Social anxiety provoked by speech-induced atrial tachycardia

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Introduction

Focal atrial tachycardia (AT) mechanisms include enhanced automaticity and triggered activity. Autonomically mediated focal atrial arrhythmias (AT or atrial fibrillation [AF]) are rare. Adrenergic stimulation such as exercise or stress can initiate focal atrial arrhythmias by enhancing automaticity, whereas vagal nerve arrhythmogenicity is associated with reentry and early afterdepolarizations due to shortening of the action potential duration.[1] We report the case of a patient with AT that was initiated with speech and dissipated with silence. The patient also had atrioventricular reentrant tachycardia.

Case report

A 78-year-old Caucasian female without a history of structural heart disease was referred for electrophysiology study for palpitations of 1-year duration. She noted that her heart rate would suddenly speed up while she was speaking and would subside spontaneously after she stopped speaking. She reported fluttering in her chest, dyspnea, and fatigue with these episodes. The symptoms could be triggered by the slightest cadence, including whispering. The palpitations lasted until speech cessation. There was no association with coughing, sneezing, eating, drinking, or Valsalva maneuver. Symptoms had progressed to the point that she preferred to stay alone at home. She avoided family gatherings and even answering her home phone. She experienced occasional chest discomfort and had a few near-syncopal episodes. She also had a few episodes of sustained palpitations for a few minutes that would start and stop spontaneously and were unrelated to speech. She originally was diagnosed with paroxysmal AT near 150 to 170 bpm. She was started on metoprolol and then switched to sotalol therapy, but her symptoms persisted.

During hospitalization, her bedside telemetry demonstrated periods of AT during conversation or during spontaneous speech that returned to normal sinus rhythm with silence (see Online Supplemental Video). During electrophysiologic study, she had reproducible speech-induced focal right atrial tachycardia, which was mapped to the mid–crista terminalis, with cycle length of 370 ms. Episodes were easily prompted by having the patient talk in the laboratory and were prolonged with isoproterenol infusion. The focal source had a fractionated multicomponent signal on the local atrial electrogram, whereas the unipolar electrogram showed a sharp QS configuration with atrial activation preceding surface P-wave onset by 28 ms (Figure 1). Radiofrequency energy application at that site terminated the AT (Figure 1). AT could no longer be initiated by the patient speaking or with isoproterenol infusion. The presence of eccentric retrograde atrial activation during ventricular pacing was also noted. She was found to have a concealed left posterolateral accessory pathway that was an integral component of orthodromic atrioventricular reciprocating tachycardia at a cycle length 410 ms initiated with atrial extrastimulus testing (Figure 2). This tachycardia was eliminated by ablation of the accessory pathway. On follow-up, the patient was able to communicate with family and friends with no further episodes of palpitations, discomfort, or malaise.

Discussion

This case report demonstrates the interaction between the cardiac and the autonomic nervous systems (vagal stimulation) on heart rhythm. AT and AF have been reported with coughing, speaking, and swallowing.[2–7] One case localized the site of earliest activation to the roof of the left atrium, whereas another noted earliest activation was in the anteroseptum of the left atrium.[2,3] Fan et al[2] postulated that it may be due to an anomalous efferent input from the recurrent laryngeal nerve to the left atrial ganglionic plexuses. Omori et al[6] believed the arrhythmogenicity of the vagal nerve stimulation in their case was supported by the effectiveness of atropine and the propensity to increase the arrhythmia with digitalis though direct vagotonic action. The vagus nerve provides parasympathetic input to the heart and lungs; it also controls laryngopharyngeal function. Vagal stimulation of the sinus node causes slowing of the heart rate. On the other hand, it can induce atrial tachyarrhythmia by enhancing delayed afterdepolarizations resulting in focal firing from the pulmonary veins.

KEYWORDS Focal atrial tachycardia; Speech-induced tachycardia; Atrioventricular reentrant tachycardia

ABBREVIATIONS **AF** = atrial fibrillation; **AT** = atrial tachycardia

(Heart Rhythm Case Reports 2015;1:117–119)

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http://dx.doi.org/10.1016/j.hrcr.2015.01.002

KEY TEACHING POINTS

- Patients may experience specific symptoms associated with arrhythmias when they perform actions such as swallowing, coughing, or speaking. Although this is rare, it can be very debilitating for the patient.
- The arrhythmias usually can be captured by having the patient reproduce the action while undergoing electrocardiographic monitoring.
- The arrhythmias are thought to be vagally mediated due to innervation of the atria, although this is only speculative. There is complex relationship between autonomic nervous system and atrial tissue stimulation.
- Patients may have several factors leading to their symptoms and disease process. In this case, the patient had a demonstrable speech-induced atrial tachycardia. However, careful study also revealed a concealed left posterolateral accessory pathway that was an integral component of orthodromic atrioventricular reciprocating tachycardia.

In addition, vagal stimulation (eg, with adenosine) has also been shown to activate I_{KACh}, resulting in shortening of the atrial refractory period, which predisposes atrial tissues and pulmonary veins to reentrant atrial arrhythmias (AT/AF).[8]

Our patient is unique because she had incessant and reproducible AT during speech without any change in voice cadence. Our case shows the interplay between vagal and adrenergic drive given that the tachycardia was easily initiated with speech and stopped immediately after speaking ended. With isoproterenol infusion, the tachycardia initiated with speech but persisted after cessation of speech. Having the patient speak provided a reproducible trigger during electrophysiologic mapping and ablation, which led to successful termination of the arrhythmia. Therefore, we postulate that our patient most likely had supramedullary mechanisms controlling voice production. This may abnormally modulate vagal activity due to aberrant innervation and physiology resulting in initiation of the focal AT at the mid–crista terminalis. Atrioventricular reentrant tachycardia may possibly explain the sustained palpitations. Previous cases of speech-induced tachycardia were unable to elicit dual atrioventricular node physiology or ventricular pre-excitation.[3,5,6]

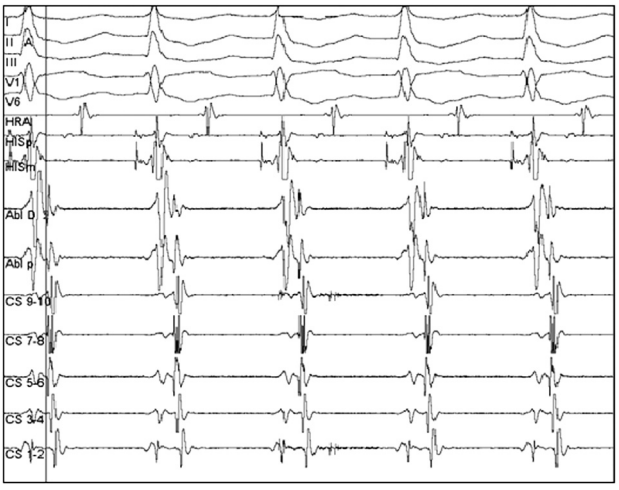

Figure 2 Atrioventricular reentrant tachycardia using a left posterolateral accessory pathway with earliest activation in the mid–coronary sinus (CS 5,6) and distal ablation (Abl D) leads. From top to bottom: HRA = high right atrium; HISp = His proximal; HISm = His middle; Abl p = ablation proximal; Abl D = ablation distal; Abl U = ablation unipolar; CS 9-10 = coronary sinus proximal; CS 1-2 = coronary sinus distal.

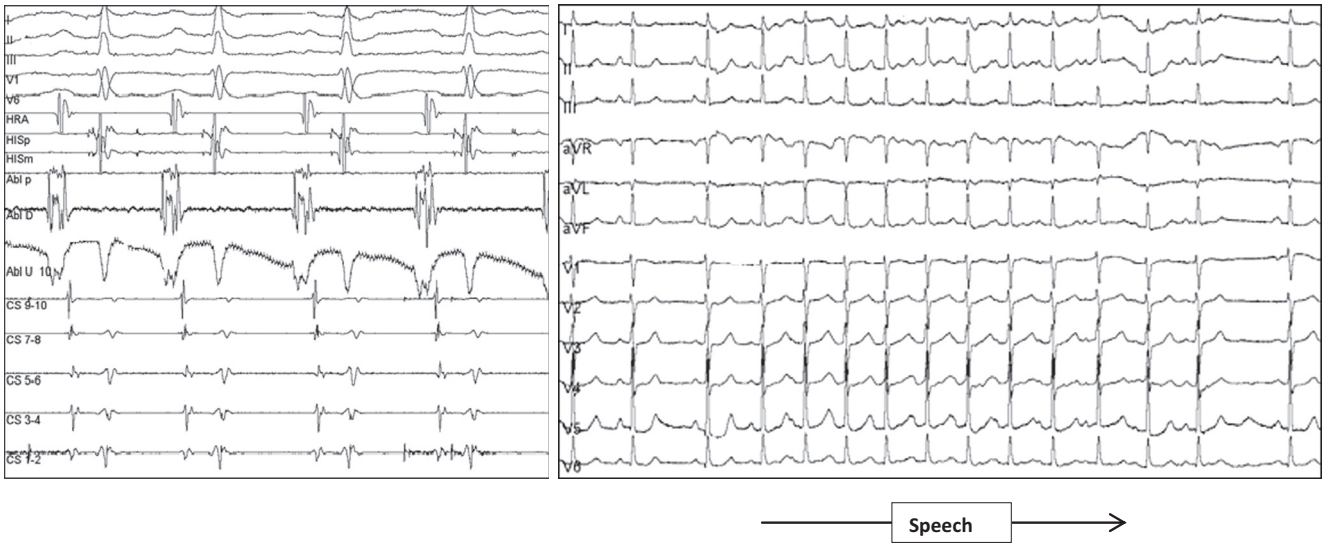

Figure 1 **Left:** Intracardiac electrogram showing earliest activation close to the mid–crista terminalis. **Right:** Surface electrogram showing atrial tachycardia initiating with speech and subsiding with silence. From top to bottom: HRA = high right atrium; HISp = His proximal; HISm = His middle; Abl p = ablation proximal; Abl D = ablation distal; Abl U = ablation unipolar; CS 9-10 = coronary sinus proximal; CS 1-2 = coronary sinus distal.

To our knowledge, there are only a few limited case reports of speech-induced atrial tachyarrhythmia, and this is the first with concealed atrioventricular reentrant tachycardia. It highlights the complex interplay between autonomic and atrial tissue stimulation during speech. Although the physiologic basis for speech-induced atrial tachyarrhythmia is only speculative, it may be due to a combination of increased vagal stimulation, enhanced automaticity, and microreentry in a patient with a susceptible myocardial substrate.

Appendix

Supplementary data

Supplementary material cited in this article is available online at http://dx.doi.org/10.1016/j.hrcr.2015.01.002.

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