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【 CASE REPORT 】

Swallowing-induced Paroxysmal Atrial Tachycardia Causes Weight Loss and Fainting During Mealtime: A Case Report and Literature Review

Tsugumi Hosoe, Kenzo Uzu, Kiyohiro Hyogo, Ryosuke Takahashi, Sho Tamai, Tomoyuki Nagano, Yasushi Shimokawa, Soichiro Ota, Hideaki Okubo and Hiroki Shimizu

Abstract:
Swallow or deglutition syncope is an unusual disorder. We herein report an 80-year-old man with paroxysmal atrial tachycardia induced by swallowing, causing syncope. Initially, we suspected a digestive disorder and found no significant findings. Finally, a swallowing test with monitoring of the heart rate and blood pressure helped in the diagnosis. The patient was treated with antiarrhythmic drugs and catheter ablation. The mechanism underlying swallowing-induced tachycardia presumably involves mechanical stimulation of the esophagus and autonomic nervous system effects. However, few cases have been reported, and the exact mechanism remains unclear.

Key words: swallowing-induced tachycardia, paroxysmal atrial tachycardia, syncope, weight loss

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Introduction

Swallowing-induced atrial tachyarrhythmias are rare and are defined as a reproducible occurrence of supraventricular tachyarrhythmias, such as premature atrial contractions, paroxysmal atrial tachycardia, and paroxysmal atrial fibrillation, during swallowing. They occur in approximately 0.6% of patients with paroxysmal atrial arrhythmias (1).

We herein report a rare case of a patient presenting with an eating disorder, lightheadedness, and weight loss who was diagnosed by a swallowing test with swallowing-induced paroxysmal atrial tachycardia and later experienced syncope. All symptoms could be traced to swallowing-induced atrial tachyarrhythmia. He was able to eat, and his weight loss was ameliorated following treatment with antiarrhythmic drugs and radiofrequency catheter ablation therapy.

Case Report

An 80-year-old man presented to the hospital with a chief complaint of fainting during meal times. He began to have dysphagia during meals seven days prior to his visit, accompanied by fainting. He visited his previous doctor because of anorexia and weight loss of 5 kg in a single week. As he had a history of transverse colon cancer surgery six years earlier, a detailed examination of his digestive tract was necessary, and he was referred to our hospital.

On an initial assessment, his vital signs were stable, and no abnormalities were noted in his physical examination, except for yellowing of the conjunctiva of the eyes due to physiological jaundice, which had been noted previously. Laboratory results of blood tests showed elevated bilirubin levels, a decreased renal function, dyslipidemia, and elevated brain natriuretic peptide levels (Table). Chest radiography was normal, and computed tomography (CT) of the chest and abdomen showed no findings suggestive of malignant disease.

Since the patient had a history of vasospastic angina, he underwent myocardial deviated enzyme measurement, an electrocardiogram (ECG), and transthoracic echocardiography, but no significant abnormal findings were found. The ECG showed sinus rhythms with negative T waves in the III and aVf leads (Fig. 1). After admission to the hospital, the

Department of Cardiology, Konan Medical Center, Japan
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Correspondence to Dr. Kenzo Uzu, kenzouzu@gmail.com

patient underwent a videoendoscopic evaluation of swallowing, upper gastrointestinal endoscopy, and contrast-enhanced thoraco-abdominal CT to examine the gastrointestinal tract further; however, no significant abnormalities were observed in the pharynx or esophagus. Thus, we found there to be no structural or functional abnormality of the gastrointestinal tract that could cause weight loss and anorexia.

We decided to scrutinize the syncope further, so we took a detailed history of the fainting spells. Symptoms appeared within 1 min of swallowing and lasted 2-3 s, and fainting occurred even when swallowing saliva. After awakening, there was no confusion in consciousness. Since there were scattered supraventricular arrhythmias on monitoring during hospitalization, we decided to record the pulse with a 24-h Holter ECG to understand the relationship between the patient's lightheadedness and tachycardia in more detail. Frequent paroxysmal atrial tachycardia (PAT) occurred during mealtime, and the occurrence of PAT coincided with lightheadedness during meals (Fig. 2). Therefore, a swallowing test was performed to investigate vital signs and ECG changes during swallowing. After ingesting carbonated water, PAT appeared within 1 min of swallowing, the pulse rate increased from 94 bpm to 180 bpm, and the blood pressure decreased from 123/80 mmHg to 80/68 mmHg (Video). The patient complained of pre-syncope symptoms, such as lightheadedness and a floating sensation during the blood pressure drop. The same tachycardia and pre-syncope symptoms were observed when cold water or solid food was ingested. Therefore, we hypothesized that swallowing would induce PAT, which would decrease blood pressure and cause syncope in this elderly and sensitive patient.

After 50 mg of intravenous pilsicainide, the patient was able to ingest cold water and jelly without experiencing PAT. From the next day, 75 mg of pilsicainide was administered orally daily. Because of the efficacy of oral pilsicainide and the polymorphic P wave during PAT on the surface ECG, drug therapy was chosen over catheter ablation. However, regardless of swallowing, spontaneous regular tachycardia with a narrow QRS occurred frequently, and a 12-lead ECG showed a pseudo-s pattern in the lower limb leads and an rSr' pattern in the V1 lead, indicating that the mechanism of this tachycardia was atrioventricular nodal reentry tachycardia (AVNRT) (Fig. 3). In contrast to PAT, AVNRT appeared at rest, regardless of swallowing, and caused severe symptoms of palpitations and pre-syncope. In addition, catheter ablation of the right atrium only was considered curative. Therefore, an electrophysiological study and radiofrequency catheter ablation were conducted the day

Table. Laboratory Data on Admission.

Total protein (g/dL)	7.5	Chloride (mEq/L)	108
Albumin (g/dL)	4.5	Calcium (mEq/L)	9.6
TB (mg/mL)	2.2	LDL-C (mg/dL)	152
DB (mg/mL)	0.6	HDL-C (mg/dL)	74
AST (U/L)	33	TG (mg/dL)	86
ALT (U/L)	25	CRP (mg/dL)	0.33
ALP (U/L)	60	Glucose(mg/dL)	150
γGT(U/L)	17	BNP (pg/mL)	222.1
LDH (U/L)	224	NT-proBNP (pg/mL)	1110
CK (U/L)	627	WBC (/uL)	6,290
CK-MB(ng/mL)	9.5	RBC (/uL)	4.5×10^6
BUN (mg/dL)	49.8	Hb (g/dL)	14.6
Creatinine (mg/dL)	1.33	Platelet (/uL)	174×10^3
eGFR(mL/min/1.73m^2)	40.4	Trop I(ng/mL)	0.012
Sodium(mEq/L)	146	TSH (uIU/mL)	1.036
Potassium(mEq/L)	3.3	fT4 (ng/dL)	1.3

TB: total bilirubin, DB: direct bilirubin, AST: aspartate transaminase, ALT: alanine transaminase, ALP: alkaline phosphatase, γGT : γ-glutamyl trans peptidase, LDH: lactate dehydrogenase, CK: creatine kinase, BUN: blood urea nitrogen, eGFR: estimated glomerular filtration LDL-C: low density lipoprotein cholesterol, HDL-C: high density lipoprotein cholesterol, TG: triglyceride, CRP: C-reactive protein, BNP: brain natriuretic peptide, WBC: white Blood Cell, RBC: red blood cell, Hb: hemoglobin, Trop I: troponin I, TSH: thyroid-stimulating Hormone, fT4: free total thyroxine

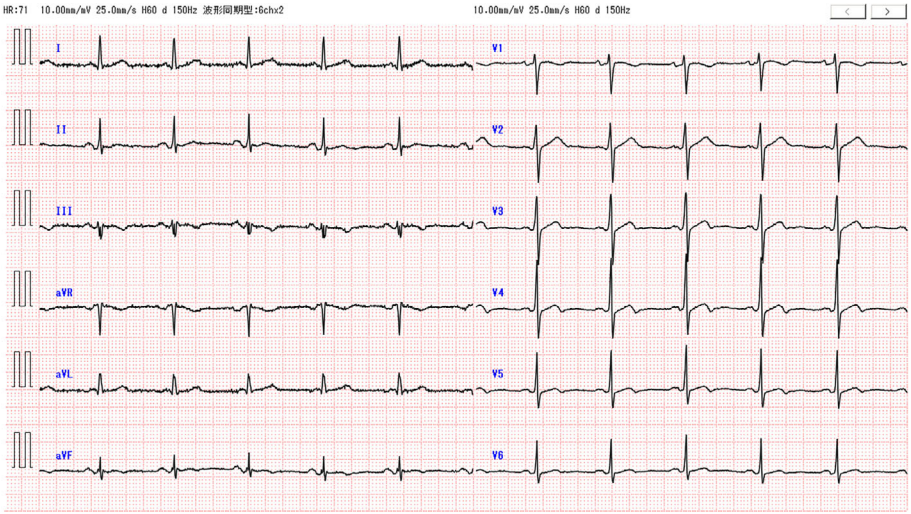

Figure 1. Electrocardiogram data on admission showing sinus rhythms with negative T wave in the III and aVf leads.

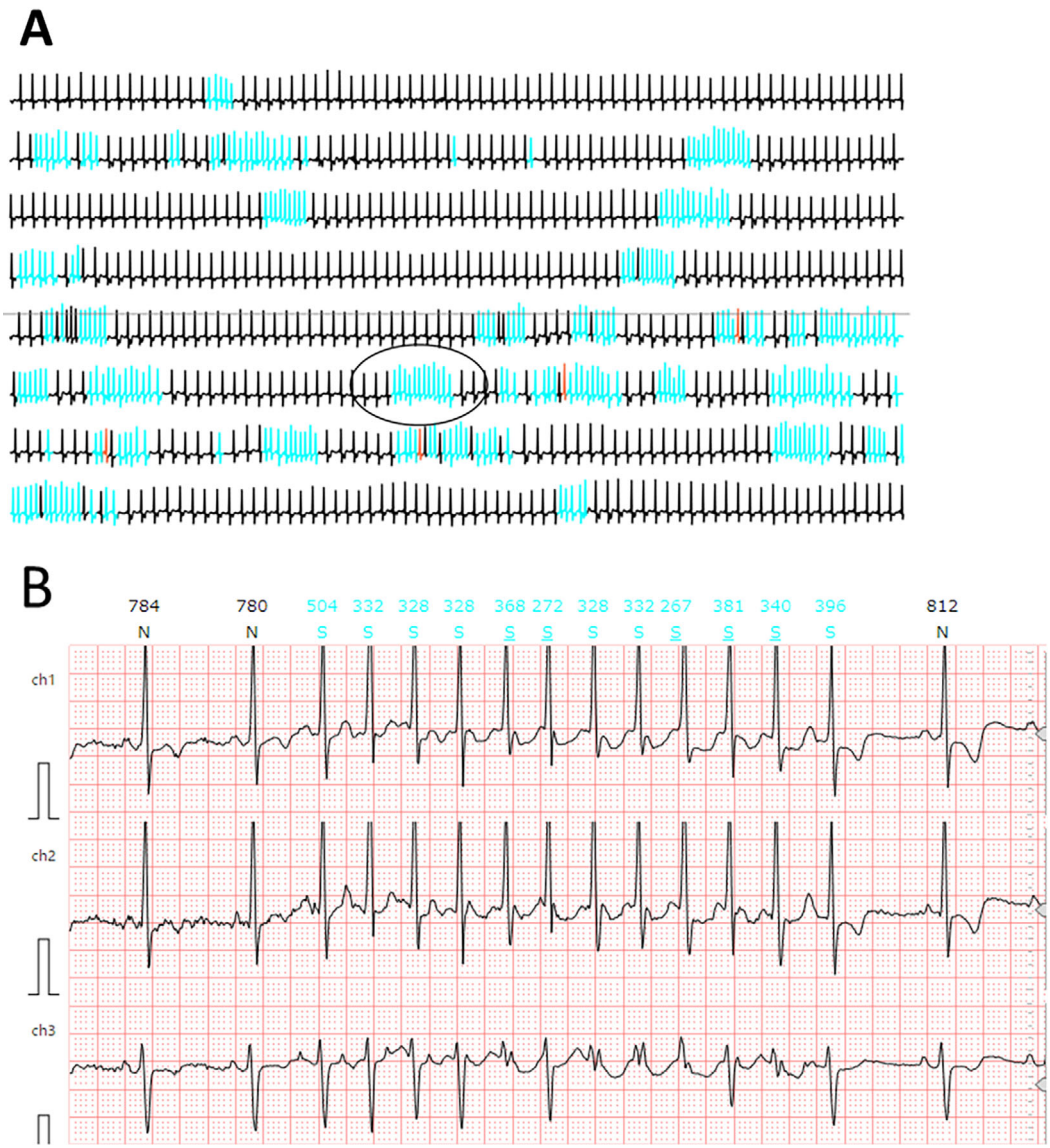

Figure 2. **A: A 24-h Holter electrocardiogram (ECG) showing tachycardia during mealtime. The gray area on the ECG indicates tachycardia. B: Magnification of the circled area showing tachycardia due to paroxysmal atrial tachycardia.**

after the initiation of oral pilsicainide. Atrial extrastimulation methods were able to repeatedly induce the same tachycardia with a narrow QRS and short RP that appeared clinically. Based on electrophysiological findings, a definite diagnosis of slow-fast type AVNRT was made. Therefore, radiofrequency catheter ablation was performed to modify the slow pathway below the Koch's triangle, and AVNRT was no longer induced.

We subsequently discontinued pilsicainide administration on a trial basis, and PAT due to swallowing did not reappear. On the 6th postoperative day, we performed a 24-h Holter ECG to monitor the patient without prescribing antiarrhythmic drugs. It revealed 93 single atrial premature contractions, 6 double contractions, and 1 triple or more contraction, which was a marked improvement over his earlier condition. No relationship was observed between swallowing and premature atrial contractions. The patient was able to eat without any further symptoms and gained 3 kg in 7 days. The patient was discharged on the 10th postoperative day, which was the 22nd day of hospitalization.

Discussion

There are two types of arrhythmias induced by swallowing: bradycardia and tachycardia. While most cases are bradycardia, very rarely, swallowing can also induce tachycardia. Most cases of swallowing-induced bradycardia are complicated by diseases of the heart or esophagus; in contrast, most patients with swallowing-induced tachycardia have a normal gastrointestinal tract and heart (1, 2). Swallowing-induced atrial tachycardia was first described by Sakai and Mori in 1926 as Schlucktachycardie, and to date, only about 50 cases have been reported in the literature (1). Although the underlying mechanism is unclear, several pre-

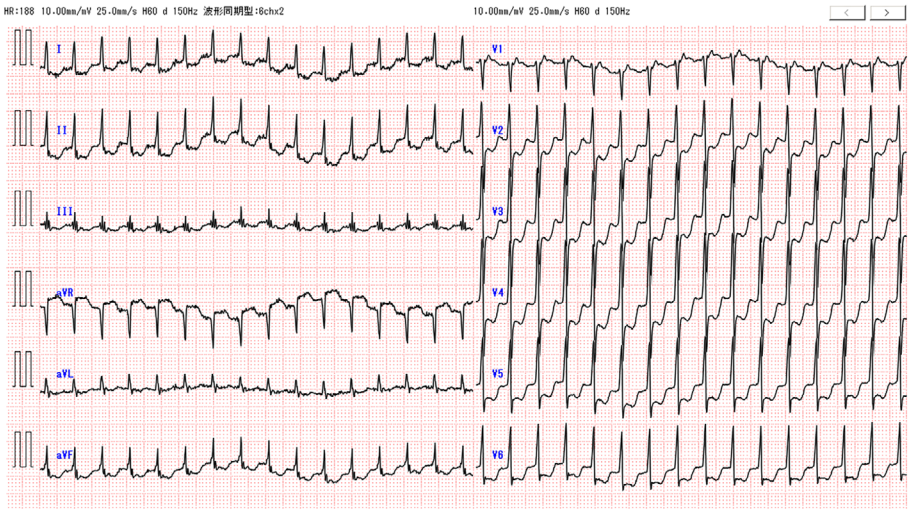

Figure 3. **The patient shows frequent narrow QRS tachycardia after oral pilsicainide, regardless of swallowing. A retrograde P wave was observed, and the electrocardiogram was suggestive of atrioventricular nodal re-entrant tachycardia.**

dictive mechanisms have been suggested in previous reports. One theory suggests mechanical stimulation of the left atrium by the distended esophagus as the cause, as Cohen et al. reproduced atrial fibrillation by inflating a balloon in the esophagus at the level of the left atrium (1, 2). However, this theory does not apply to all patients, as the reproducibility of this method of inducing tachycardia is not consistent, regardless of the position of the balloon in the esophagus, and dry swallowing can also induce tachyarrhythmias (1). The other putative mechanism involves the influence of the autonomic nervous system. Parasympathetic nerve stimulation generally causes bradycardia, but vagal nerve stimulation can shorten the relative refractory period of the atria and can induce an abnormal rhythm, resulting in tachycardia (3). In addition, it has been reported that heterogeneous depolarization of atrial muscle caused by sympathetic nerve stimulation is involved in the formation of focal re-entry (1, 2).

A diagnosis of swallowing-induced tachycardia is made by consistently recording data at the onset of symptoms and ruling out other tachyarrhythmia mechanisms. Past reports have recommended the use of a 24-h Holter ECG. In addition, upper gastrointestinal endoscopy may also be performed to confirm the presence of esophageal disease. Since swallowing-induced atrial tachycardia is common in patients with normal cardiac structures, an echocardiogram is also recommended to confirm a lack of structural heart abnormalities (1). It is not uncommon for swallow-induced tachycardia to develop suddenly in adulthood, as in the present case (4, 5). Although the mechanism is currently unknown, it is thought to include a decrease in esophageal peristalsis with age, exacerbation of autonomic dysreflexia, and progressive structural and electrical remodeling of the atrial musculature. Based on the present findings, we believe that the swallowing stress test, which examines vital signs and ECG changes during swallowing, effectively detects swallowing-induced tachycardia and may be useful when encountering patients with similar symptoms.

When treating swallowing-induced tachycardia, the first step is to avoid ingesting stimulants, such as coffee and cold drinks. As for pharmacotherapy, in some cases, like the present case, class I antiarrhythmic drugs are used. In other cases, atropine, catecholamines, and beta-blockers, which act directly on the autonomic nervous system, have been used for swallowing-induced tachycardia (6, 7). Since the effectiveness of certain drugs varies from case to case, the preferred choice has not yet been determined. In addition, surgical treatment has also been used to manage swallowing-induced tachycardia, including physical separation of the left atrium from the esophagus by repositioning the esophagus, and circular myotomy of the esophagus, as performed by Kalloor et al. in the past (2, 8). However, in recent years, surgical approaches have not been considered appropriate (1). Radiofrequency catheter ablation should be considered for ectopic foci in patients with a certainty of inducibility and only one or a few arrhythmia foci, as it can provide a complete cure (4). In the present case, the modification of the AVN slow pathway by radiofrequency catheter ablation completely relieved the symptoms of AVNRT. Furthermore, despite the absence of ablation therapy for PAT, PAT did not recur after discontinuation of pilsicainide; it is possible that the source of PAT was in the vicinity of the ablation site for AVNRT, but this does not fully explain why PAT with multiform P waves was cured. Since swallowing-induced tachycardia is considered to be affected by the autonomic nervous system, energization to the site in question might have affected the endogenous autonomic nervous system of the heart.

The present patient came to our hospital with a chief complaint of rapid weight loss, anorexia, and syncope. Although gastrointestinal diseases, such as malignant tumors, were first suspected, the symptoms were ultimately ex-

plained in a unified manner by tachycardia caused by swallowing. A swallowing stress test with an ECG and vital sign monitoring was useful in the diagnosis of swallowing-induced tachycardia. Swallowing-induced tachycardia is indeed a very rare condition but should be kept in mind as a potential cause of syncope.

The authors state that they have no Conflict of Interest (COI).

References

1. Mathew R, Green MS, Nery PB. Swallowing-induced atrial tachycardia. CMAJ **190**: E507-E509, 2018.
2. Burton JR, Sachs HJ, Keon WJ, FitzGibbon GM. Intrapleural positioning of esophagus for treatment of swallowing-induced arrhythmia. Chest **79**: 367-368, 1981.
3. Gordon J, Saleem SM, Ngaage DL, Thorpe JA. Swallow syncope associated with paroxysmal atrial fibrillation. Eur J Cardiothorac Surg **21**: 587-590, 2002.
4. Tada H, Kaseno K, Kubota S, et al. Swallowing-induced atrial tachyarrhythmias: prevalence, characteristics, and the results of the radiofrequency catheter ablation. Pacing Clin Electrophysiol **30**: 1224-1232, 2007.
5. Suarez LD, Chiozza MA, Foye R, Mosso H, Perosio AM. Swallowing-dependent atrial tachyarrhythmias. Their mechanism. Journal Electrocardiol **13**: 301-305, 1980.
6. Tandeter H, Kobal S, Katz A. Swallowing-induced atrial tachyarrhythmia triggered by salbutamol: case report and review of the literature. Clin Cardiol **33**: E116-E120, 2010.
7. Wilmshurst PT. Tachyarrhythmias triggered by swallowing and belching. Heart **81**: 313-315, 1999.
8. Kalloor GJ, Singh SP, Collis JL. Cardiac arrhythmias on swallowing. Am Heart J **93**: 235-238, 1977.