Successful ablation of biatrial tachycardia with preserved electrical activation of left atrial appendage by unidirectional connection via Bachmann’s bundle: A case report

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A B S T R A C T

A 57-year-old man underwent his seventh ablation session for atrial tachycardia (AT). His previous ablations involved several regions of the right atrium (RA) and left atrium (LA). The AT was characterized as biatrial tachycardia with a circuit involving the mitral annulus and septal RA. The AT was terminated by ablation through the insertion site of Bachmann’s bundle (BB) in both atria. After 3 months, the patient underwent his eighth ablation session because of AT recurrence. Activation maps showed that the connection from the RA to LA and vice versa was maintained via BB and the coronary sinus, respectively. The ablation target to interrupt the AT circuit was the mitral isthmus (MI), not BB, because BB supplied the electrical activation of the left atrial appendage (LAA) via a unidirectional electrical connection from the RA to LA. Ablation attempts from within the coronary sinus were performed to target the epicardial connection in the MI and led to complete blockage of the connection from the LA to RA. Otherwise, the connection from the RA to LA was preserved via BB. The patient was free of symptoms and anti-arrhythmic drugs at the 4-month follow-up. However, he had a high risk of electrical isolation of the LAA because extensive ablations had been performed; the strategy of targeting the MI contributed to the balance between preserving the electrical activation of the LAA and treating the biatrial tachycardia. Verification of the connective pathway between the two atria might be helpful to determine the optimal target.

1. Introduction

Biatrial tachycardia (BiAT) is a unique form of macro-reentrant atrial tachycardia (AT) in which the circuit involves both the left atrium (LA) and right atrium (RA) [1,2]. Most previous reports of BiAT [1–3] described Bachmann’s bundle (BB) as a successful ablation site because BiAT has been frequently found to involve BB. However, in patients with a history of extensive ablation including the mitral isthmus (MI) and roof line of the LA, complete blockage of the BB pathway can lead to electrical isolation of the left atrial appendage (LAA) [4]. Electrical isolation of the LAA is associated with a long-term risk of thromboembolism, although it has beneficial effects on maintaining sinus rhythm in patients with persistent atrial fibrillation (AF) [5]. In patients with BiAT involving the mitral annulus (MA), ablation of the MI might theoretically be effective for interrupting the circuit while preserving electrical conduction for the LAA. We herein describe a patient with BiAT who achieved balance between treatment of AT and avoidance of electrical isolation of the LAA.

2. Case report

A 57-year-old man was referred to our institution for catheter ablation of AT. He had previously undergone six ablation sessions: pulmonary vein isolation, LA posterior wall isolation, linear ablation of the anterior LA, linear ablation of the cavotricuspid isthmus, ablation of complex fractionated atrial electrograms in the lower septa of the RA and LA for paroxysmal AF/atrial flutter, and ablation of AT. Although ablation of the MI had also been attempted during the sixth session, bidirectional block could not be achieved. The patient had a history of pacemaker implantation for sick sinus syndrome.

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After obtaining written informed consent from the patient, the seventh ablation session was performed. AT was sustained when initiating the procedure. A 12-lead electrocardiogram during AT, with a tachycardia cycle length (TCL) of 260 ms, exhibited positive P waves in the inferior leads. An intracardiac electrocardiogram showed that the atrial potentials of AT were conducted from the distal to proximal bipolar of the coronary sinus (CS). The activation map of both atria recorded by an electroanatomical mapping system (CARTO 3; Biosense Webster, Irvine, CA, USA) revealed that the wavefront propagated clockwise around the MA, advanced to the RA via the CS, ascended along the septal RA, and returned to the anterosuperior LA, which was the insertion of BB (Video 1). Pacing studies revealed that points at which both manifest entrainment and a <20-ms difference between the post-pacing interval and TCL were obtained were distributed around the MA, proximal CS, septal RA, and anterior LA (Fig. 1A–C, orange tags). Based on these findings, the AT was identified as BiAT with a circuit comprising the MA and septal RA. The TCL of AT was delayed to 330 ms by ablation attempts of 30 W using an open-irrigated contact force-sensing catheter (Navistar ThermoCool SmartTouch; Biosense Webster) from the right atrial insertion site of BB (Fig. 1A, star) and was finally terminated by attempts from the left atrial insertion site of BB (Fig. 1B, star). Recurrence of AT was expected because the activation map of the LA during right atrial appendage (RAA) pacing via the atrial lead of the pacemaker, which was evaluated after the ablation, showed a persistent connection from the RA to LA via BB. The session was terminated because the AT was not induced by burst pacing or programmed stimulation from the CS.

Three months after the ablation session, the patient reported recurrence of palpitation, and his pacemaker log detected recurrence of AT with the same TCL. Therefore, we planned an eighth ablation session in which the connective pathway between the LA and RA was evaluated. The activation map of the LA during RAA pacing showed that the electrical conduction of the LA was initiated from BB (Fig. 2A). The activation map of the RA during LAA pacing via a 20-polar Lasso catheter (Biosense Webster) showed that the electrical conduction of the RA was initiated from the CS (Fig. 2B).

Given that ablation of the MI had been attempted in the previous (sixth) session, it was unclear whether the electrical connection from the RA to LA in the MI was retained. Thus, the target of ablation to interrupt the AT circuit was determined to be the MI, not BB, because BB supplied the electrical activation of the LAA via a unidirectional electrical connection from the RA to LA. Ablation attempts of 20 W from within the CS were performed to target the epicardial connection in the MI during LAA pacing (Fig. 3A and B).

The ablation attempts rapidly led to loss of capture of pacing from the LAA. The stimulations from the LAA only captured the local myocardium of the LAA (Fig. 4A, red circle). The beats were maintained from the RAA irrespective of pacing from the LAA (Fig. 4A, blue circle). Otherwise, the electrocardiogram during RAA pacing showed that the electrical conduction from the RA to LA was retained (Fig. 4B). The above-described findings suggested that electrical isolation of the LAA could be averted because of the unidirectional conduction via BB. AT was not induced by programmed stimulation and burst pacing with isoproterenol infusion from the CS and RA. The patient was free of symptoms and anti-arrhythmic drugs at the 4-month follow-up after the procedure. The pacemaker log detected neither AT nor AF.
3. Discussion

Electrical isolation of the LAA by extensive ablation is reportedly an effective therapeutic option for persistent AF that does not respond to pulmonary vein isolation [4,5]. However, patients who undergo this procedure have a higher risk of thromboembolism [5]. In our case, AF was not detected by the pacemaker log after pulmonary vein isolation despite the fact that the patient was not taking any anti-arrhythmic drugs; hence, the strategy of achieving LAA isolation by ablation to BB might have more risks than benefits. However, Kawamura et al. [3] reported that the electrical connection for the LAA might be preserved via the CS when bidirectional block of BB is achieved. Regardless, we avoided targeting BB because retaining the connection to the LA via the CS was uncertain given that the patient had undergone ablation of the MI in the previous session. Additionally, the activation map of the LA during RAA pacing suggested no conduction via the CS. In cases such as ours, bidirectional block of BB is associated with a risk of extensive isolation of the LAA because of the history of extensive ablations including the MI. Instead, the strategy of targeting the MI might balance preservation of electrical activation of the LAA and interruption of the BiAT circuit. The anatomical isthmus might serve as a reasonable target for ablation of BiAT. Kitamura et al. [1] described two patients with BiAT in whom the circuit involving the tricuspid annulus was treated by ablation of the cavotricuspid isthmus. Consistent with these cases, our patient was also treated by ablation of the MI because the AT circuit in our case involved the MA. To the best of our knowledge, this is the first case in which BiAT was successfully treated by targeting the MI. When ablating BiAT involving the MA in a patient with a history of extensive ablation involving the LA, verification of the connective pathway between the two atria might be helpful to determine the target that balances preservation of the electrical activity of the LAA and treatment for BiAT.

In conclusion, we have herein described a case of successful ablation of BiAT with preserved electrical activation of the LAA by a unidirectional connection via BB. However, our patient had a high risk of electrical isolation of the LAA because extensive ablations had been performed. The strategy of targeting the MI might contribute to the balance between preserving electrical activation of the LAA and successfully treating the BiAT.

Fig. 3. (A) Ablation attempts from within the CS to target the epicardial connection of the mitral isthmus (red tags). (B) Fluoroscopy (left anterior oblique view) during ablation of the epicardial connection of the mitral isthmus (star). The fluoroscopic image also showed the position of the 20-pole electrode catheter monitoring the HRA and CS. The 20-pole Lasso catheter was positioned in the LAA, CS, coronary sinus; IVC, inferior vena cava; HRA, high right atrium; LAA, left atrial appendage; ABL, ablation catheter.

Fig. 4. (A) Intracardiac electrograms during pacing from the LAA after ablation from within the CS. The stimulations only captured the local myocardium of the LAA (red circle). The beats were maintained from RAA pacing via the atrial lead of the pacemaker irrespective of pacing from the LAA (blue circle). (B) Intracardiac electrograms during pacing from the RAA after ablation from within the CS. The stimulations captured both the HRA and LAA. LAA, left atrial appendage; RAA, right atrial appendage; HRA, high right atrium; CS, coronary sinus.
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**Declaration of competing interest**

There are no conflicts of interest to declare.

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