Case Reports

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Successful radiofrequency ablation of swallowing-induced atrial tachycardia arising from left superior ganglionated plexus

Jie Yin[1,*], Ye Wang[1,*], Xiaolu Li[2], Mei Xue[1], Wenjuan Cheng[1], Xinran Li[1], Yugen Shi[1], Yu Wang[1], Hangji Lu[3], Hesheng Hu[1,*] and Suhua Yan[1,*]

Abstract

A man in his early 40s developed palpitations brought on by swallowing and was found to have short runs of atrial tachycardia induced by swallowing solid food. Atrial tachycardia during swallowing was documented on electrocardiography and 24-hour Holter monitoring. No structural heart disease or esophageal disorders were found by echocardiography. The patient then underwent an electrophysiological study and catheter ablation. We mapped the left atrium with a multipolar mapping catheter while the patient swallowed bread and found that the earliest endocardial breakthrough was on the left anterior superior atrium, where the left superior ganglionated plexus was located. We successfully eliminated the paroxysmal atrial tachycardia at this site. Interestingly, in the process of ablation, atrioventricular node reentrant tachycardia was triggered. After the slow-pathway ablation procedure, no further tachycardia was induced.

Keywords

Swallowing-induced atrial tachycardia, ablation, ganglionated plexus, electrocardiography electrophysiological study, case report

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[1]Department of Cardiology, The First Affiliated Hospital of Shandong First Medical University, Jinan, China
[2]Department of Emergency, The First Affiliated Hospital of Shandong First Medical University, Jinan, China
[3]Shandong First Medical University, Jinan, China

*These authors contributed equally to this work.

Corresponding author:
Hesheng Hu, Department of Cardiology, The First Affiliated Hospital of Shandong First Medical University, No. 16766 Jingshi Road, Lixia District, Jinan, Shandong Province 250014, China.
Email: hyperhhs@163.com

Introduction

Swallowing-induced atrial tachycardia (SIAT) is a rare disease occurring in about 0.6% of patients with paroxysmal atrial arrhythmias.[1] SIAT is generally assumed to be caused by abnormal autonomic nervous reflexes. However, the exact underlying mechanism is controversial. The effectiveness of radiofrequency catheter ablation (RFCA) has recently been confirmed. We herein report a case of atrial tachycardia in a patient with palpitations induced by swallowing. RFCA of the left superior ganglionated plexus (GP) was performed with no recurrence of symptoms.

Case report

A man in his early 40s presented to the cardiovascular department of our hospital with a 14-day history of palpitations induced by swallowing of solid food. Dry swallowing itself did not precipitate any symptoms or changes. The palpitations consisted of multiple episodes of tachycardia lasting for less than 1 minute before spontaneously converting back to sinus rhythm. The P wave on the surface electrocardiogram was upright in leads II, III, AVF, and V1 during atrial premature contractions (Figure 1(a), (b)). Heart rate variability frequency domain analysis showed fewer low-frequency (LF) components and a lower LF/high-frequency (HF) ratio during swallowing-related tachycardia, suggesting that the vagal nerve reflex was closely related to the occurrence of SIAT (Figure 1(c), (d)). Because of the severe discomfort of the palpitations, the patient consented to catheter ablation. A diagnostic electrophysiological examination was then performed. Intravenous isoprenaline administration plus programmed S1S1 stimulation did not provoke atrial tachycardia, which further confirmed our speculation that the tachycardia was due to the activation of the parasympathetic nervous system. Swallowing bread triggered a cluster of atrial tachycardia. Therefore, following the swallowing procedure, we created an activation map of the left atrium using a PentaRay® NAV catheter with a three-dimensional mapping system (CARTO® 3 system; Biosense Webster Inc., Irvine, CA, USA). The earliest activation site of atrial premature contractions was the left anterior superior atrium, where the left superior GP was located (Figures 2 and 3). We performed RFCA at that site (maximum of 30 W). The ablation initiated narrow QRS tachycardia. Following the procedure, the patient underwent slow-pathway ablation for slow/fast atrioventricular nodal reentrant tachycardia (AVNRT) (Figure 4). Eventually, we confirmed that there was no further induction of paroxysmal atrial tachycardia by swallowing solid food, and the session was ended. Subsequent follow-ups remained uneventful.

Discussion

SIAT is an underrecognized cause of paroxysmal supraventricular tachycardia. Cases of SIAT have been rarely described in the literature, and most reported cases had a right pulmonary vein and right atrial origin.[2–4] To the best of our knowledge, this is the first report of SIAT originating from the left superior GP. The exact mechanism of SIAT is unclear. Direct stimulation of the left atrium by the passage of food contents or contraction of the esophagus has been hypothesized as a cause. This hypothesis is strengthened when atrial tachycardia can be elicited by inflation/deflation of a balloon in the esophagus. However, it does not explain why the earliest ablation site of paroxysmal atrial tachycardia in most reported cases, including ours, was not adjacent to the esophagus. Other recent reports have suggested a neural reflex as the cause of SIAT. Some

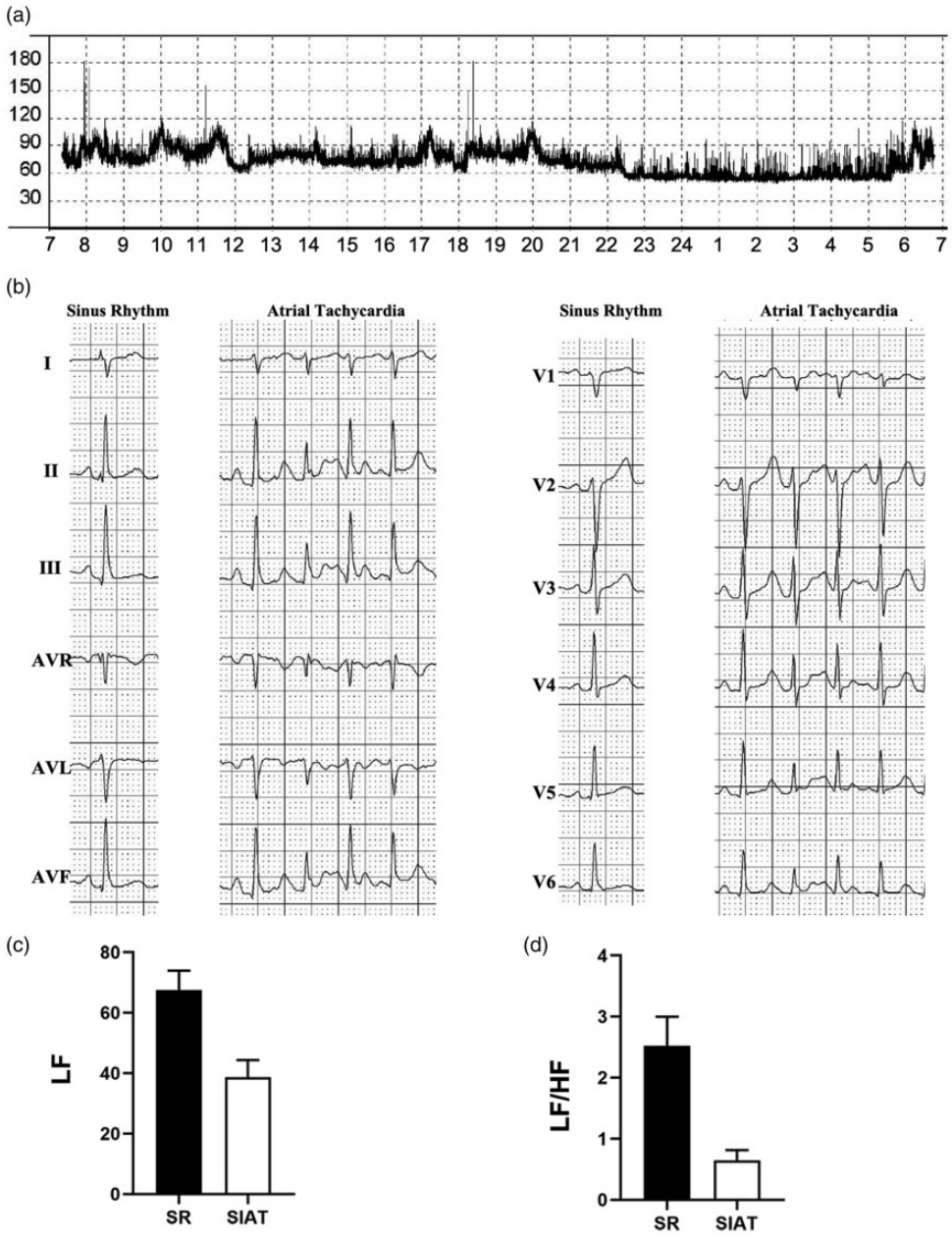

Figure 1. (a) Holter electrocardiogram recorded an increase in SIAT during dinner, with each episode lasting a few seconds. (b) Twelve-lead electrocardiogram showed SR (left) and SIAT (right). (c, d) Few LF components and a lower LF/HF ratio were noted during SR and SIAT.
SIAT, swallowing-induced atrial tachycardia; LF, low-frequency; HF, high-frequency; SR, sinus rhythm.

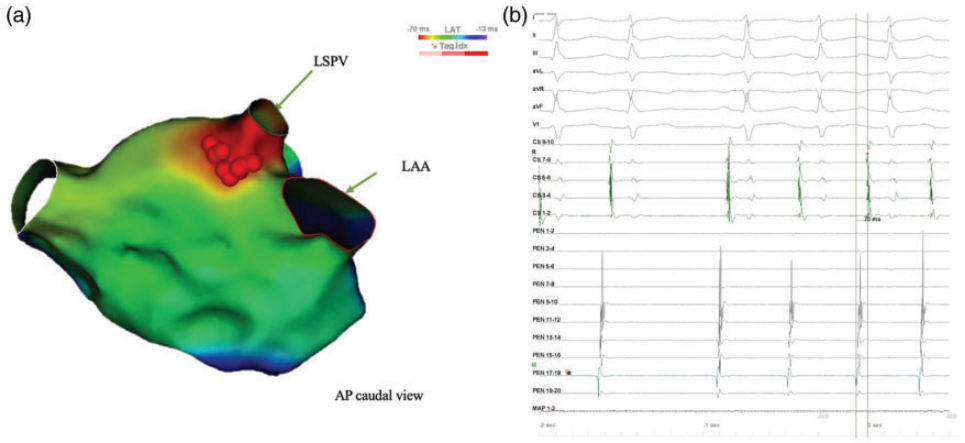

Figure 2. (a) Earliest activation site of atrial premature contractions recorded from the PentaRay® NAV catheter preceded the reference CS 7,8 by 70 ms. (b) The earliest activation site of atrial premature contractions recorded from the ablation catheter preceded the reference CS 7,8 by 96 ms. (c) Cardiac image from computed tomography synchronized with the image from the contact mapping system. The red circle shows the ablation targets.
CS, coronary sinus; LSPV, left superior pulmonary vein; LAA, left atrial appendage; AP, anteroposterior.

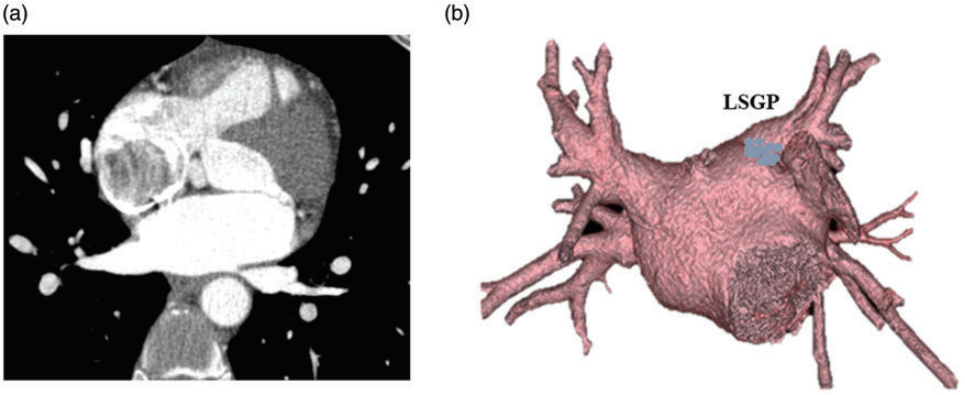

Figure 3. (a) Anatomic relationship of the pulmonary vein and esophagus. (b) Anatomic location of the LSGP, which is located in the superolateral area around the root of the left superior pulmonary vein
LSGP, left superior ganglionated plexus.

authors suggested sympathetic activation because activating adrenergic reflexes originating in the esophagus can result in asynchronous atrial depolarization and trigger atrial activity through delayed depolarizations, leading to focal reentry and atrial arrhythmias.[5] However, the most likely possibility is a vagally mediated neural reflex involving a neurotransmitter other than acetylcholine because atropine and bethanechol do not affect the onset of SIAT.

In our case, we speculate that extrasystoles were provoked by the vagal neural reflex, not by direct contact from the esophagus, because the ablation site was on the

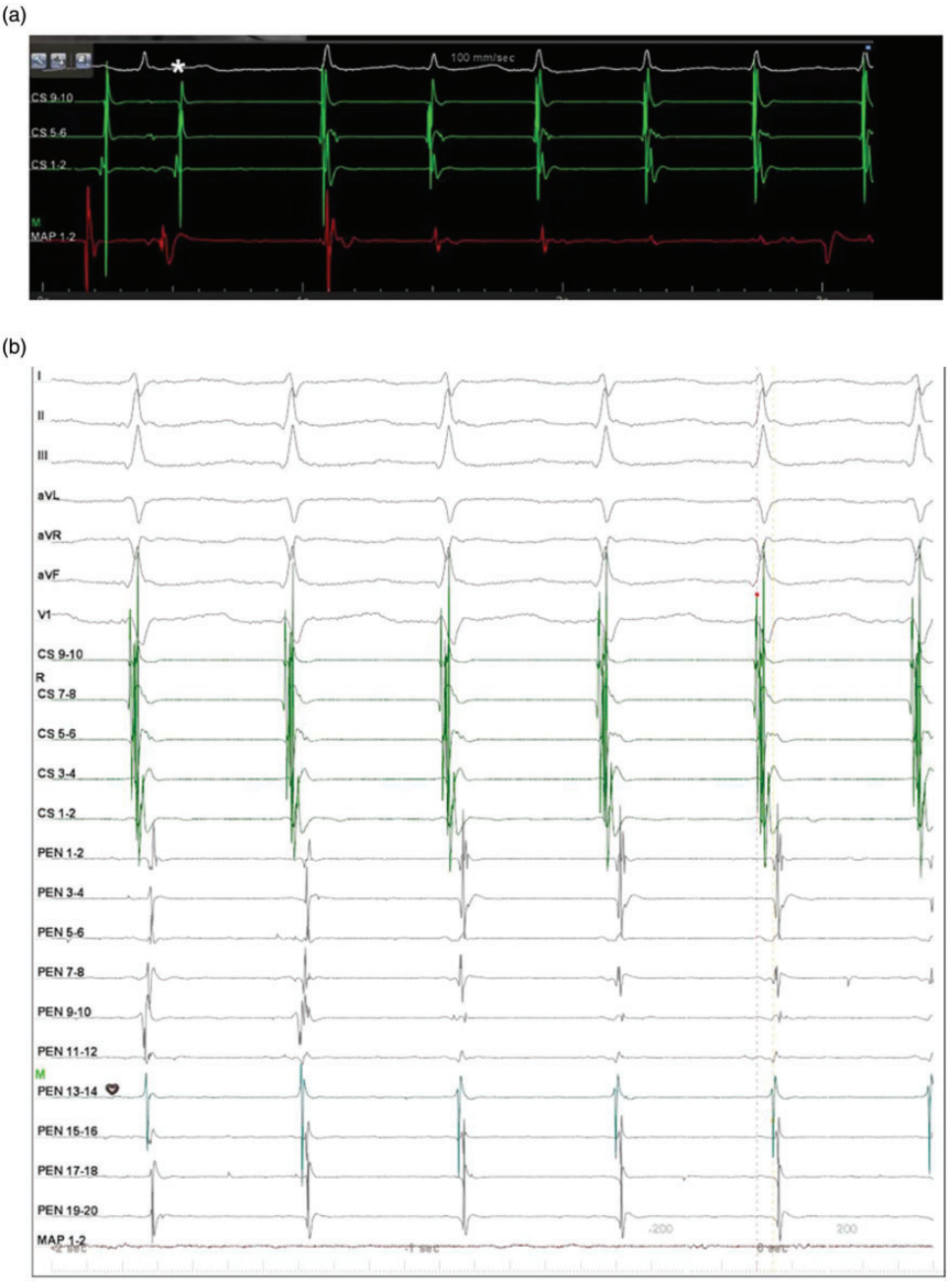

Figure 4. (a) Intracardiac electrogram showed that atrial premature contractions initiated atrioventricular node reentrant tachycardia. (b) Intracardiac electrograph also showed antegrade conduction by the slow pathway and retrograde conduction by the fast pathway.

left superior GP near the ligament of Marshall, where parasympathetic innervations were clustered.[6] Activation of the GP by a mechanical stimulus to the posterior wall of the left atrium may have acted as the trigger. GP ablation is used to treat atrial fibrillation and vasovagal syncope.[7,8] We did not apply HF stimulation to identify the GP site before ablation based on a report by Pokushalov et al.,[9] who showed that because of the similar locations of GPs in the majority of patients, an anatomical ablation performed empirically (i.e., at presumed GP sites) was associated with similar or even better results than ablations driven by GP localization using HF stimulation. In our patient, it is possible that the ablation lesion was not deep enough to affect the adjacent GP, thereby eliminating the "exit" of SIAT. These points are potential limitations in the present case.

Interestingly, the extrasystoles provoked AVNRT during the ablation procedure. After the slow-pathway ablation procedure, no further tachycardia was induced. Whether the occurrence of AVNRT was a vagally mediated neural reflex remains unclear. Parasympathetic innervation distributes to the slow AV nodal pathway. Moreover, enhanced vagal tone is conducive to induction of AVNRT with a premature atrial complex.[10] Therefore, the concomitant AVNRT might have been associated with the GP as well as the preexisting arrhythmic substrate. This case provides evidence that RFCA of the GP or a site close to this plexus is effective in patients with SIAT or swallowing-induced AVNRT.

Conclusion

We have herein reported a case of SIAT arising from the left superior atrial GP that was permanently cured by catheter ablation. Anatomically guided endocardial catheter ablation of a GP may be an effective technique in patients with SIAT.

The authors state that written informed consent was obtained from this patient for publication of his case history and associated images in line with the COPE recommendations. The reporting of this study conforms to CARE guidelines.[11] All procedures were conducted according to the approved protocols and guidelines established by the ethics committee of Shandong First Medical University.

Declaration of conflicting interest

The authors declare that there is no conflict of interest.

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ORCID iD

Suhua Yan https://orcid.org/0000-0002-7746-6787

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