway potential," resulted in block of conduction over the slow pathway. After ablation, neither spontaneous nor induced arrhythmias

Fig. 4. Simultaneous recording of four surface ECG leads (I, III, aVF, and V1) and intracardiac electrograms. Symbols as in Fig. 3.
could be observed during electrophysiologic study. Figure 6 compares the 24-hour heart rate profiles recorded before and after the procedure, in the absence of antiarrhythmic treatment, showing complete suppression of tachycardia.

Discussion

Simultaneous fast and slow pathway conduction to the ventricles over dual AV nodal pathways may occur spontaneously for single (13) or consecutive (5-12) atrial impulses, in the latter case resulting in a tachycardia with ventricular rate twice the atrial rate. Very few cases of such arrhythmia, often called “paroxysmal non-reentrant supraventricular tachycardia,” have been described (6-12). In our patient the Holter recording revealed a spectrum of ECG patterns that could be misinterpreted as AV junctional accelerated rhythm, atrial fibrillation, ventricular extrasystoles, or runs of ventricular tachycardia. The marked PR interval change (from less than 0.20 s to more than 0.40 s) during sinus rhythm was a key for the interpretation of this case.

AV Conduction and Dual AV Pathways

In the presence of dual AV nodal pathways, conduction of sinus impulses to the ventricles may occur in three different modalities: (1) over the fast pathway; (2) over the slow pathway; (3) simultaneously over both fast and slow pathways (1:2 response, illustrated in the ladder diagram of Fig. 1). The sinus impulse approaches simultaneously the proximal edge of both pathways, but in the majority of subjects conduction to the ventricles occurs through one single pathway, and 1:2 response does not occur because of (1) concealed retrograde conduction into the “nondominant” pathway (the slow pathway during anterograde fast pathway conduction and vice versa) (3,7), and (2) refractory state of the distal common pathway, occurring whenever its recovery time exceeds the difference between slow and fast pathway conduction time.

The determinants of conduction over dual AV nodal pathways include sinus rate changes (6,8,9,14), atrial or ventricular premature beats (7,8,13-15), and electrophysiologic properties of both the pathways and the distal conduction system (conduction velocity, refractoriness, retrograde conduction) (3,6,8,9). Autonomic nervous system (7,14) and drugs (2,6-10) can modify these properties and, consequently, the modality of AV conduction. In the present case, slow pathway conduction time ranged from 0.40 to 0.80 s, with longer intervals recorded during the night, suggesting a strong vagal influence on slow pathway conduction velocity.
One of the ECG manifestations of dual AV nodal pathways is “atypical” second-degree Wenckebach type AV block (14). In our patient second-degree AV block was frequent, usually occurring after one or more double responses (Fig. 2d). Completely blocked sinus impulses always follow a QRS complex resulting from conduction over the slow pathway; the block occurs whenever the ensuing sinus impulse, which is very “early” in the cardiac cycle, traverses the fast pathway but finds the final common pathway refractory and cannot reach the ventricles. On the other hand, if the same impulse also undergoes a block in the slow pathway, a total absence of conduction to the ventricles ensues. In this situation, AV block is not an expression of AV nodal impairment, but rather a manifestation of dual AV nodal pathways. It is worth noting that in our patient AV block was totally absent in two 24-hour recordings following slow pathway ablation.

In the presence of dual AV nodal pathway, persistence of exclusive slow pathway conduction is not, in itself, an expression of poor fast pathway conduction, but usually results from concealed retrograde conduction into the fast pathway; following a sinus impulse conducted over the slow pathway only, the fast pathway is invaded retrogradely, and becomes refractory to the ensuing supraventricular impulse, which is again conducted to the ventricles over the slow pathway, with retrograde concealed conduction into the fast pathway, and so on. Such a mechanism, however, was not operating in our patient, since it was impossible to assume retrograde invasion of the nondominant pathway, a phenomenon that would have prevented 1:2 conduction. It is more likely that exclusive anterograde conduction over the slow pathway was occurring, because the impulse conducted over the fast pathway was blocked in the final common pathway. This block was favored by the very long slow pathway conduction time; as a consequence, the common pathway was still refractory when it was reached by the ensuing sinus impulse conducted over the fast pathway.

Accordingly, the finding of exclusive slow path-
way conduction in the reported case did not cause any concern for potential poor fast pathway conduction; this is also proved by the postablation follow-up, demonstrating normal anterograde fast pathway conduction.

Incidence of Paroxysmal Tachycardia Caused by 1:2 Conduction

The prevalence of paroxysmal tachycardia resulting from double response to sinus impulses is presumably very low. We are aware of only seven reported cases (6-12). One-to-two AV conduction is probably rare even among patients with electrocardiographic evidence of dual AV nodal pathways. In a series of 10 such patients identified from analysis of 3,200 Holter recordings, 1:2 AV conduction was not mentioned even though the majority of patients had their longest PR interval ranging from 0.40 to 0.64 s (15). More recently, Fisch et al. (13) reported on 21 patients with ECG evidence of dual AV node physiology during sinus rhythm. Only 1 of these subjects showed episodic 1:2 conduction of single impulses. Although supraventricular tachycardia due to 1:2 AV conduction is rare, it is likely that its prevalence is underestimated because of the difficulties in differentiating this arrhythmia from other more common ones.

Diagnosis of Paroxysmal Tachycardia Caused by 1:2 Conduction

The possibility of dual AV nodal pathways with simultaneous fast and slow conduction should be suspected in the presence of irregular paroxysmal supraventricular tachycardia associated with marked PR changes during sinus rhythm. Sustained cycle length alternans during tachycardia could be a further indicator of this arrhythmia. In fact, initiation of stable 1:2 conduction over dual AV nodal pathways results in RR cycle alternans regardless of slow pathway conduction velocity, with the single exception in which the difference between slow and fast conduction times is half of the sinus cycle length (Fig. 7). Cycle length alternans also occurs in more common supraventricular arrhythmias such as atrial flutter or tachycardia, AV nodal reentrant tachycardia, and AV reentrant tachycardia. Sustained cycle length alternans, however, is unusual both in AV nodal and in AV reentrant tachycardia, but is relatively common in atrial tachycardia associated with 3:2 AV conduction ratio and Wenckebach periodicity. On the other hand, cycle length alternans appears as the rule in the case of tachycardia due to 1:2 AV conduction; all published reports show ECGs with RR alternans during tachycardia (6-9,11,12), although this finding is not mentioned. Thus, detection of sustained cycle length alternans during supraventricular tachycardia of undetermined origin should raise the suspicion of dual AV nodal pathways and dictate the need for accurate and detailed P wave sequence analysis in multiple ECG leads. A number of P waves half that of QRS complexes will establish the diagnosis of 1:2 conduction.

The electrophysiologic study may provide further but not conclusive clues, such as the constancy of HV interval of “normal” and “premature” beats, a response to programmed atrial stimulation consisting in lengthening coupling of the apparently “ectopic” beat with increasing prematurity of extrastimuli (11), and evidence of dual AV nodal physiology (6,9). Nevertheless, it is theoretically impossible to exclude an AV junctional ectopic focus giving rise to premature interpolated beats often occurring in bigeminy, or an accelerated AV junctional rhythm.

Antiarrhythmic drugs have been either ineffective or detrimental in most reported cases (6–11), as in our patient. Clinical improvement has been obtained with flecainide (9) or amiodarone (10), which was ineffective in our case. In contrast,
radiofrequency ablation of the slow AV nodal pathway represents a safe and definitive cure for this unusual arrhythmia (11,12). Disappearance of the multiple ECG manifestations of 1:2 AV conduction following successful radiofrequency application is probably the strongest element supporting the diagnosis.

References