

# Swallowing-induced Paroxysmal Atrial Fibrillation Associated with Neurocardiogenic Syncope – A Case Report

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## Abstract

Swallowing-induced tachyarrhythmias is a rare phenomenon, with only over 50 cases reported in the literature. We reported a unique case of swallowing-induced paroxysmal atrial fibrillation (AF) associated with Type IIB neurocardiogenic syncope. A 71-year-old woman presented with intermittent palpitation happening mostly at night time and during meal when she swallowed food. The symptom of palpitation could be relieved by walking exercise. Paroxysmal atrial fibrillation was documented during palpitation. She also had an episode of syncope. Brief episodes of AF could be easily induced by drinking water in room temperature. Tilting table test results showed a very short duration of repetitive premature atrial contractions (PACs) followed by cardiac asystole up to 4.86 seconds after provocation with sublingual nitroglycerin. Under the diagnosis of swallowing-induced AF and Type IIB neurocardiogenic syncope, she was successfully treated with a DDDR pacemaker and oral propafenone 150mg three times a day. The clinical symptoms were under well control and there was no more syncope after treatment. (J Intern Med Taiwan 2019; 30: 42-46)

**Key Words:** Swallowing, Tachyarrhythmias, Atrial fibrillation

## Introduction

Atrial fibrillation (AF) is the most common sustained tachyarrhythmia encountered clinically. The etiology of AF is complicated and it might be associated with autonomic nervous system activation, renin-angiotensin-aldosterone system activation, genetic variants or other extracardiac factors, such as hypertension, hyperthyroidism, or alcohol consumption<sup>1</sup>. Swallowing-induced dysrhythmias can be classified into tachyarrhythmias and bradyarrhythmias, with the latter being more common.

They typically manifest as transient atrioventricular block in the presence of either an esophageal abnormality or known coronary artery disease, and its mechanism has been considered mainly due to vagotonic reflex. Swallowing-induced tachyarrhythmias are less frequent with unclear etiology, and usually not associated with cardiac or esophageal abnormalities<sup>2,3</sup>. We report a case of swallowing-induced paroxysmal AF (SIAF) associated with neurocardiogenic syncope which has not been reported before.

## Case Report

A 71-year-old woman, who had been suffering from intermittent palpitation for over 10 years, was referred to us because of recent syncope. Paroxysmal AF had been documented during her previous palpitation episodes. Detailed history revealed that her palpitation happened mostly either at night time period or during meal, and could be relieved by exercise. She had no dysphagia or dyspepsia. Esophagogastroduodenoscopy did not find any pathological change in her esophagus and stomach. She was a nonsmoker and did not consume alcohol or caffeinated beverages. Her family history was negative for cardiac dysrhythmias or other heart diseases.

On physical examination, there was no remarkable finding except anxious personality. Her resting 12-lead ECG and thyroid function were normal. Echocardiography revealed normal left and right ventricular function with no regional wall motion abnormality. 24-hour Holter ECG showed several episodes of paroxysmal AF, with asystole up to 6 seconds following one of the AF episodes. Brief episodes of AF could be easily induced by drinking water in room temperature (Figure 1). A tilting

table test was arranged in order to understand the possible mechanism of her syncope. A very short duration of repetitive PACs followed by cardiac asystole up to 4.86 seconds was induced during the test after provocation with sublingual nitroglycerin (Figure 2). Frequent paroxysmal AF episodes were recorded immediately after tilting table test. Under the diagnosis of swallowing-induced AF and Type IIB neurocardiogenic syncope, she was successfully treated with a DDDR pacemaker and oral propafenone 150mg three times a day. The clinical symptoms were under well control and there was no more syncope after treatment.

## Discussion

The first case of a swallowing-induced tachyarrhythmia was reported back in 1926 by Sakai and Mori<sup>4</sup>, approximately over 50 cases have been reported since then. The prevalence of swallowing-induced tachyarrhythmia was reported to be 0.6% among patients presented with symptomatic atrial arrhythmias. The clinical characteristics were: (1) males predominated 9:1 over females, (2) most cases occurred over 35 years of age, (3) tachyarrhythmias occurred consistently and reproducibly shortly after

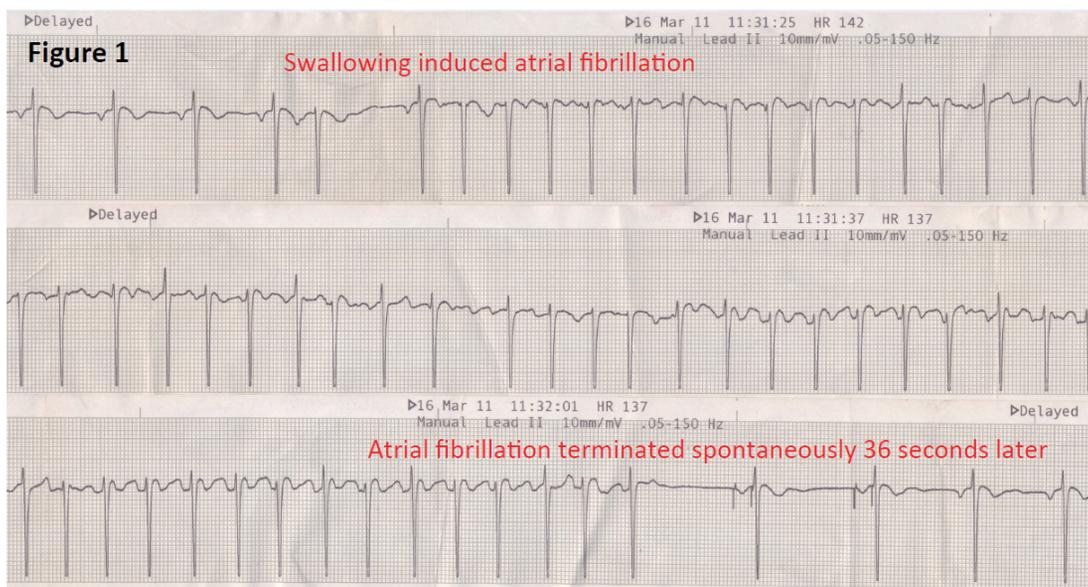


Figure 1. Paroxysmal atrial fibrillation easily induced by water swallowing.

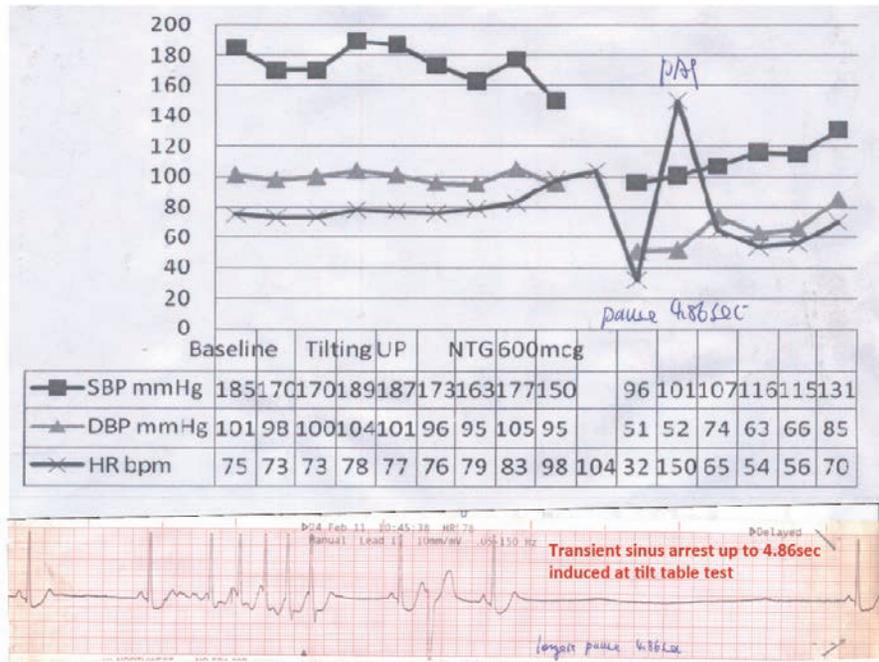


Figure 2. Cardiac asystole up to 4.86 seconds was induced during tilt table test after provocation with sublingual nitroglycerin.

each swallow, (4) 90% of the patients had premature atrial contractions (PACs) and/or atrial tachycardia (AT) as the manifesting arrhythmia, (5) the PACs provoked by swallowing usually had the same P-wave morphology as the first beat of the AT and AF<sup>5</sup>. Moreover, atrioventricular (AV) nodal reentrant tachycardia and AV reciprocating tachycardia as well as AF can be induced by swallowing<sup>6,7,8</sup>. There were few cases about the SIAF. Our patient was relatively older in comparison with previous cases, and presented with SIAF which was rarely reported.

The true mechanism of swallowing-induced tachyarrhythmias is unclear. Several mechanisms have been postulated in previous studies but with inconsistent suggestions. Cohen et al first reported atrial fibrillation triggered by balloon dilatation of the esophagus at the level of left atrium, thus suggested that mechanical stimulation of left atrium by distended esophagus was the mechanism<sup>9</sup>. Burton et al even reported a case in which the patient needed surgery to reposition the esophagus to cure swallowing arrhythmia<sup>10</sup>. However, this has not been repro-

ducibly demonstrated. This theory was also not able to explain cases in which the focus of tachyarrhythmias was not in the left atrium, such as pulmonary veins, right atrium and superior vena cava<sup>3,11,12</sup>. Gastroendoscopy and image study in our patient did not suggest any evidence of direct contact between esophagus and left atrium.

Autonomic nervous system activation can induce significant and heterogeneous changes of atrial electrophysiology and induce atrial tachyarrhythmias. Lindsay et al proposed a vagal nerve-mediated neural reflex as the initiating mechanism of swallowing-induced atrial tachyarrhythmias. The increased intra-esophageal pressure associated with swallowing activated the afferent and efferent branches of the vagus nerve. Atrial ectopy may result from preferential vagal discharge to the atrial myocardium, which in turn may produce AT or AF<sup>13</sup>. In a case report by Morady et al, an esophageal manometric study demonstrated that the swallowing tachycardia was coincident with relaxation of the upper esophageal sphincter and pre-

ceded the peristaltic activity in the esophageal body. They also described that the most possible mechanism was a vagally mediated neural reflex involving a neurotransmitter other than acetylcholine because atropine and bethanechol did not affect the swallowing-induced AT<sup>14</sup>. Results from Heart rate variability evaluation also suggested an increase of parasympathetic nerve activity with suppression of sympathetic nerve activity when swallowing-induced AT occurred. The high frequency (HF) component was suppressed after ablation with suppression of PACs, suggesting that parasympathetic nerve activity was strongly involved in the cause of this arrhythmia rather than the sympathetic nerve system<sup>11</sup>. However, this hypothesis does not explain the poor effectiveness of vagolytic medications such as atropine in these patients. Recently, epicardial adipose tissue (EAT) which contains autonomic ganglionated plexus with both adrenergic and cholinergic nerves, was shown to be related to the mechanism of swallowing-induced AT as a neural reflex<sup>15</sup>.

Adrenergic reflexes originating in the esophageal wall have been thought to play a role in swallowing-induced tachyarrhythmias. Exaggerated sympathetic activity, manifesting as hyperresponsiveness of the myocardium to catecholamines has been described in swallowing-induced atrial fibrillation<sup>16</sup>. Tandeter et al reported a case of swallowing-induced atrial tachyarrhythmia which was initiated by the beta-agonist salbutamol also suggested the role of sympathetic activity<sup>17</sup>.

Our patient experienced palpitation mostly either at night time period or during meal, and could be relieved by walking exercise. Usually the vagal tone is higher at night time especially during sleep. It is reasonable to speculate that vagal reflex plays an important role in swallowing-induced AF in our patient. During tilt table test, a very short duration of repetitive PACs followed by cardiac asystole up

to 4.86 seconds was induced. This also indicated clearly that our patient had an exaggerated vagal-mediated response. There has no known previous case report of this exact condition like ours demonstrating the combination of swallowing-induced AF and cardioinhibitory neurocardiogenic syncope.

Management of swallowing-induced tachyarrhythmias can be difficult. Given the variations in the trigger and type of the arrhythmias, different mechanisms may apply to each individual patient. There is no universally successful treatment for swallowing-induced tachyarrhythmias. There were a variety of medication including verapamil, beta-blockers, class Ia, Ic antiarrhythmic drugs, and amiodarone has been reported to achieved satisfactory symptoms control<sup>2,3,4</sup>. Newer techniques of radiofrequency catheter ablation of the arrhythmogenic source have also been attempted successfully on swallowing-induced tachyarrhythmias refractory to medical treatment<sup>11, 12,18,19</sup>.

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## 吞嚥誘發陣發性心房顫動合併神經心源性昏厥： 病例報告

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### 摘要

吞嚥導致之心搏過速心律不整是相當罕見的現象，過去文獻約只有 50 幾例個案報告。本文報告一位吞嚥引起陣發性心房顫動合併第二類 B 型神經心因性昏厥的案例。一位 71 歲女性主訴間歇性心悸，常發生於夜間及用餐吞嚥時。心悸的症狀在走路活動之後會緩解，心悸時心電圖記錄到陣發性心房顫動，患者有一次昏厥的病史。給予飲用常溫水可以很容易引發心房顫動的短暫發作。傾斜床測試中，在給予舌下硝化甘油 (Nitroglycerin) 後，誘發重複性心房早期收縮，緊接著發生心跳休止達 4.86 秒。在吞嚥誘發心房顫動合併第二類 B 型神經心因性昏厥的診斷下，經由雙腔節律器植入合併 Propafenone 150mg 一天三次治療，病患的臨床症狀得到良好控制，沒有再發生暈厥。