

CASE REPORT

Swallowing-induced atrial tachycardia: case report

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Introduction

Swallowing-induced atrial tachycardia (SIAT) was described by Sakai in 1926 [1]. Rare cases of SIAT have been previously reported in the literature. Despite several proposed mechanisms, the exact pathophysiology of this rare entity remains speculative. Management of this tachycardia has been difficult, with variable degrees of success with antiarrhythmic therapy. Medication, surgical denervation, and radiofrequency ablation have been used with possible success. We present the case of a patient with swallowing-induced high right atrial tachycardia who underwent dissection of esophageal leiomyoma.

Case Report

A 53-year-old man with no prior cardiac history presented with recurrent palpitations while swallowing. Palpitations only occurred when the patient swallowed solids. The palpitations lasted for about 30 sec. Gastroscopy revealed a leiomyoma of esophagus which bulged inwards, 2 cm diameter and smooth boundary. Physical examination was unremarkable. Electrocardiogram showed sinus rhythm with complete left bundle branch block. Echocardiogram was normal. Event

Key Clinical Message

A 53-year-old man presented with heart palpitations while swallowing. Electrophysiologic study (EPS) and immunohistochemical results of his esophageal leiomyoma suggested that swallowing-induced atrial tachycardia is related with neural reflex. S100-immunopositive nerve fibers are demonstrated sympathetic nerves which possibly explain the mechanism. Metoprolol tartrate tablets are effective in our patient.

Keywords

Esophageal leiomyoma, swallowing-induced atrial tachycardia, sympathetic nerve

monitoring revealed short runs of wide complex tachycardia at rates of 140–160 bpm lasting for 3–10 beats (Fig. 1). The episodes were noted only when eating dry foods. The patient was nonresponsive to calcium channel blockers as well as propafenone.

Electrophysiologic study was performed. Multipolar catheters were placed in the right atrium, his-bundle region, right ventricle, and coronary sinus. Programmed electrical stimulation and burst pacing of the right atrium failed to induce tachycardia. Isoproterenol (30% increase in heart rate) failed to induce the tachycardia. The patient was asked to swallow cookies. The action induced short runs of nonsustained atrial tachycardia. The earliest site of activation was mapped to high-posterior right atrium (Fig. 2). Given the esophageal leiomyoma and the risk of injuring the sinus node, ablation was not attempted. Endoscopic submucosal dissection (ESD) was subsequently performed with the patient under intravenous anesthesia. Monitor did not reveal any tachycardia during this process (Fig. 3). Pathologic diagnosis confirmed the lesion to be esophageal leiomyoma. Immunohistochemistry showed CD34 (vessel +), SMA (++) , DES (+++), S100 (+), and Ki-67 (1% +). Use of beta receptor blocker for 1 month after discharge revealed no recurrence of symptoms.

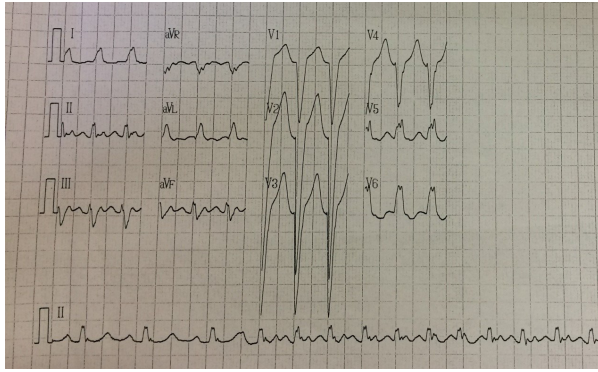


Figure 1. Atrial tachycardia was induced while the patient swallowed.

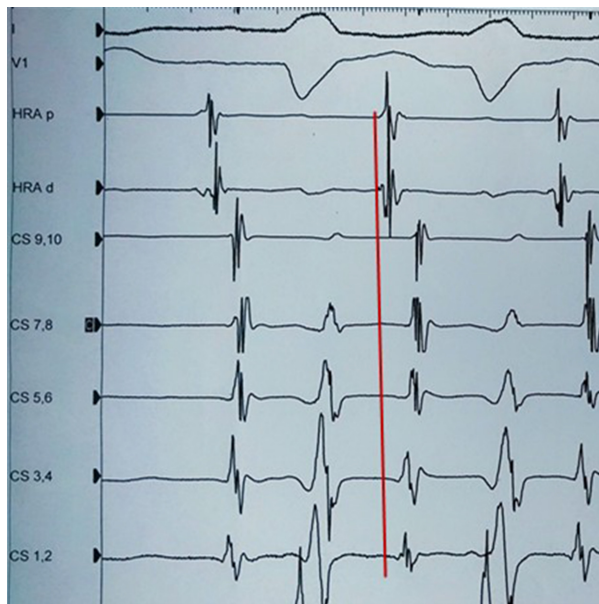


Figure 2. Excitation of A wave is seen to be early in lead HRAd.

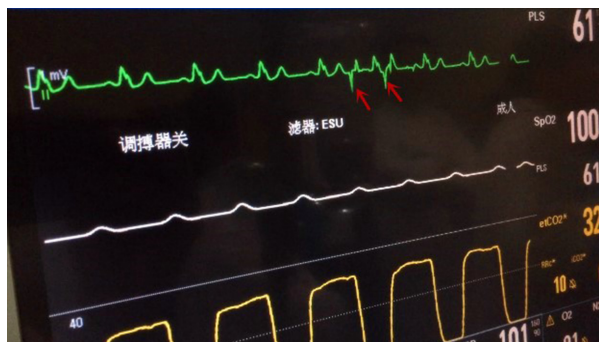


Figure 3. Unusual signal (Red arrow) was regarded as current interference during the dissection.

Discussion

Review of the published data suggests that the condition more commonly affects men over a wide age range (49 ± 14 years) [2]. Most of these patients have no evidence of esophageal disease. Swallow-induced dysrhythmias have been separated into two categories: bradydysrhythmias and tachydysrhythmias. Swallowing-induced atrial tachycardia is rarely reported.

The mechanism of swallowing-induced atrial tachycardia (SIAT) remains controversial. One theory is direct mechanical interaction between the esophagus and the left atrium. Others have suggested that the neural reflex may play a role in SIAT. Mechanical stimulation of the left atrium with swallowing food has been thought to be a potential causal factor in some patients [3–5]. Direct mechanical stimulation might not be responsible for the arrhythmia in our case based on the following findings: (1) Esophageal leiomyoma bulged into the lumen and it was not pressing the left atrial. (2) Electrophysiologic study proved that tachycardia was right atrial in origin. (3) SIAT was not occurred during the ESD.

Electrophysiologic studies suggest that the origin of the ectopic atrial activity is either the left or the right atrium, with no inducibility during programmed electrical stimulation. The mechanism appears to be activation of neural reflexes during episodes of swallowing. To our knowledge, seven cases of successful SIAT treatment using radiofrequency ablation (RFA) have been reported between 2005 and 2014. In two of the seven cases, the origin of SIAT was the low posterior right atrium (RA); in two cases, the ostium of the right superior pulmonary vein (PV); in one case, the ostium of the right inferior PV; in one case, the ostium of both right PVs; in one case, the ostium of superior right atrial, the fat pad between the superior vena cava (SVC) and ascending aorta [2, 6–9]. The origins of SIAT seem to be near the high-density ganglionated plexus (GP) areas. This result suggests that the origin of SIAT is close to the GP and the mechanism is related to the vagal nerve reflex (Fig. 4). In our case, the origin of the arrhythmia was in the superior right atrium, close to the sinus node, near the GP area. Vagal modulation of the ectopic focus was the likely mechanism for tachycardia in this case.

Stimulation of the cardiac sympathetic nervous system has been shown to alter atrial repolarization causing focal reentry and to promote triggered activity via delayed after depolarizations. Previous literature showed the density of S100-immunoreactive nerve fibers in the ventricles to be significantly higher in patients with a history of ventricular arrhythmia than in patients without ventricular arrhythmia [10]. Most S100-immunopositive nerve fiber-like structures are proved to be related with sympathetic nerves. Some authors have seen sympathetic nervous system blocking agents to be successful in either reducing the duration

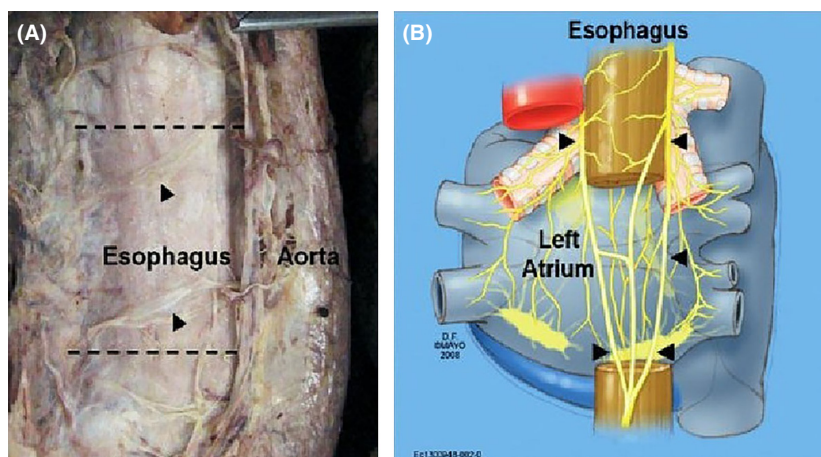


Figure 4. A large number of vagal nerve fibers were distributed on the surface of the left atrium and esophagus.

or abolishing the tachycardia in some cases [11–13]. In our case, immunohistochemical results of esophageal leiomyoma in our patient showed S100 (+). As most S100-immunopositive nerve fiber-like structures are demonstrated sympathetic nerves [11], it is suggested that there may be a high density of sympathetic nerve between atrium and esophagus in our patient. The mechanism for SIAT may be a sympathetic reflex in our case. Furthermore, suppression of symptoms by beta receptor blocker in our patient is consistent with this speculation. Therefore, we speculate that one or more of the mechanisms mentioned above may be involved with our patient's arrhythmia.

Conclusion

We report a case of SIAT with arising from the superior right atrial, near to the GP area, which was examined by EPS. The immunohistochemical analysis of esophageal leiomyoma revealed S100 (+), which is connected with sympathetic nerves. Beta receptor blocker has been used successfully in this patient. We hypothesized that the mechanisms were related to the vagal nerve reflex and the sympathetic reflex in our patients.

Conflict of Interest

None declared.

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