Cardiac vagus nerve denervation by pulmonary vein isolation was effective for swallowing-induced atrial tachycardia

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Abstract
Swallowing-induced atrial tachycardia (SIAT) is a relatively rare arrhythmia. A 56-year-old woman was admitted to treat atrial tachycardia that occurs by not only eating and drinking but also yawning. Both the right and left upper pulmonary veins were suspected as the earliest activation site of the tachycardia and the abnormal activation of ectopies themselves were suppressed after pulmonary vein isolation (PVI). In a 24-hour Holter electrocardiogram, the HF component of the analysis of heart rate variability was suppressed both at 1 day and at 2 years after ablation. In this case, cardiac vagal nerve denervation by PVI was effective for SIAT.

KEYWORDS
cardiac vagus nerve denervation, pulmonary vein isolation, Swallowing-induced atrial tachycardia

1 | INTRODUCTION

Swallowing-induced atrial tachycardia (SIAT) is reported to be caused by mechanical and autonomic nerve activities, such as swallowing of solid food, liquids, or both (Morady et al., 1987), and it can be effectively treated by catheter ablation at the earliest activation site or pulmonary vein isolation (PVI) (Tada et al., 2007). PVI may modify vagal innervation at PV ostium (Qin et al., 2016), and PVI could also directly affect vagal fibers on the surface of esophagus, leading to vagal nerve denervation. Here, we report an unusual case of SIAT that was suppressed by cardiac vagal nerve denervation through PVI.

2 | CASE REPORT

A 56-year-old woman without a history of pharyngoesophageal disease was admitted to our hospital for the evaluation and treatment of palpitation and dizziness that occur while eating. Since then, she has experienced similar episodes when drinking and eating food, particularly bread. Her family doctor performed Holter electrocardiogram (ECG), which revealed frequent paroxysmal atrial tachycardia. The patient experienced palpitation and dizziness during the recording. Twelve-lead ECG conducted while the patient was eating bread demonstrated paroxysmal atrial tachycardia.

An electrophysiological study using a three-dimensional mapping system (EnSite NavX®) was attempted while the patient swallowed food and drinks. First, the patient swallowed bread after placing ring catheters in the right superior pulmonary vein (RSPV), LSPV, and the superior vena cava. Thereafter, the patient had an episode of paroxysmal atrial tachycardia, and the earliest activated site was identified to be the RSPV (Figure 1). Second, the patient swallowed Gastrografin after placing a ring catheter in the RSPV, LSPV, and roof of the left atrium. Then, she presented an episode of tachycardia, and the earliest activated site was found to be the LSPV (Figure 2). In addition, when the patient yawned, transient bradycardia occurred, and she had an episode of atrial tachycardia at the RSPV. Based on these results, PVI was performed. After all these ablation procedures, we attempted to perform the same swallowing test (bread, Gastrografin, and yawning) again while the patient was...
FIGURE 1 To evaluate the origin of the SIAT, ring catheters were positioned in the right and left superior pulmonary veins and the superior vena cava (Panel a). During atrial tachycardia occurred after swallowing bread, intracardiac electrocardiogram (Panel b) and 3D mapping system (EnSite NavX®) (Panel c) demonstrated that the earliest atrial activation was observed in the right superior pulmonary vein.
awake and failed to induce the ectopic rhythm. There were no gastrointestinal symptoms, and abdominal radiography did not reveal gastric distention after ablation.

We conducted 24-hour Holter ECG before, 1 day, and 2 years after the catheter ablation, and the power spectral analysis of heart rate variability was performed for 10 min every hour using the fast Fourier transform algorithm, and the low-frequency (LF: 0.04–0.15 Hz) and high-frequency (HF: 0.15–0.4 Hz) spectral components were calculated. Compared with before ablation, the LF/HF ratio did not differ; however, the HF component was consistently suppressed both at 1 day and at 2 years after ablation (Figure 3). In addition, the total heart rate was increased after the ablation. The patient never experienced palpitation during outpatient visit.

3 | DISCUSSION

The mechanism of SIAT remains controversial. Direct mechanical interaction between the distended esophagus and the adjacent left atrium was suggested as one possible mechanism of SIAT (Lindsay, 1973). Others have suggested that a vagal nerve-mediated neural reflex may play a role in initiating SIAT (Higuchi et al., 2016). Swallowing can stimulate the efferent cardiac branches of the vagal nerve via the solitary nucleus in the medulla oblongata from the afferent vagal nerve through the increased sensitivity of mechanoreceptors in the esophagus. The efferent cardiac branch shortens the relative atrial refractory period and induces abnormal atrial excitation (Chock & Gill, 2002). Extrasystole arising from pulmonary vein or superior vena cava was reported to trigger SIAT (Higuchi et al., 2016; Onishi et al., 2016), and abnormal atrial ectopy in RSPV and LSPV triggered SIAT in this case. Vagal nerve reflex could be responsible for the arrhythmia in our case based on the following reasons. First, tachycardia was induced by not only eating foods and drinking but also yawning. Second, both RSPV and LSPV in which the triggering extrasystole arose in this case were not adjacent to the esophagus. Finally, sinus bradycardia was observed just before the occurrence of tachycardia induced by yawning, which suggested that an increase in the vagal nerve activity could trigger SIAT in this case.
The analysis of power spectral analysis of heart rate variability in this patient showed that the HF power had decreased and that the total heartbeats were increased after ablation, and this tendency was continued for at least 2 years. These results suggested that cardiac vagal nerve denervation through PVI would suppress SIAT in this case. The ganglionated plexi are located around the pulmonary vein and are parts of the intrinsic cardiac autonomic nervous system. PVI may modify vagal innervation at PV ostium (Qin et al., 2016), and PVI could also directly affect vagal fibers on surface of esophagus, leading to vagal nerve denervation.

4 | CONCLUSION

We report a rare case of SIAT induced by not only eating and drinking but also yawning. The origins of this tachycardia were multiple, and the occurrence of transient bradycardia just before the onset of the tachycardia indicated that increased vagal nerve activity can trigger the tachycardia. Cardiac vagal nerve denervation by PVI could play an important role for suppression of the SIAT in this patient.

ETHICS STATEMENT

The authors have obtained the patient’s informed consent.

CONFLICT OF INTEREST

None.

DATA AVAILABILITY STATEMENT

Data openly available in a public repository that issues datasets with DOIs.

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