Adenosine sensitivity of retrograde fast pathway conduction in patients with slow-fast atrioventricular nodal reentrant tachycardia: A prospective study

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BACKGROUND It is suggested that the adenosine resistance of retrograde fast pathway in slow-fast atrioventricular nodal reentrant tachycardia (AVNRT) confirms the participation of a concealed retrograde atrio-Hisian pathway rather than the conventional fast pathway in the arrhythmia circuit of slow-fast AVNRT.

OBJECTIVE To prospectively assess the retrograde fast pathway response to the intravenous administration of adenosine in patients with typical AVNRT and the control group.

METHODS Electrophysiological parameters and adenosine sensitivity of retrograde fast pathway were studied in 21 consecutive patients (18 women; mean age 57 ± 10 years) with slow-fast AVNRT and 24 patients (11 women; mean age 46 ± 16 years) as the control group.

RESULTS Fifteen (71%) patients with AVNRT and 18 (75%) patients in the control group developed transient ventriculoatrial (VA) block after the intravenous administration of adenosine ($P = .79$). In patients with slow-fast AVNRT, female sex ($P = .003$), longer VA interval during right ventricular pacing ($P < .001$), and longer tachycardia cycle length ($P < .001$) predicted transient VA block after the intravenous administration of adenosine. In patients in the control group, a shorter VA interval during fixed rate right ventricular apical pacing ($P = .009$) and the presence of dual atrioventricular nodal physiology ($P = .002$) were associated with the adenosine resistance of the retrograde fast pathway.

CONCLUSIONS The prevalence of the adenosine resistance of retrograde fast pathway's conduction is comparable between patients with and those without slow-fast AVNRT. This finding can be explained better by the existence of an insulated intranodal tract with Purkinje-like properties or a superior atrionodal connection to the nodo-Hisian region of the atrioventricular node rather than the presence of an atrio-Hisian pathway.

KEYWORDS Atrioventricular node; Fast pathway; Adenosine; Ablation; Atrioventricular nodal reentrant tachycardia; Retrograde conduction

ABBREVIATIONS AV = atrioventricular; **AVNRT** = atrioventricular nodal reentrant tachycardia; **HA** = His-atrial; **RV** = right ventricular; **VA** = ventriculoatrial

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Introduction

Atrioventricular nodal reentrant tachycardia (AVNRT) is the most common regular supraventricular tachycardia.[1] There has been accumulating evidence suggesting that AVNRT most probably results from reentry in various locations in the atrioventricular (AV) nodal *and* atrial perinodal area.[2] Katritsis and colleagues[3,4] showed the antegrade and retrograde patterns of slow conduction by means of inferior, right and left, AV nodal extensions. Human studies have demonstrated histological and electrophysiological evidence of multiple atrial inputs to the AV node.[5,6] The heterogeneity of fast pathway (antegrade and retrograde) conduction has been well described in AVNRT. This heterogeneous retrograde conduction during slow-fast AVNRT and ventricular pacing is against an anatomically single retrograde fast pathway and more in favor of multiple superior nodal extensions resulting in different breakthrough sites in right and left atria.[6]

Otomo et al[7] assessed the retrograde fast pathway response to the administration of adenosine triphosphate in patients with typical AVNRT.[7] The authors showed that the retrograde fast pathway conduction was resistant in one-third of the cases, and therefore this finding in addition to the

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absence of the lower common pathway suggests a lower turnaround point in the His bundle region in the slow-fast AVNRT circuit and thus confirms the participation of a concealed retrograde atrio-Hisian pathway rather than the conventional fast pathway in the arrhythmia circuit.[7] These findings, however, did not exclude an insulated intranodal tract with Purkinje-like properties and/or a superior atrionodal connection to the nodo-Hisian region of the AV node as the additional possible explanation(s).[8,9]

We prospectively assessed the retrograde fast pathway response to the intravenous administration of adenosine and its electrophysiological properties in patients with typical AVNRT and compare their response with those of patients without clinical documentation of a regular supraventricular tachycardia who underwent an electrophysiological study that excluded the presence of an accessory pathway and showed the noninducibility of AVNRT in the control group. We postulated that if such a concealed atrio-Hisian pathway does exist and participates in the arrhythmia circuit of typical AVNRT, its prevalence should be higher in these patients compared with those in the control group.

Table 1 Patients' baseline characteristics

Characteristic	Group 1*	Group 2†	Total	P‡
No. of patients	21	24	45	
Sex: female	18 (86)	11 (45)	29 (64)	.001
Age (y)	57.3 ± 9.7	45.7 ± 16.1	51.1 ± 15.1	.005
DAVNP	21 (100)	14 (58)	35 (78)	.001
VA-AdT (ms)	161 ± 32	164 ± 34	162 ± 32	.82
HA-AdT (ms)	91 ± 34	102 ± 41	97 ± 38	.34
VA block (RFP)	15 (71)	18 (75)	33 (73)	.79

Values are presented as mean ± SD and as n (%).

AVNRT = atrioventricular nodal reentrant tachycardia; DAVNP = dual atrioventricular nodal electrophysiology; HA-AdT = His-high right atrium interval (in ms) during fixed rate ventricular pacing (S1-S1: 500 ms) before the intravenous administration of adenosine; RFP = retrograde fast pathway; VA = ventriculoatrial; VA-AdT = VA interval (in ms) during fixed rate ventricular pacing (S1-S1: 500 ms) before the intravenous administration of adenosine.

*Patients with slow-fast AVNRT.

†Control group (see text).

‡Comparison between groups 1 and 2.

Methods

Patients

Twenty-one consecutive patients (18 women; mean age 57 ± 10 years) with slow-fast AVNRT who underwent an electrophysiological study and slow-pathway ablation were enrolled in this prospective study. Classic electrophysiologic pacing maneuvers and criteria were used to confirm the diagnosis of slow-fast AVNRT and exclude other tachycardias, especially an accessory AV pathway.[10] In the control group, we included 24 (11 women; mean age 46 ± 16 years) consecutive patients without documented supraventricular tachycardia who underwent an invasive electrophysiological study for other reasons such as unexplained palpitation or syncope, which showed stable retrograde conduction via fast pathway during right ventricular (RV) apical pacing (S1-S1: 500 ms) and excluded AVNRT, accessory pathway, and other supraventricular tachycardias during the invasive electrophysiological study (Table 1).

Electrophysiological study and ablation

All antiarrhythmic drugs including β-blockers and calcium channel blockers were discontinued for at least 5 half-lives before the procedure. Each patient gave written informed consent before the procedure. Two quadripolar catheters (Inquiry, Irvine Biomedical Inc, St Jude Medical, Irvine, CA) were introduced from left femoral veins and placed in the RV apex and high right atrium (and coronary sinus), respectively. Another bipolar catheter was introduced from the left femoral vein and positioned at the superior septal tricuspid annulus for recording the *most proximal* His signal. We did not record the left-sided His signal and/or intervals. An ablation catheter (4-mm-tip IBI Therapy, Irvine Biomedical Inc) was inserted via the right femoral vein for mapping and ablation of slow pathway in the right atrium. Tachycardia induction and the assessment of antegrade and retrograde conduction properties were done using incremental and programmed stimulation (S1-S1: 500; 430; 370; and 330 ms with up to 3 extrastimuli) from the RV apex, high right atrium, and coronary sinus.

Ventriculoatrial (VA) and His-atrial (HA) intervals during ventricular pacing (S1-S1: 500 ms) were measured in all patients before the intravenous administration of adenosine (see below). In patients with slow-fast AVNRT tachycardia cycle length, VA and HA intervals during tachycardia and ventricular pacing with the tachycardia cycle length were also measured (Figure 1).

Finally, the radiofrequency ablation of slow pathway was conventionally done by using a 4-mm non–irrigated-tip ablation catheter during sinus rhythm targeting the slow pathway potential in the right posteroseptal region with a maximum power of 40 W and a target temperature of 60°C by using widely accepted criteria as end point(s), as described elsewhere.[10]

Intravenous adenosine

Patients were excluded if adenosine administration was contraindicated. During fixed rate RV apical pacing (500 ms) with stable 1:1 VA conduction over the retrograde fast pathway, a single dose of adenosine (maximum 20 mg) was administered intravenously through a central venous sheath followed by rapid saline bolus. Patients were divided into 2 groups with (n = 33 [73%]) and without (n = 12 [27%]) transient VA block after the intravenous administration of adenosine.

Statistical analysis

Variables are expressed as mean ± SD (or median [range], if appropriate) and number (percentage). Differences in the frequency of characteristics were assessed by using the independent sample *t* test for continuous variables. For