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An uncommon case of spontaneous conversion from AV re-entry tachycardia to AV nodal re-entry tachycardia in a patient with dual tachycardia

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ABSTRACT

We report the case of a 46-year old patient in whom an electrophysiology study (EP) was performed due to paroxysmal supraventricular tachycardia documented in 12-lead ECG. During the EP study, supraventricular tachycardia was induced easily and it corresponded to orthodromic AV reentry tachycardia (AVRT) using a concealed left free wall accessory pathway. However, during the study AVRT spontaneously and repeatedly converted to the typical slow-fast AV node reentry tachycardia (AVNRT). Both accessory and AV nodal slow pathways were ablated, due to the finding that both AVRT and AVNRT were independently inducible during the EP study.

Introduction

Electrophysiology (EP) study with catheter ablation is the favourable method in treatment of patients with paroxysmal supraventricular tachycardia (SVT) according to current ACC/AHA/ESC guidelines [1], with high success and low complication rates. The two most common paroxysmal SVTs are atrioventricular nodal reentry tachycardia (AVNRT) and orthodromic atrioventricular reentry tachycardia (AVRT) using an accessory pathway (AP) [1,2]. However, both types of tachycardias can rarely be found in the same patient [2,3]. We describe a patient with both orthodromic AVRT and slow-fast AVNRT, in whom the AVRT spontaneously and repeatedly converted to the typical slow-fast AVNRT and both tachycardias were independently inducible during the EP study.

Case report

Clinical history

A 46-year old woman, with history of hypertension and documented regular, narrow QRS tachycardia, was referred to our institution for EP study. In the last five months she was treated 6 times in the Emergency Room (ER), always presenting with palpitations, dizziness, hypotension and narrow QRS complex tachycardia. In available ECGs during the

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tachycardia, there were no visible P waves. Tachycardia was always terminated with adenosine. At admission, ECG showed normal sinus rhythm without pre-excitation and echocardiography demonstrated no structural heart disease except mild left ventricular hypertrophy with grade I diastolic dysfunction.

Electrophysiology study and tachycardia characteristics

An electrophysiology study was performed with the patient in a non-sedated state. Quadripolar diagnostic catheters JSN 5F (St. Jude Medical, St. Paul, Minnesota, USA) were positioned in high right atrium, right ventricular apex and His area, and steerable decapolar catheter EZ Steer (Biosense Webster, Diamond Bar, California, USA) was positioned into coronary sinus.

At baseline, sinus cycle length, AH and HV intervals were normal. During atrial programmed stimulation jump was induced at 500/280 ms. Supraventricular tachycardia was induced repeatedly with atrial and ventricular pacing and it corresponded to orthodromic AV re-entrant tachycardia – AVRT (cycle length 330 ms) using a concealed, left free wall accessory pathway (AP). After cessation of ventricular overdrive pacing, the same tachycardia resumed with a measurement that correlated to AVRT (VAV pattern, post-pacing interval (PPI) – tachycardia cycle length (TCL) = 100 ms, His synchronous extra-stimulus advanced the atrial activation) (Fig. 1).

However, during the EP study the AVRT spontaneously converted (Fig. 2) to the typical slow-fast AVNRT (cycle length 360 ms), with characteristics: VA < 30 ms, PPI – TCL > 150 ms, jump noted at programmed pacing (500/280 ms) from right atrium as well as the echo beat.

Noteworthy, both tachycardias were independently inducible during atrial and ventricular pacing (Fig. 3), without conversion of AVNRT to AVRT.

A slow pathway was localized and then ablated using radio-frequent (RF) catheter ablation (temperature controlled mode 50°C/30 W; Blazer II XP Standard curve, Boston Scientific, Massachusetts, USA), with slow-rate junctional rhythm observed during ablation and no signs of slow AV node pathway after ablation (ERP N antegrade 260 ms, antegrade suprahisian AV block <300 ms). After RF ablation, AVNRT could not be induced; however the AVRT was repeatedly induced without conversion to AVNRT. By performing a transseptal puncture of the interatrial septum guided by fluoroscopy, the left lateral wall AP was also successfully localized and then ablated using RF catheter ablation (temperature controlled mode 50°C/30 W; Blazer II XP Standard curve, Boston Scientific, Massachusetts, USA) (Fig. 4). During the RF ablation, retrograde AP conduction block was achieved.

After the RF ablation, neither AVRT nor AVNRT could be induced at baseline nor after the infusion of isoproterenol (up to 6 mcg/min iv.). No procedure-related complications occurred during or immediately after the procedure, as well as during 1-year follow-up after ablation. During 1-year follow-up neither AVRT nor AVNRT recurred.

Discussion

AVRT and AVNRT have prevalence of 2.25/1000 persons and the incidence of 35/100000 person-years in general population [2], with the fact that these two most common supraventricular tachycardias can occasionally be found together in a single patient [2–4]. Some authors achieved complete cure by ablating the common limb of both tachycardias: slow AV node pathway [4] or only AP [5] in patient in whom the AVNRT did not seem to be the patient's "clinical" tachycardia but tachycardia-induced tachycardia.

In this case, the ablation of both pathways was done due to two facts. First, both tachycardias were independently inducible, which is very rare, therefore determining the "clinical" tachycardia was rather difficult. Although the ECGs from the ER correspond most likely to AVNRT (no P waves

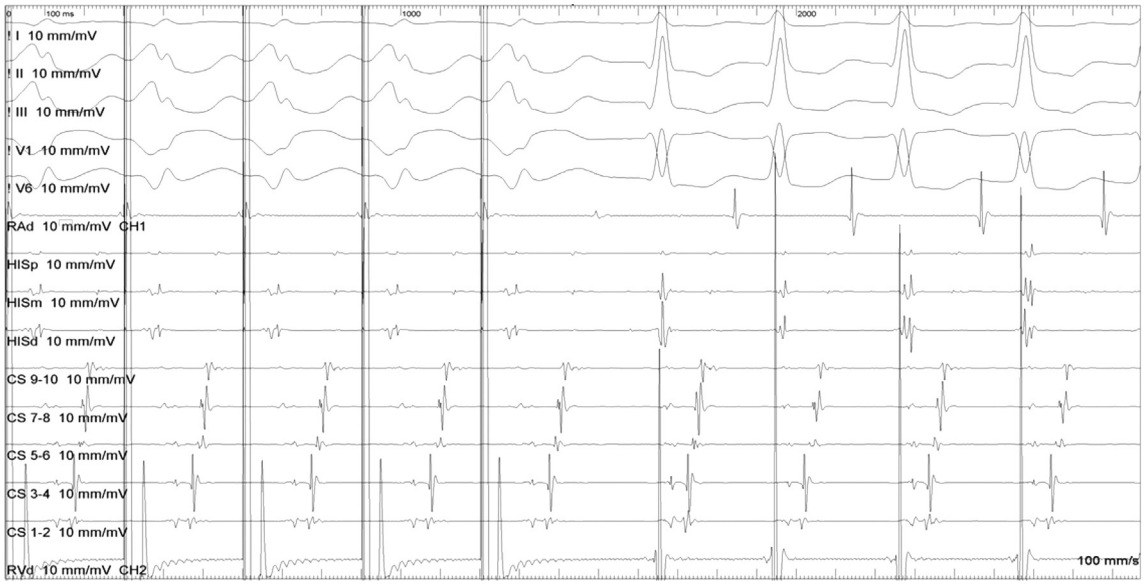

Fig. 1 – Entrainment during AV re-entry tachycardia.

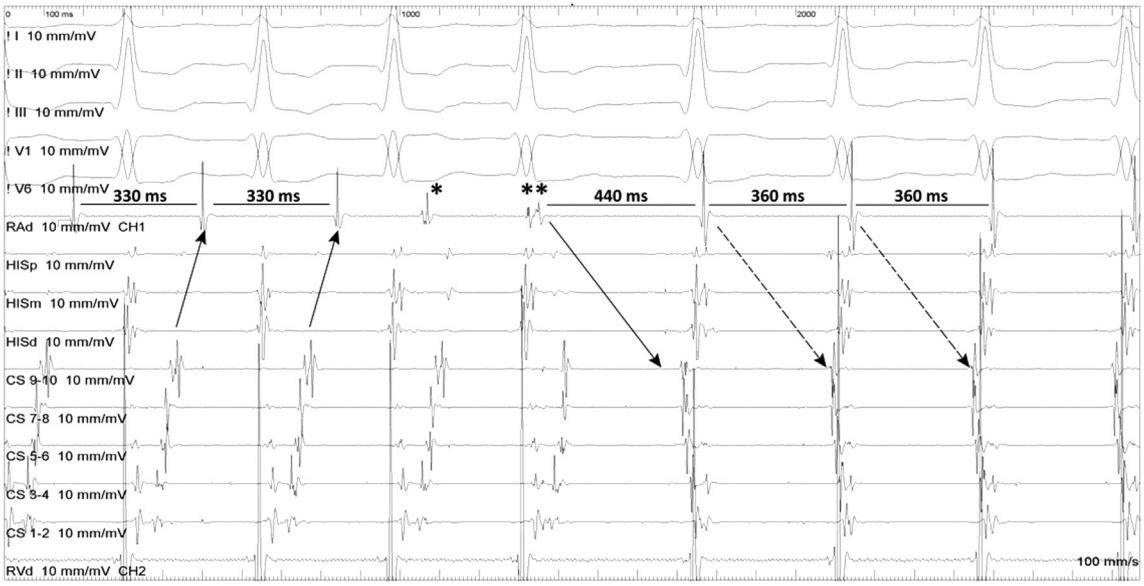

Fig. 2 – Spontaneous conversion from AV re-entry tachycardia to AV nodal re-entry tachycardia. Black arrows – show the retrograde conduction trough accessory pathway (eccentric atrial activation). * and ** show premature atrial contractions. The first one, marked with * did not affect the tachycardia while the other marked with ** found the fast pathway refractory and engaged the slow pathway initiating the AVNRT. Black discontinuous arrows – show the antegrade conduction trough slow AV node pathway.

visible) the tachycardia which was induced more easily in the EP lab was AVRT which spontaneously converted to AVNRT. It can only be speculated whether this might have been the clinical case. Second, the spontaneous and immediate transition from one tachycardia into another is rather uncommon. It should be emphasised that Kuo et al. [3] proved that the conversion of one tachycardia into another is most common with tachycardia whose cycle lengths difference is < 25 ms with 80% positive predictive value, whereas in our case the difference was 30 ms.

Considering the previously mentioned, we ablated both slow and accessory pathways. It is recommended that during each EP study a complete EP protocol should be applied and every possible manoeuvre should be made in order to obtain a confident diagnosis since multiple tachycardia can appear in one patient and in different combinations [1,3–5].

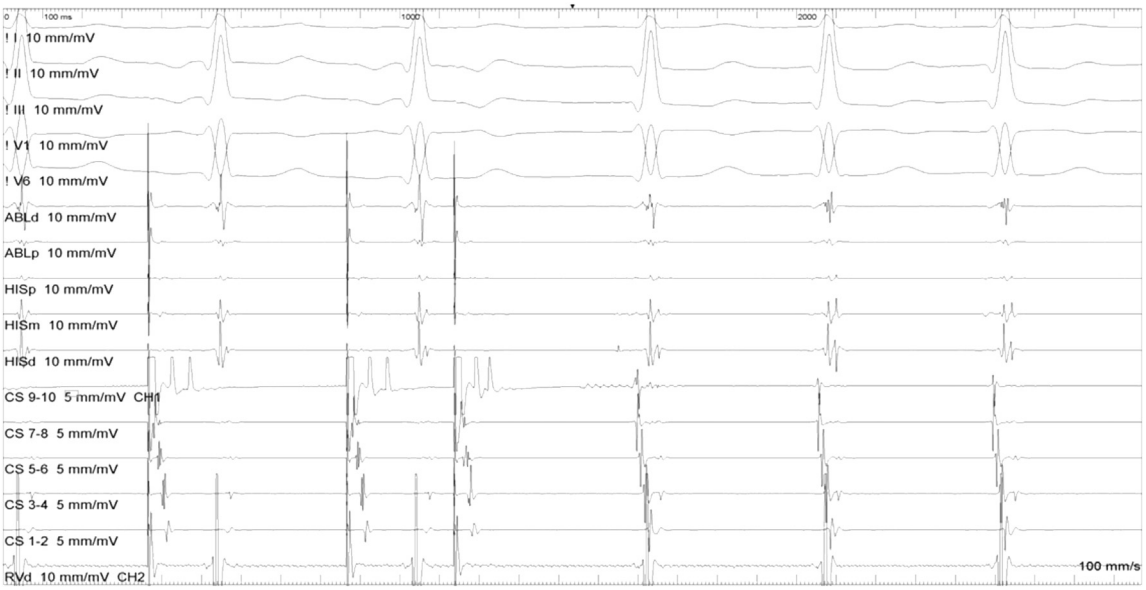

Fig. 3 – Induction of AV nodal re-entry tachycardia.

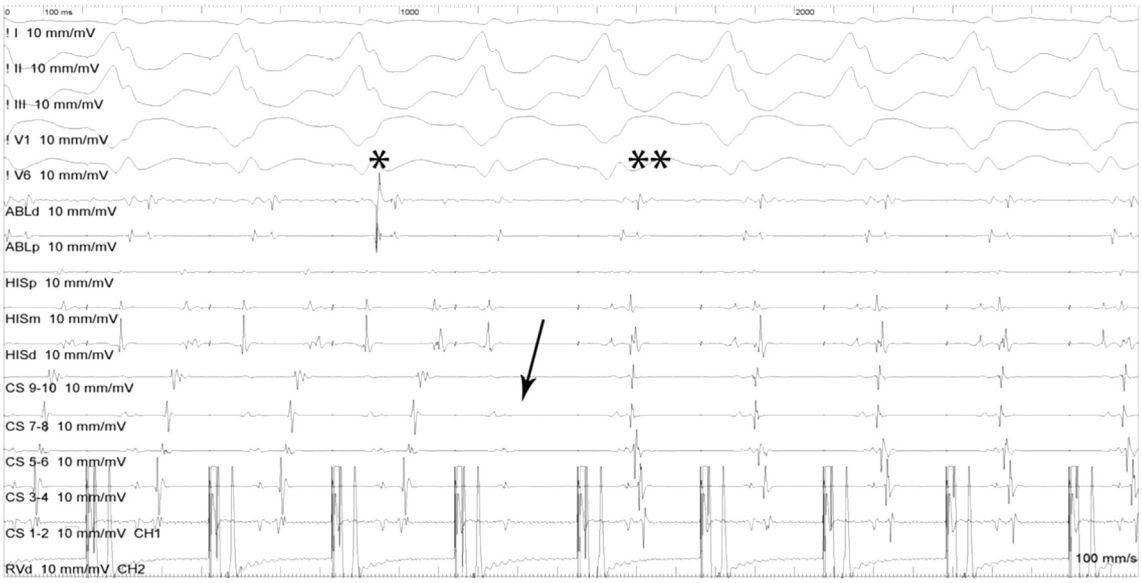

Fig. 4 – Mapping of the left lateral accessory pathway.
*** start of the RF energy application.**
Black arrow –points to the retrograde block.
**** change in retrograde atrial activation from eccentric to concentric (conduction over the AV node).**

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