

Coronary sinus reentrant tachycardia after atrial fibrillation ablation: From bad to worse*

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ABSTRACT

Herein we present a case of atrial tachycardia as a sequel of AF ablations. A 42-year-old man was admitted to our department because of a very symptomatic tachycardia. The patient, because of paroxysmal AF and typical atrial flutter, had been already submitted (three times) to ablation procedures in both left (pulmonary vein insulation) and right atria (cavo-tricuspidal isthmus). During the electrophysiological study, a huge and very fast atrial tachycardia was induced: 230 ms cycle length, 1/1 atrio-ventricular conduction with the ventricular rate of 260 bpm, complete left bundle branch block, and clinically recognized by the patient. Four minutes later, a 2/1 AV conduction without branch block permitted mapping and ablation. A high-density mapping around isthmus and coronary sinus (CS) was performed. The analysis of the chronological activation clearly showed a circuit propagation around the CS ostium with a very slow conduction in the anterior zone enlightened by the tight color progression, and counterclockwise activation of the right atrium lateral wall. In anterior zone of CS ostium diastolic fragmented electrograms were detected. After preventing his position localization, radiofrequency delivering (35 W) was effective

to interrupt the arrhythmia in 3 seconds. Energy delivering was continued to anchor the lesion to the inferior vena cava. Confirmation of successful ablation was determined by unsuccessful attempts at reinduction of the arrhythmia, in basal state and during infusion of isoproterenol.

KEYWORDS

Atrial Flutter; Atrial Fibrillation; Transcatheter Ablation; Three-Dimensional Mapping

1. INTRODUCTION

The increased number of transcatheter ablations in the atria for treatment of atrial fibrillation (AF) contributes to an increased incidence of organized macro reentrant tachycardia [1-4].

Herein we present a case of atrial tachycardia as a sequel of AF ablations.

2. CASE REPORT

A 42-year-old man was admitted to our department because of a very symptomatic tachycardia. The patient, because of paroxysmal AF and typical atrial flutter, had been already submitted (three times) to ablation procedures in both left (pulmonary vein insulation) and right atria (cavo-tricuspidal isthmus).

In spite of anti-arrhythmic therapy, the patient, four days before his admission, experienced a new very symptomatic (sweating, hypotension and dizziness) tachycardia for which was submitted to ablation procedure again.

At the procedure the patient, on pharmacological wash-out, presented a stable sinus rhythm.

By coronary sinus (CS) stimulation, with extra stimulus technique, a huge very fast atrial tachycardia was induced: 230 ms cycle length, 1/1 atrio-ventricular con-

*Core Tip. The increased number of transcatheter ablations in the atria for treatment of atrial fibrillation is contributing to an increased incidence of organized macro reentrant tachycardia.

Herein we present a case of atrial tachycardia as a sequel of atrial fibrillation ablations. During EP study a huge very fast, 260 bpm, atrial tachycardia was induced. A high-density mapping (Carto) indicated a reentry circuit around CS. In anterior edge of CS diastolic fragmented electrograms were localized. Radiofrequency delivering was effective to interrupt the arrhythmia. Energy delivering was continued to anchor the lesion to the inferior vena cava. In conclusion, extensive liner lesions in right and left atria and normal anatomical barriers (tricuspidal annulus, Eustachian ridge and crista terminalis, scars) could contribute to the onset of conduction corridors (man-made maze) that could sustain more aggressive arrhythmias.

duction with ventricular rate of 260 bpm, complete left bundle branch block and clinically recognized by the patient (**Figure 1**). Four minutes later a 2/1 AV conduction without branch block permitted mapping and ablation (**Figure 2**).

A 7.5 F 3.5 mm irrigated-tip roving catheter (Navistar Thermocool, Diamond Bar, CA, USA) inserted by right femoral vein for electroanatomical mapping and ablation was used.

Electroanatomical mapping (CARTO 3, Diamond Bar, CA, USA) commenced in the right atrium because of the CS proximal-distal activation sequence. In order to reconstruct the chronology of the entire macroreentry circuit and to identify the mid-diastolic isthmus (MDI), a specific setting of the window of interest was used, as previously described [1]. Chronological activation analysis on the entire cycle (230 ms) indicated the MDI (the interface between the red and the purple colors) of typical atrial flutter, but more anterior. A decapolar catheter showed a typical counterclockwise activation along tricuspidal annulus and an enormous delay from low lateral right atrium-CS ostium, more than would be expected in case of a slow conduction (low lateral-proximal CS interval of 120 ms.). A high-density mapping around isthmus and CS was performed (**Figure 3**). The re-analysis of the chronological activation clearly showed a circuit propagation around the CS ostium with a very slow

conduction in the anterior zone enlightened by the tight color progression, and counterclockwise activation of the right atrium lateral wall (**Figure 3**). In anterior zone of CS ostium diastolic fragmented electrograms were detected. Entrainment that could have confirmed that suspected path was truly in the circuit, was not performed to avoid atrial fibrillation degeneration. After preventive His position localization, radiofrequency delivering (35 W) was effective to interrupt the arrhythmia in 3 sec. Energy delivering was continued to anchor the lesion to the inferior vena cava. Confirmation of successful ablation was determined by unsuccessful attempts at reinduction of the arrhythmia, in basal state and during infusion of isoproterenol.

3. DISCUSSION

In the era of transcatheter ablation procedures in the right and left atria to treat AF, incidence of organized macro-reentrant tachycardia, as a sequel of extensive linear ablation, is 5% - 30% [2-4]. In Chugh *et al.* series on atrial flutters that arose after left atrial circumferential ablation of AF, in approximately 25% of cases they used the musculature of the CS [2]. Furthermore, Chae *et al.* reported a series of 21 micro-reentrant atrial tachycardias that occurred after circumferential pulmonary vein ablation. They indicated that the most common sites of origin

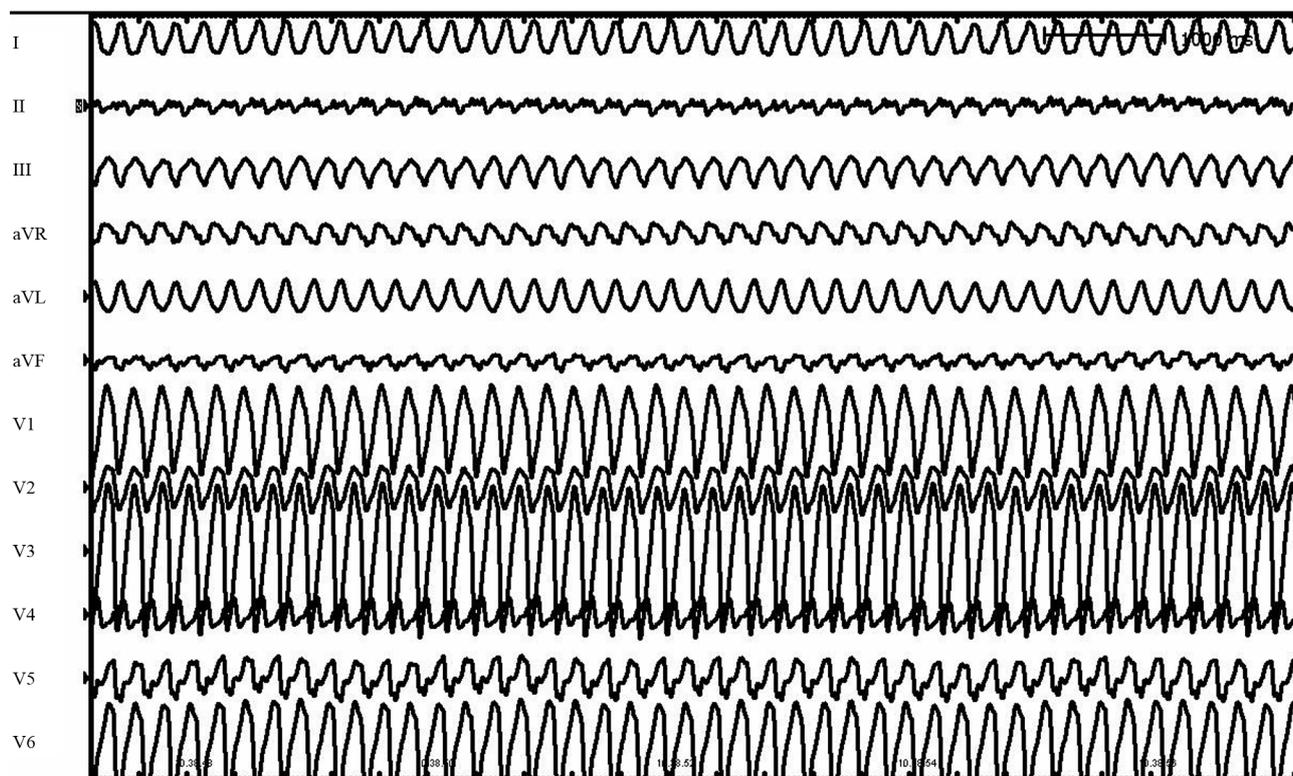


Figure 1. Induction of the huge very fast atrial tachycardia, 230 ms cycle length with 1/1 atrio-ventricular conduction (ventricular rate of 260 bpm) and complete left bundle branch block. I, II, III, aVR, aVL, aVF, V1, V2, V3, V4, V5, V6 are surface electrograms.

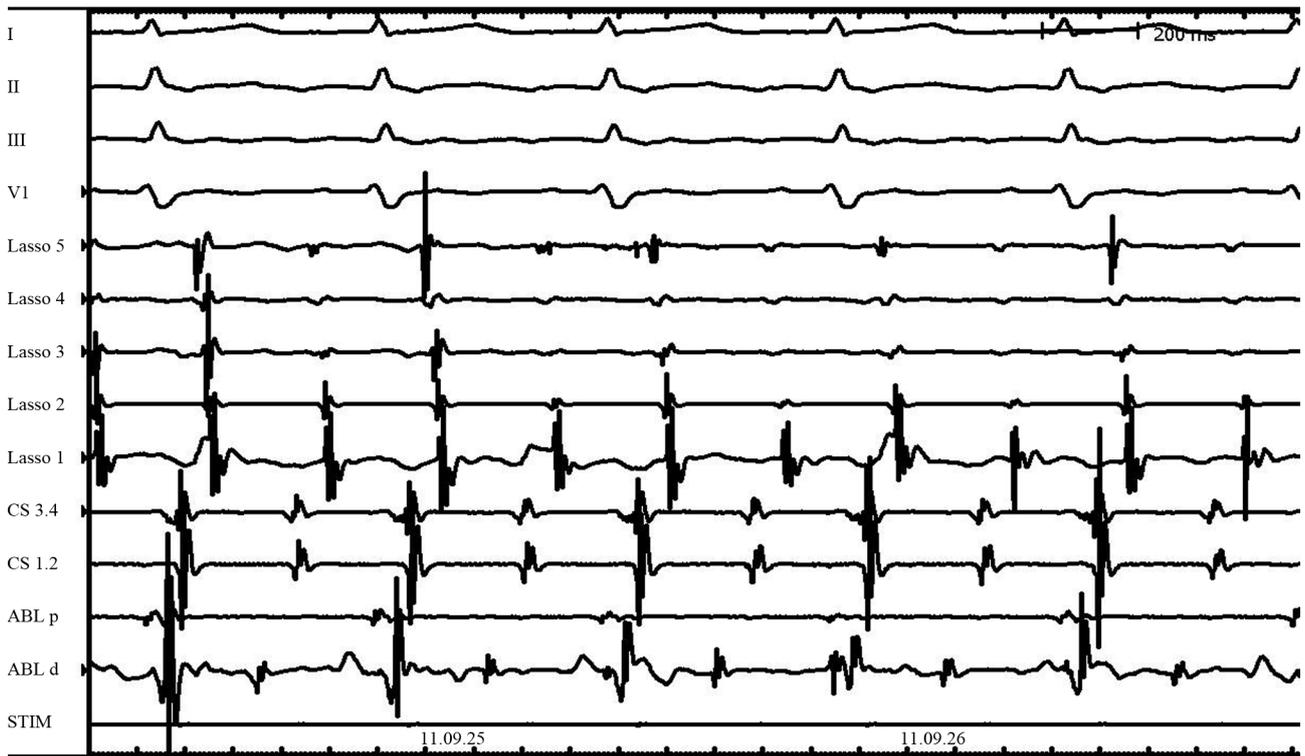


Figure 2. The atrial tachycardia with a 2/1 AV conduction without branch block. A decapolar catheter shows a typical counterclockwise activation along tricuspidal annulus and an enormous delay from low lateral right atrium-CS ostium (120 ms interval). I, II, III, V1 are surface electrograms; Lasso 5, Lasso 4, Lasso3, Lasso 2, Lasso 1 are electrograms from decapolar catheter; CS 3-4, CS 1-2 are electrograms from coronary sinus; ABL p, ABL d are proximal and distal electrograms from mapping/ablation catheter.

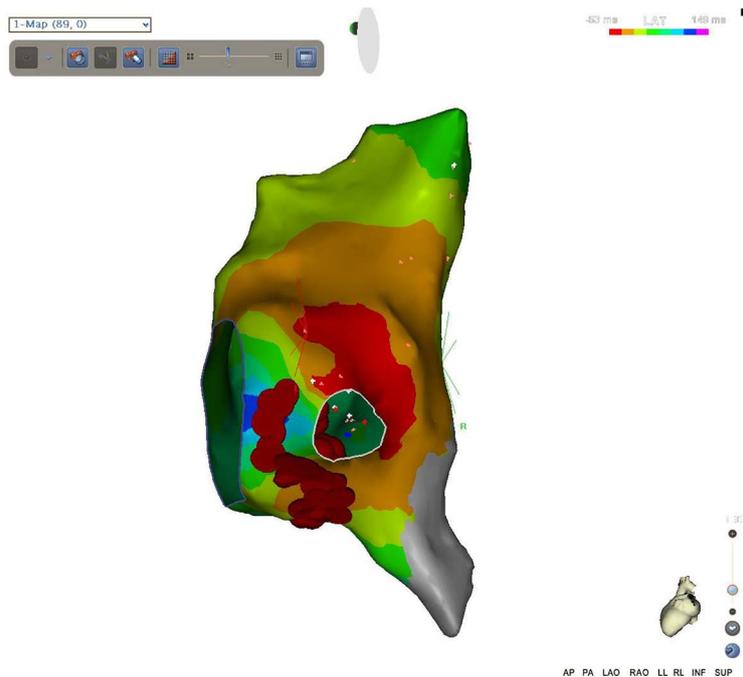


Figure 3. Isochronal map (LAO view) of tachycardia reentry counterclockwise circuit around coronary sinus ostium. Electrogram steps from -53 ms to + 149 ms (about 90% of cycle length) are encoded with colors from red (earlier) to pink (latter). To note the slow conduction in the anterior zone enlightened by the tight color progression.

were the CS, the atrium of a pulmonary vein, and the anterior wall of the left atrium or base of the left atrial appendage [3].

Morady *et al.* reported that 75% of atrial tachycardias occurred after wide-area radiofrequency ablation to isolate the pulmonary veins is caused by macroreentry also around CS [4].

Extensive linear lesions in right (isthmus, intercaval, superior vena cava) and left atria (circumferential pulmonary vein isolation, roof, anterior and posterior left isthmus) and normal anatomical barriers (tricuspidal annulus, Eustachian ridge and crista terminalis, scars) could contribute to the onset of conduction corridors: a man-made maze. In our case the stimulus goes slowly around CS ostium, especially in anterior zone, and spreads through the right atrium corridors with a fast repetitive sequence with a counterclockwise activation of the lateral wall.

The result of extensive linear ablations is the onset of more organized tachyarrhythmias, often with rapid ventricular rates, persistent and difficult to suppress with rhythm control medications: from bad to worse.

4. CONCLUSIONS

In conclusion, our experience suggests that in case of extensive ablation lines for persistent atrial fibrillation or redo of paroxysmal atrial fibrillation, the highest therapeutic effectiveness is obtained by mapping and ablating all remaining circuits. At the last step, for the remaining circuit underlying a tachycardia with a more or less constant cycle length, electroanatomical mapping and ablation are mandatory.

This behaviour could prevent the arising of new tachyarrhythmias (man-made) which in the majority of cases is more symptomatic than atrial fibrillation.

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