Recurrent spontaneous clinical perimitral atrial tachycardia in the context of atrial fibrillation ablation

Shinsuke Miyazaki, MD, Ashok J. Shah, MD, Mélèze Hocini, MD, Michel Haïssaguerre, MD, Pierre Jaïs, MD

From the Hôpital Cardiologique du Haut-Lévêque and the Université Victor Segalen Bordeaux II, Bordeaux, France.

BACKGROUND Recurrent perimitral atrial tachycardia (AT) is a challenging arrhythmia and is frequently encountered in the context of atrial fibrillation (AF) ablation.

OBJECTIVE The purpose of this study was to investigate the clinical characteristics and the procedural and clinical outcomes in patients with recurrent perimitral atrial tachycardia (PMAT) after AF ablation.

METHODS Among 520 consecutive ablation procedures for recurrent AT/AF after AF ablation, 40 procedures (patients) were performed for clinically recurrent PMAT 12.1 ± 13.6 months after the last procedure (total 2.2 ± 1.3 procedures). Previously, mitral isthmus (MI) linear ablation was performed in 26 of 40 procedures, including 13 procedures with complete block and 13 with 159.0 ± 23.0 ms of conduction delay without block. As a reference group, conduction delay was evaluated in 55 patients with incomplete MI block and absence of spontaneous PMAT during the follow-up period.

RESULTS Recurrent PMATs were terminated by MI linear ablation in 26 of 40 patients. Bidirectional block across the MI and anterior line joining the mitral annulus and left atrial roof was achieved in 33 (82.5%) and 2 (5%) patients, respectively. At mean follow-up of 26.7 ± 14.5 months, 2 patients (5%) underwent reablation for spontaneously recurrent PMAT. At 12 months after the ablation procedure for PMAT, 73.5% of the patients were free from AT/AF. Conduction delay >149 ms predicted the occurrence of spontaneous PMAT with 80.0% sensitivity and 87.3% specificity.

CONCLUSION PMAT can recur even after successful bidirectional MI linear block. Substantial conduction delay without block across the MI from a previous procedure(s) could predispose to recurrent PMAT. Although most clinical PMATs can be successfully treated by catheter ablation, very late recurrence is possible.

KEYWORDS Atrial fibrillation; Catheter ablation; Linear ablation; Mitral isthmus

ABBREVIATIONS **AF** = atrial fibrillation; **AT** = atrial tachycardia; **CS** = coronary sinus; **IQR** = interquartile range; **LA** = left atrium; **MI** = mitral isthmus; **PMAT** = perimitral atrial tachycardia; **PV** = pulmonary vein; **RF** = radiofrequency

(Heart Rhythm 2015;12:104–110)

Introduction

Catheter ablation of atrial fibrillation (AF) is widely performed in symptomatic, drug-refractory patients.[1,2] However, despite the success of catheter ablation in treating AF, recurrent atrial tachycardia (AT) is a major problem of AF ablation.[3–8] A previous report showed that some of the spontaneous and induced ATs in the context of AF ablation resolved spontaneously.[9] Perimitral atrial tachycardia (PMAT) is the most common macroreentrant AT in the context of AF ablation and is the most challenging arrhythmia because of the difficulty in creating permanent complete block across the line, which is the only strategy for preventing further recurrence.[10,11] However, the characteristics of the patients with clinical spontaneous recurrent PMAT in the context of AF ablation have not been fully evaluated. The aim of this study was to investigate the clinical characteristics and the procedural and clinical outcomes in patients with spontaneous recurrent PMAT in the context of AF ablation.

Methods

Between January 2007 and November 2010, patients who underwent repeat ablation of clinical PMAT occurring after an AF ablation procedure were included in the study. To focus on clinical PMAT, we included patients with spontaneous clinical AT that persisted and allowed complete diagnostic mapping during the procedure. PMAT induced by programmed stimulation including entrainment maneuver or mechanically during catheter manipulation and those arising from the organization of AF during ongoing radiofrequency (RF) application were excluded. PMAT was diagnosed by deductive mapping strategy, which consists of activation mapping and entrainment mapping without the use of a 3-dimensional system.[12]

Address reprint requests and correspondence: Dr. Shinsuke Miyazaki, Hôpital Haut Lévêque 33604 Bordeaux-Pessac, France. E-mail address: mshinsuke@k3.dion.ne.jp.

As a reference group, we selected consecutive patients during the same study period in whom spontaneous clinical PMAT was not observed during the follow-up period and perimitral conduction delay evaluated at the end of procedure revealed incomplete MI block. AF was defined according to the HRS/EHRA/ECAS 2012 Consensus Statement on Catheter and Surgical Ablation of AF.[13] All patients gave written informed consent. The study protocol was approved by the hospital's institutional review boards. The study complied with the Declaration of Helsinki.

Electrophysiologic study

All antiarrhythmic medications except for amiodarone were discontinued at least 5 half-lives before ablation. All patients were anticoagulated with warfarin for at least 1 month before the procedure (target international normalized ratio 2–3), and therapeutic anticoagulation was maintained with intravenous or low-molecular-weight heparin after warfarin discontinuation 3 days before intervention. Transesophageal echocardiography was performed within 48 hours before the procedure to exclude LA thrombus. Warfarin was restarted on the day of the procedure, and effective anticoagulation was maintained with heparin until the international normalized ratio was >2.0.

Surface ECG and bipolar endocardial electrograms (filtered from 30–500 Hz) were continuously monitored and stored on a computer-based digital amplifier/recorder system (Labsystem Pro, Bard EP, Lowell, MA). Electrophysiologic study was performed with the patients under mild sedation. The following catheters were introduced via the right femoral vein: (1) a deflectable quadripolar or decapolar catheter (Xtrem, ELA Medical, France) positioned within the coronary sinus (CS). with the distal electrode positioned at the 4 o'clock position along the mitral annulus in the 30° left anterior oblique radiographic projection; (2) a 10 pole, fixed-diameter (20-mm) circumferential mapping catheter to guide pulmonary vein (PV) isolation (Lasso, Biosense Webster, Diamond Bar, CA), introduced with the help of a long sheath (Preface multipurpose, Biosense Webster, or SL0, St. Jude, St. Paul MN), which was continuously perfused with heparinized saline; and (3) a 3.5-mm externally irrigated-tip quadripolar ablation catheter (ThermoCool, Biosense Webster). After transseptal puncture, a 50 IU/kg heparin bolus was administered and repeated only if the procedure lasted for more 4 hours.

Catheter ablation of AF

RF ablation was performed with maximum power of 25 to 35 W using external irrigation at 10–60 mL/min to a target tissue temperature ≤ 43°C. Power was limited to 30 W on the posterior wall. At the outset, ostial PV isolation was performed segmentally under the guidance of a circumferential mapping catheter positioned in the PV. In patients with ongoing AF after PV isolation, electrogram-based ablation was performed for 20–60 seconds at sites showing characteristic electrograms,[2,14] with an end-point of local organization and prolongation of local AF cycle length. If AF continued after PV isolation and electrogram-based ablation, linear ablation of the roof followed by the mitral isthmus (MI) was undertaken with an end-point of elimination of local electrograms. If sinus rhythm was restored, bidirectional conduction block assessed using a differential pacing technique was the end-point of successful linear ablation.[10,15,16]

The procedural end-point for paroxysmal AF was electrical isolation of all PVs. Induction of AF was attempted, and AF lasting ≤ 10 minutes was considered noninducible. If AF was inducible, substrate ablation was performed. The procedural end-point for persistent AF was conversion to sinus rhythm or organized AT, which was subsequently mapped and ablated.[12] When AF was not terminated by ablation, electrical cardioversion was undertaken. If required, ablation was continued in sinus rhythm to achieve PV isolation and conduction block across the linear lesion(s).[10,15,17]

Catheter ablation of perimitral AT

Ablation of PMAT consisted of creating a line of conduction block between the left inferior PV and the lateral mitral valve annulus.[10,11] RF energy was limited to a maximum power output of 35 W endocardially, maximum temperature was limited to 43°C, and irrigation rate was titrated to maintain an electrode temperature >38°C. Epicardial ablation from the CS was attempted when no obvious atrial potential was mapped on the MI line or when a large atrial potential was observed in the CS catheter despite ablation endocardially. RF energy was limited to a maximum power output of 25 W. Maximum irrigation rate was 60 mL/s, and temperature was limited to 43°C. The end-point of MI ablation was establishment of bidirectional conduction block, which was assessed using a differential pacing technique in sinus rhythm.[15] In brief, pacing the distal bipole of the CS catheter placed just septal to the linear MI lesion results in a longer stimulus-to-electrogram time, measured with the ablation catheter positioned just lateral to the line, compared with pacing from the next more septal bipole of the CS catheter (Figure 1A). On pacing from the LA appendage located lateral to the line of block, a proximal-to-distal activation sequence is seen on the CS catheter positioned with its distal bipole located septal to the line (Figure 1B). This confirms bidirectional conduction block across the MI lesion. Widely separated local double potentials equidistant along the length of the ablation line were often observed. In patients in whom PMAT could not be terminated by ablation, PMAT was terminated by atrial burst pacing or direct current cardioversion. After termination of PMAT, further RF application was undertaken on the MI line while pacing just septal to the line of block at a cycle length of 600 ms. Perimitral conduction delay was measured as the temporal delay to the second component of the double potential on the MI line.[16]

Follow-up

Patients were hospitalized for up to 5 days postprocedure and again for 1 day at 1, 3, 6, and 12 months for clinical interview and ambulatory monitoring in addition to routine follow-up with the referring cardiologist and Holter undertaken in the