A CASE OF ATRIOVENTRICULAR NODAL REENTRANT TACHYCARDIA WITH 2:1 INFRA-HIS BLOCK AND TYPE I GAP PHENOMENON

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We report a case of a 48-year-old female who had atrioventricular nodal reentrant tachycardia (AVNRT) with 2:1 infra-His block and type I gap phenomenon.

The atrioventricular nodal reentrant tachycardia (AVNRT) is the most common type of supraventricular tachycardia. To initiate and sustain AVNRT, functional dissociation of A-V nodal tissue is required. These pathways consist of a fast pathway with a longer refractory period and a slow pathway with a shorter refractory period. When an impulse is transferred from the fast pathway to the slow pathway, producing a significant conduction delay in the atrioventricular node, AVNRT would be initiated.

On the other hand, a gap phenomenon is sometimes recognized in the atrioventricular conduction system during atrial extrastimulation study. In particular, the type I gap phenomenon is known to require a conduction delay in the atrioventricular node to resume infra-His conduction! Therefore, the existence of dual atrioventricular pathways is one of the factors for the type I gap phenomenon.

Atrioventricular block accompanied by AVNRT is often observed during atrial stimulation study, but it is rare during a spontaneous attack. Combination of the gap phenomenon and atrioventricular block in a patient with AVNRT is relatively uncommon. We report a patient in which the type I gap and spontaneous 2:1 infra-His block coexisted during AVNRT.

CASE REPORT

The patient was a 48-year-old female. She visited our hospital with the chief complaint of recurrent episodes of palpitation. She had been suffering from the symptom since 20 years of age. It occurred several times per week, but she recovered within 30 min by rest. Recently, the frequency and duration of her tachycardia increased, therefore she was admitted to our hospital for further evaluation. Her past, and family history, were not contributory. Physical examination, laboratory findings, and chest X-ray were unremarkable.

A standard 12-lead electrocardiogram (ECG) at rest showed regular sinus rhythm, mild left axis deviation, no PQ prolongation, and no delta wave (Fig. 1A). The ECG at the attack revealed narrow QRS complex with the heart rate of 170 per minute without

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Fig. 1A. 12-lead electrocardiogram of normal sinus rhythm at rest.

Fig. 1B. 12-lead electrocardiogram on palpitation attack.

Fig. 2. Surface lead I, and an intracardiac electrogram from the high right atrium (HRA), a His bundle electrogram (HBE), and coronary sinus (CS) during supraventricular tachycardia with 2:1 infra-His block.

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any visible P wave (Fig. 1B). An electrophysiological study was performed at the nonabsorptive and nonsedated state after informed consent was obtained.

A quadripolar catheter was introduced percutaneously from the right femoral vein, a distal pair of electrodes was used for electrical stimulation and a proximal pair of electrodes for recording the electrogram of the high right atrium. Two bipolar catheters were introduced in the same fashion—one was positioned at the atrioventricular junction to record the His bundle electrogram, and the other was placed in the right ventricular apex for pacing. Another catheter was introduced percutaneously via the left subclavian vein and placed in the coronary sinus.

The patient was in sinus rhythm at the
beginning of the study. However, before the stimulation study, supraventricular tachycardia accompanied by 2:1 infra-His atrioventricular block was initiated spontaneously but it was terminated within about 30 sec (Fig. 2). During the sinus rhythm, the A-H interval was 75 msec, the H-V interval 45 msec. An atrial extra stimulation study was performed with the basic cycle length of 667 msec (S1-S1). When an atrial premature depolarization (APD) with a coupling interval of 390 msec (S1-S2) was delivered, both A2-H2 (110 msec) and H2-V2 (45 msec) interval were normal (Fig. 3A). Subsequently, an APD with a coupling interval of 380 msec induced the H2-V2 block with slight prolongation of the A2-H2 interval (Fig. 3B). The H2-V2 block was observed with gradual prolongation of the A2-H2 interval until a coupling interval of 320 msec (Fig. 3C). When an APD with a coupling interval of 310 msec was delivered, the A2-H2 interval markedly increased from 180 msec to 250 msec and H2-V2 conduction resumed (Fig. 3D).

When the coupling interval was shortened to 300 msec, supraventricular tachycardia was initiated after a significant increase of the A2-H2 interval (Fig. 4). The initiated tachycardia had the same activation sequence and the ventricular cycle length was doubled as in a spontaneous attack at the beginning of the study, and could be terminated by atrial overdrive pacing. Induction of supraventricular tachycardia was observed until the APD reached the atrial refractory period, with the coupling interval ranging from 300 msec to 240 msec of APD. The atrioventricular conduction curve showed discontinuation in the A2-H2 interval and H1-H2 interval. The plots of the H2-V2 interval also revealed discontinuation due to the gap phenomenon.

**DISCUSSION**

The gap phenomenon in the atrioventricular conduction system is classified by Damato et al. into 6 types of antegrade conduction of and 2 types retrograde conduction. In the type 1 gap, initial block occurs in the distal site of the His bundle, and its conduction is resumed when a conduction delay occurs in the atrioventricular nodes. Dual atrioventricular nodal pathways were considered to have close relation with the type 1 gap phenomenon. In our case, due to the shortening of the coupling interval of APD, the initial block was revealed in the His-Purkinje system at the coupling interval of 380 msec, and then His-Purkinje conduction resumed at the coupling interval of 310 msec with a sudden prolongation of the A-H interval.

These findings suggest that dual A-V nodal pathways and the type 1 gap phenomenon coexisted. In addition, our case consequent-
ly initiated atrioventricular nodal tachycardia. Mirvis et al\textsuperscript{3} reported a case who had dual A-V nodal pathways and the type 1 gap phenomenon. But their case had not been accompanied by supraventricular tachycardia. In concern with the atrioventricular block during AVNRT, Wellens et al\textsuperscript{4} reported that it was not rare. In their study, six were encountered in 58 patients with AVNRT, and 5 of them had 2:1 atrioventricular block. One of them was an unusual case accompanied with the gap and 2:1 block in the atrioventricular node.

Vassalo et al\textsuperscript{5} cited a case of AVNRT with 2:1 H-V block. Our case also showed infra-His 2:1 block. Wellens\textsuperscript{6} reported that the 2:1 block was often encountered during programmed stimulation, but was very rare spontaneously. In our case, it is of interest that it occurred spontaneously before the programmed stimulation study. Because the incremental atrial pacing up to 200/min during EPS did not induce the infra-His block, the vagal influence was strongly suspected in this phenomenon. Coexistence of spontaneous 2:1 infra-His block and the type 1 gap phenomenon during AVNRT in same patient is relatively rare.

REFERENCES