



Case Report

Focal ablation for atrial tachycardia from the double-exit of the Marshall bundle inducing atrial fibrillation

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ABSTRACT

Atrial fibrillation (AF) from the ligament/vein of Marshall (LOM/VOM) has previously been described. We report the case of a 23-year-old woman with an antiarrhythmic drug-resistant AF induced by two distinct atrial tachycardias (ATs). Focal ablation of these ATs from the double-exit of the Marshall bundle using a three-dimensional map eliminated AF triggering, even though pulmonary vein electrical isolation is the cornerstone for paroxysmal AF. Such mechanisms are important as triggering factors to plan ablation for paroxysmal AF. Focal ablation for triggering and inducing AF, originating from the double-exit of the Marshall bundle may be effective in eliminating AF in young patients.

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1. Introduction

Atrial fibrillation (AF) is the most common sustained atrial arrhythmia with various distinct mechanisms. In addition to the pulmonary veins (PVs), AF also originates from the catecholamine-sensitive ligament/vein of Marshall (LOM/VOM). The muscle sleeves of the LOM/VOM are electrically active structures; rapid electrical firing from the Marshall bundles can initiate and degenerate paroxysmal AF [1,2].

We report a case with catecholamine-sensitive paroxysmal AF induced by bi-focal atrial tachycardia (AT) from the double-exit of the Marshall bundle, which was successfully ablated using focal radiofrequency (RF).

2. Case report

The patient was a 23-year-old woman who had a 3-year history of palpitation and chest discomfort associated with stressful events. The clinically documented ECG (*electrocardiogram*) showed a paroxysmal irregular, narrow QRS complex tachycardia, which indicated paroxysmal AF (Fig. 1A and B). The arrhythmia was uncontrolled with anti-arrhythmic medications, including bisoprolol, propafenone, and flecainide. All antiarrhythmic drugs were discontinued for more than five half-lives before the electrophysiological study. A baseline electrophysiology study was

planned, in which quadripolar electrode catheters (St. Jude Medical, Inc., Minnetonka, MN, USA) were positioned to record the activity of the His bundle and right ventricular (RV) apex; the catheters were inserted via the left femoral vein. The high right atrium (RA), low RA, and coronary sinus (CS) were mapped with a deflectable duo-decapolar catheter (St. Jude Medical, Inc., St. Paul, MN, USA) inserted via the left femoral vein. Intracardiac electrograms were recorded using the Prucka CardioLab™ electrophysiology system (General Electric Health Care System, Inc., Milwaukee, WI, USA). Dual transeptal puncture was performed and an SL1 sheath (St. Jude Medical, Inc.) was advanced to the left atrium (LA). Mapping was performed using a ten-pole circular mapping catheter (Navistar Lasso catheter, Biosense Webster, Diamond Bar, CA, USA). RF ablation was delivered using an open irrigated 7 F, 3.5 mm tip, deflectable ablation catheter (Thermocool Navistar, Biosense Webster, Inc. Diamond Bar, CA, USA). Generator power was limited to 30 W during ablation. RF ablation was continued at each site until local electrograms were abolished, or for 30 s. A three-dimensional rendering of the LA was created from a multi-slice computed tomography image of the LA integrated with an electroanatomical map to guide ablation catheter navigation (CARTO Merge®, Biosense Webster, Inc.). The decapolar circular mapping catheter was positioned in the LA, the PVs, or was navigated around the LOM/VOM to assess the direction of propagation. Activation mapping using the CARTO mapping systems was undertaken to identify the locations of residual breakthroughs in the double-exit of the LOM/VOM (Fig. 2A and B).

Baseline intervals were obtained, which were within normal limits. Ventricular burst pacing revealed *ventriculo-atrial* dissociation. The spontaneous non-sustained AT revealed two distinct atrial activation sequences in the intracardiac recordings. We

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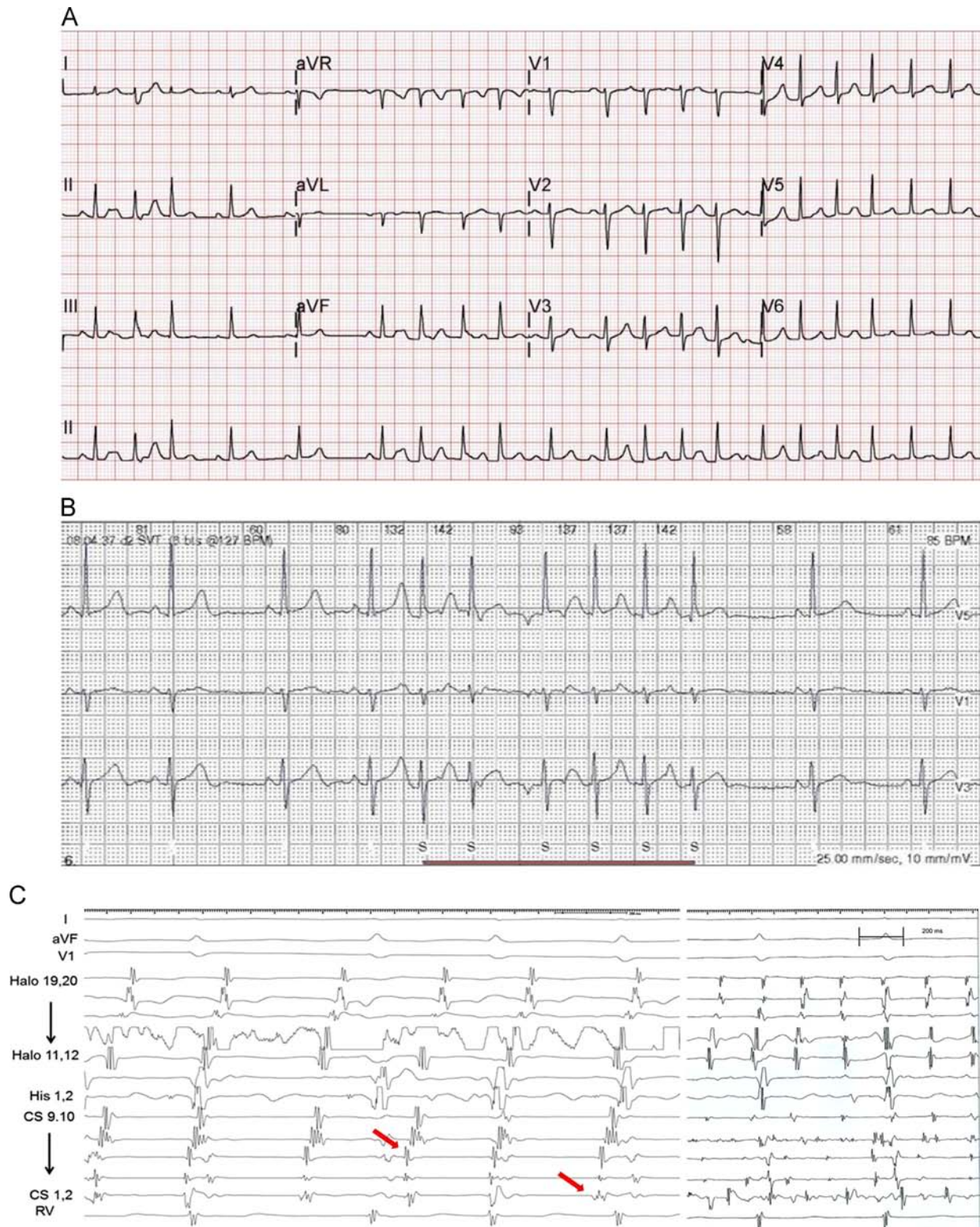


Fig. 1. A. ECG showed an irregular narrow QRS complex tachycardia, which indicated paroxysmal atrial fibrillation. B. Holter monitor representing the irregular narrow QRS complex tachycardia, which indicated paroxysmal atrial fibrillation. C. Intracardiac recording during the organized atrial tachycardia (cycle length: around 350 ms) revealed distinct earliest atrial activation sequences in the CS 1–2 (AT1) and CS 5–6 (AT2) (left panel), which were subsequently induced as atrial fibrillation (right panel).

divided two atrial activation sequences as AT1 and another atrial activation sequence as AT2 (indicated by the arrow in Fig. 1C, left panel). Subsequently, a disorganized atrial activation sequence degenerated AF (Fig. 1C, right panel). Activation mapping located the earliest atrial activation of AT1 at the proximal VOM in the postero-lateral CS at the Halo 5–6 (Fig. 2A). After the first focal RF

application at that site, AT1 was terminated and it became non-inducible; however, repetitive, non-sustained AT2 and activation mapping located the earliest atrial activation of AT2 at the distal LOM/LA ridge adjacent to the left PV (Fig. 2B). After the second focal RF application at that site, AT2 was also terminated and it became non-inducible. Finally, AF also became non-inducible,

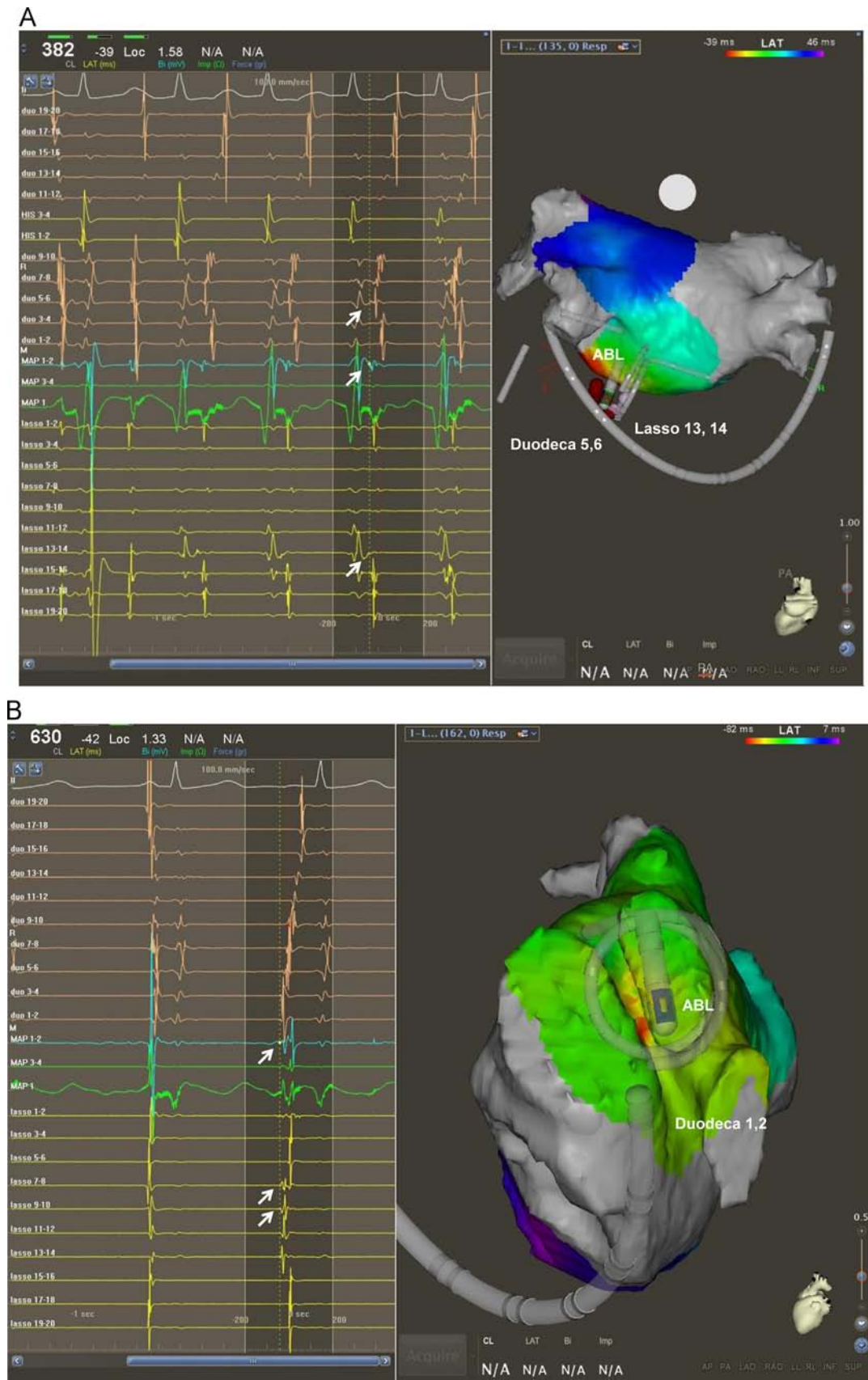


Fig. 2. A. Intracardiac recording representing the earliest atrial activation as AT1 before ablation. B. Intracardiac recording representing the earliest atrial activation as AT2 before ablation.

despite an isoproterenol administration and programmed electrical stimulation. In addition, there has been no recurrence of any tachyarrhythmia without any antiarrhythmic drugs during the 6-month follow-up period.

3. Discussion

AF may be induced by various distinct mechanisms, and the focal triggering AT was also capable of mimicking and inducing AF [3]. However, rare cases have been reported on focal ablation for focal ATs from the LOM/VOM, which mimic and induce AF [1].

A previous study demonstrated that the LOM/VOM was innervated by sympathetic nerve fibers [1,4]. Furthermore, the active Marshall bundle was electrically insulated by fibro-fatty tissues with the most common insertion sites at the proximal CS musculature, close to the origin of the VOM, or the LA ridge superior to the CS; this creates an anatomical and electrical substrate for atrial arrhythmias. In addition, the Marshall bundle may have connections with both CS and the LA ridge and the wave fronts from this site for electrical activation [4]. Rapid electrical interaction between the PV and Marshall bundle has been shown to contribute to inducing AF in a canine model [5]. The endocardial breakout was diffuse and it usually consisted of a broad line of early activity, extending from the PV-LA ridge region to the CS, along the route of the VOM. Previous reports demonstrated that endocardial ablation could interrupt Marshall bundle connections to the CS and terminate ATs [1,4]. This suggested that the focal electrical firing from the PV-LA ridge/LOM/VOM region was critically involved in the induction of AF. Although ectopic firing arises from the myocardial sleeves of the PVs initiating AF, focal ablation may successfully terminate atrial arrhythmia; further, its non-inducibility with program stimulation during isoproterenol infusion showed that it eliminated triggering as the underlying mechanism.

In the case, the anatomy of the proximal VOM was confirmed using three-dimensional computer tomography (3D-CT). In Fig. 2B, the atrial potential in the ablation catheter was discrete, fragmented,

and multiphasic, with > 30 ms duration (Lasso 7-8 and 9-10), which was considered as the local atrial electrogram of the LOM (the distal LOM cannot be identified on 3D-CT), unlike atrial electrograms during sinus rhythm. The unipolar potential in the ablation catheter does not seem to present a QS pattern; however, the ablation signal showed the earliest atrial potential at MAP (*mapping catheter*) 1-2 with an atrial electrogram polarity reversal at MAP (*mapping catheter*) 3-4, which was considered as the optimal ablation site. No atrial ectopic beat occurred after point ablation. Therefore, we emphasize the importance of performing a careful evaluation for triggering mechanisms, especially in young patients to plan ablation for paroxysmal AF. Although PV electrical isolation has become the cornerstone for paroxysmal AF, focal ablation for triggering and inducing AF originating from the PV-LA ridge/LOM/VOM may be effective in eliminating AF in young patients.

Conflict of interest

All authors declare no conflict of interest related to this study.

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