Mechanism of successful cryoballoon ablation of focal atrial tachycardia originating from the left inferior pulmonary vein: a case report

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Abstract

Atrial fibrillation (AF) is mainly initiated by arrhythmogenic triggers originating from the pulmonary veins, and ganglion plexus often plays a crucial role in the induction and maintenance of AF. In this report, we describe a case of successful cryoballoon ablation of focal atrial tachycardia originating from the left inferior pulmonary, in which vagal response was observed, and discuss its tachycardia mechanism.

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ABSTRACT

Atrial fibrillation (AF) is mainly initiated by arrhythmogenic triggers originating from the pulmonary veins, and ganglion plexus often plays a crucial role in the induction and maintenance of AF. In this report, we describe a case of successful cryoballoon ablation of focal atrial tachycardia originating from the left inferior pulmonary, in which vagal response was observed, and discuss its tachycardia mechanism.

CASE REPORTS

Case presentation

A 40 year-old woman who had undergone a left mastectomy and breast reconstruction for breast cancer was admitted to our hospital because of palpitations and dyspnea on exertion. A 12-lead electrocardiogram showed atrial tachycardia (AT) with a positive notched P wave in leads I, II, III, aV_F, V1, and V2 (Figure 1A).

A Holter electrogram revealed incessant AT and transient atrial fibrillation (AF). A thoracic echocardiogram indicated reduced contractility, with an ejection fraction (EF) of 35%. A radiofrequency ablation for AT was performed due to severe discomfort caused by palpitations after obtaining informed consent. AT with a constant atrial activation sequence constantly appeared, and the cycle length during tachycardia varied between 260 and 310 ms (Figure 1B). Activation mapping of the AT was performed in the left atrium (LA) using a three-dimensional mapping system (NavX, St. Jude Medical, St. Paul, Minnesota, USA). The earliest AT activation site was observed at the bottom of the left inferior pulmonary vein (LIPV) ostium (Figures 2A and 2B). Because AF and AT were identified prior to the procedure, we attempted PV isolation using a 28-mm cryoballoon (CB) (Arctic Front, Medtronic, Minneapolis, Minnesota, USA). First, CB ablation of the left superior PV (LSPV) orifice and antrum resulted in the termination of AT 51 seconds after the temperature measured at the balloon's base dropped below freezing (Figures 2C and 3A). Thereafter, a single atrial extrastimulus with the same atrial activation sequence was observed; however, AT was no longer induced. Subsequently, after the CB ablation of the LIPV (Figure 3A), the atrial extrastimulus also disappeared. In this case, CB ablation of the LSPV resulted in termination and no induction of AT originating from the bottom of the LIPV ostium. Hence, this study aimed to identify the mechanism of AT.

Discussion

AF is primarily initiated by arrhythmogenic triggers originating from the PV. PV isolation is the cornerstone of AF ablation, and PVI with CB ablation has been proven to be non-inferior to radiofrequency ablation with regard to safety and clinical outcomes.[1] In this case, the AT was a focal tachycardia originating from the LIPV ostium. Kistler et al. demonstrated that in the vast majority of AT originating from the PVs, the tachycardia focus originated from the ostium of the PV rather than from further distal PVs, in contrast to AF.[2] Wei et al. showed that CB ablation was an effective and safe tool to treat AT originating from the PVs.[3] In this case, CB ablation of the LSPV resulted in termination and no induction of the AT originating from the bottom of the LIPV ostium. We hypothesized that the mechanism of AT initiation and maintenance might be related to ganglion plexus (GP) hyperactivity near the vicinity of the PV-LA junction.

Previous studies have shown that the intrinsic cardiac autonomic nervous system (ANS), an epicardial neural system composed of GPs and a complex network of interconnecting neurons, plays a crucial role in the induction and maintenance of AF through both sympathetic and parasympathetic nervous system stimulation.[4] The GPs act as an integration and interconnection system between the extrinsic cardiac ANS nerves, originating from the central ANS and reaching the heart through the mediastinum, and the rest of the intrinsic cardiac ANS. The function of GPs is not only to modulate the autonomic interplay between the extrinsic and intrinsic cardiac ANS, but also to independently regulate cardiac electrical and mechanical functions through the transduction of local signals. In LA, the major GPs are located close to the PV-LA junction. GPs contain both sympathetic and parasympathetic components. GPs activation creates intracellular calcium overload and shortening of action potential duration, which in turn results in early after depolarization and triggers firing in surrounding atrial tissue or neighboring PVs, thereby causing initiation and maintenance of AF.[4] Vagal reactions manifesting as bradycardia and hypotension have been reported as markers of GPs modification during PVI.[5] Vagal reactions during CB-based PVI were reported to be frequent from 36 % to 50 %. PVI with the 28-mm CB catheter is associated with a broad antral ablation area that extends from the PV ostium toward the LA, thus increasing the possibility of concomitant and extended GP ablation.[6]Yorgun et al. demonstrated that the patients with vagal response during CB ablation had a lower incidence of AF recurrence, suggesting modulation of the cardiac ANS through GP modification.[5] Other studies have suggested that adding GP ablation to PV isolation may achieve better clinical outcomes in patients with AF.[7] These findings indicate that both larger areas of transmural lesion formation and GP modification may contribute to the efficacy of CB ablation. In this case, one minute after CB ablation of the LSPV was completed, transient sinus arrest and atrioventricular block due to the vagal response were also observed (Figure 3B). Histological examination of the human heart has demonstrated that the Marshall tract GP is located within the fat pad anterior to the LSPV and LIPV, and the superior left GP is located on the roof of the LA, medial to the LSPV.[4] The interruption of axons from these hyperactive GP to the LIPV by CB ablation of the LSPV may have contributed to eliminating the initiation and maintenance of

this AT.

The present case highlighted termination and no induction of AT originating from the LIPV and the vagal response was observed with CB ablation of the LSPV. It could also be deduced that the mechanism of the AT strongly involved the GPs hyperactivity.

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FIGURE LEGENDS

Figure 1

Twelve-lead electrocardiogram during the atrial tachycardia of 105 bpm with a positive notched P wave in leads I, II, III, aV_F, V1, and V2.

Intracardiac electrograms during the atrial tachycardia with an unstable cycle length of 260 to 310 ms.

I, aV_F, V1 = surface electrogram; CS = coronary sinus; RA = right atrium. The arrow marker indicates the initiation of atrial tachycardia.**Figure 2**

Intracardiac electrograms of atrial tachycardia with catheter placement in the left inferior pulmonary vein.

Activation map in left atrium during atrial tachycardia.

The earliest activation site of atrial tachycardia was observed at the bottom of the left inferior pulmonary vein ostium.

Cryoballoon ablation of the left superior pulmonary vein orifice and antrum resulted in termination of atrial tachycardia.

AT = atrial tachycardia; LAO = left anterior oblique view; LIPV = left inferior pulmonary vein; LSPV = left superior pulmonary vein; PA = postero-anterior view. The other abbreviations are as in Figure 1. The

arrow marker indicates the earliest activation site of atrial tachycardia.**Figure 3**

Left panel: Fluoroscopic image of a circular mapping catheter placed at the left inferior pulmonary vein ostium at the earliest activation site of atrial tachycardia. Middle panel: Fluoroscopic image during cryoballoon ablation of the left superior pulmonary vein. Right panel: Fluoroscopic image during cryoballoon ablation of the left inferior pulmonary vein.

The dotted line corresponds to the bottom of the left inferior pulmonary vein.

Vagal response observed after the completion of cryoballoon ablation of the left superior pulmonary vein. Transient atrioventricular block was observed during left atrial appendage pacing due to the appearance of sinus arrest.

The other abbreviations are as in Figure 1.