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Paroxysmal Atrial Fibrillation and Brain Freeze: A Case of Recurrent Co-Incident Precipitation From a Frozen Beverage

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
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Patient: Male, 29
Final Diagnosis: Paroxysmal atrial fibrillation • cold-stimulus headache
Symptoms: Palpitations • headache
Medication: Diltiazem • Ibutilide
Clinical Procedure: None
Specialty: Emergency Medicine • Internal Medicine

Objective: Unknown ethiology • Rare disease

Background: Episodes of paroxysmal atrial fibrillation may be precipitated by the rapid ingestion of ice-cold foods and beverages. This condition has received little research attention, and its true prevalence is poorly described. Treating physicians may not identify cold ingestion as a causal factor of paroxysmal atrial fibrillation, thus compromising both history taking and patient education.

Case Report: We report a case of a healthy young-adult man who drank a slushed ice beverage that immediately induced atrial fibrillation and a brain freeze headache simultaneously. This occurred on two separate occasions, years apart. During both episodes, the acute brain freeze self-resolved quickly, but the new-onset palpitations occasioned a visit to the emergency department for diagnosis and treatment. The emergency physicians failed to make the causal link between the cold drink and the atrial dysrhythmia. Though the brain freeze headache and paroxysmal atrial fibrillation were both precipitated by rapid ingestion of an ice-cold beverage, the mediating mechanisms are distinct. We review these two cold-induced conditions, their prevalence, and their probable mechanisms.

Conclusions: The recurrent simultaneous occurrence of brain-freeze headache with paroxysmal atrial fibrillation identifies the ingestion of a frozen beverage as the precipitant of the atrial dysrhythmia. Increasing physician awareness of cold ingestion as a cause of paroxysmal atrial fibrillation will improve history taking and patient education.

MeSH Keywords: Atrial Fibrillation • Beverages • Cold Temperature • Headache Disorders • Ice Cream

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Background

It is well-known that cold stimulation to the palate can cause a temporary brain freeze headache [1,2]. Induction of atrial fibrillation (AF) by a cold drink, however, has been only rarely reported, but may be significantly more prevalent than the case literature would suggest [3–6]. The simultaneous co-occurrence of brain freeze headache with paroxysmal AF, both triggered by the same cold ingestion, has not been previously reported.

Case Report

A healthy 29-year-old man presented to the emergency department (ED) one evening complaining of sudden-onset irregular palpitations one hour prior to arrival. The symptoms began just as he started ingesting a commercial slushed ice drink. Co-incident with the palpitations, he developed a brain freeze headache. Review of systems was negative for chest pain, dyspnea, and pre-syncope. He took no medications and customarily ingested two cups of coffee every morning, as he had on the day of presentation.

Past medical history was negative for any structural cardiac or neurological conditions. He reported several prior episodes of short, self-resolving palpitations. Only once in the past had he sought medical attention for these and was found to be in AF, which was successfully cardioverted in the ED with 1 mg of intravenous ibutilide.

By the time he arrived at the ED his headache had resolved, but the palpitations persisted. His blood pressure was 166/70 mmHg, heart rate was 136 beats/min, respiratory rate was 18 breaths/min, and his oxygen saturation was 99%. Other than an irregularly irregular rhythm, his physical examination was normal. The patient was placed on a cardiac monitor, pulse oximeter, and oxygen by nasal cannula at 2 liters per minute. A peripheral IV line was placed, and a 12-lead electrocardiogram was obtained, which revealed AF with a rapid ventricular response and normal intervals. Laboratory studies, including a complete blood count, renal function, serum electrolytes, and cardiac biomarkers, were all normal.

Diltiazem, 10 mg, was administered intravenously and reduced his rapid ventricular response to less than 110 beats/min. Because he was young without structural heart disease with AF onset <48 hours prior to presentation, and moderately symptomatic AF, cardioversion was pursued. Cardiology was consulted and ibutilide was chosen because it had been effective in the past. The emergency physician discussed the treatment options with the patient and he agreed with the treatment plan. Two doses of 1 mg ibutilide were administered, each infused over 10 minutes, and separated by a 15-minute interval. Fifty-six minutes after the start of the first dose, the patient converted to a normal sinus rhythm with complete resolution of his symptoms. He was monitored in the ED for a total of four hours without complications. He was discharged home with instructions to avoid caffeine and follow-up with his primary care provider. As an outpatient, he subsequently underwent a transthoracic echocardiogram, a stress test, and Holter monitoring, all of which were normal.

Interestingly, several years later the patient returned to the ED with the identical complex of symptoms, again precipitated by a slushy ice drink. He was again treated with intravenous ibutilide and was referred to cardiology for follow-up.

Discussion

This case illustrates that ingesting a slushy ice drink can cause the simultaneous occurrence of two different medical conditions. Deglutition of a cold drink has been widely reported as a precipitant of brain-freeze or ice-cream headache, formally termed cold-induced or cold-stimulus headache. It is described by the International Headache Society as a rapid-onset, short-lasting frontal or temporal pain induced by passage of cold material over the palate and/or posterior pharyngeal wall [1,2]. Brain freeze headache develops within seconds of exposure and resolves within minutes after the removal of the cold stimulus [2,7]. Cold-induced headache is a common phenomenon among susceptible individuals, and may be more prevalent among migraneurs [8–10]. In a large survey study in Taiwan, 40% of adolescents reported having experienced brain-freeze headache at some point in their life [7]. Randomized controlled studies have demonstrated that the time course of ingestion matters: rapid or prolonged consumption of a cold beverage or ice cream increases the likelihood of developing a brain-freeze headache [11].

Although the pathogenic mechanism of cold-stimulus headache is not fully understood, it appears to be a neurovascular event mediated by the trigeminal nerve. Using transcranial Doppler, investigators have recently demonstrated a sudden, dramatic increase in cerebral blood flow through the anterior cerebral artery associated with the sensation of brain-freeze [12]. Applying warm water to the palate reversed the cold-induced vasodilation and resolved the headache.

Co-incident with the patient's brain freeze headache was the development of paroxysmal AF, both caused by the same ice-cold drink. AF triggered by the ingestion of cold food or beverage was first reported in the literature in 1994 [4]. Swallowing in general was described as a cause of paroxysmal AF decades earlier [13], but the proposal that gastrointestinal maladies can trigger cardiac dysrhythmias dates back to the 1930s [14]. Few reports of AF caused by cold-substance ingestion have

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been published [3,5,6]; more has been written on the broader diagnostic category of swallowing-induced AF [6,13,15–17].

As the esophagus descends it closely abuts the left atrium and the vagal ganglia, implicated in the genesis of AF. This anatomic proximity allows events in the esophagus to directly influence cardiac rhythms, as may be the case in the triggering of AF by gastroesophageal reflux disease [18]. Although cold material in the esophagus may directly affect the left atrium through the wall of the esophagus, the causal connection may be more indirect, through more circuitous autonomic pathways.

The autonomic nervous system is known to be important in the genesis and maintenance of AF [19]. Using an autonomic taxonomy, precipitants can be categorized as adrenergic, vagal, or random [20]. AF is deemed adrenergic when characteristically precipitated by physical exercise or extreme emotion, and is considered vagal when developing during sleep, upon coughing, burping, or swallowing, or, in our case, with the ingestion of very cold food or beverage [21,22]. Some studies have found an association between the trigger type and success of various treatments, but the nature of the association is still being studied [20,21,23–26]. Although swallowing is usually categorized as a vagal trigger, adrenergic reflexes may nevertheless sometimes be at play. Here, adrenergic reflexes in the upper and mid esophagus directly alter atrial depolarization, causing asynchronous recovery time, resulting in atrial tachyarrhythmias, including AF [15]. Tachyarrhythmias can be precipitated by swallowing [27–29], but swallowing-induced arrhythmias are more commonly bradycardic, given the predominant role of the vagus nerve [15].

Upon ingestion of cold substances, in particular, neural pathways are triggered by receptors in the pharynx and esophagus that activate the cardioinhibitory center in the medulla. The medulla then delivers a signal via the vagus nerve to the pulmonary vein region of the left atrium, reducing the relative refractory periods of the myocytes in a nonuniform manner, increasing heterogeneity, and augmenting the ability of single atrial premature beats to initiate AF in susceptible individuals [3,24,30]. This vagally-mediated mechanism of cold-induced AF is thought to be more common in younger patients and those with structurally normal hearts [21,24,30,31]. The vagal stimulus need only be brief, just sufficiently long to initiate the AF, but not strong enough or sustained enough to dampen the rapid ventricular response. Our patient illustrates this pattern, as the cold-mediated vagal reflex triggered AF, but did not curtail the ensuing tachycardia. If the vagal drive is exaggerated, patients with cold-induced AF may present without tachycardia [24]. Ibutilide is known to be an effective cardioversion agent in patients with vagally-mediated AF, as it was in our case, perhaps through the inhibition of the muscarinic potassium current [24,32].

The prevalence of cold-swallow-induced AF is not known. Many cases may be self-resolving and thus are not brought to medical attention. We are currently undertaking a retrospective study of ED patients across 21 medical centers in California over seven years who received intravenous ibutilide, procainamide, or amiodarone for the cardioversion of AF or flutter (the Pharm CAFÉ Study). One of the variables we are identifying in a systematic fashion from the electronic health record is the suspected precipitant of the atrial dysrhythmia. An interim analysis of our first 414 cases identified five cases (1.2%) of cold-drink-induced AF, higher than we would have thought if this were a very rare phenomenon. Unlike our case report above, none of these patients complained of co-incident brain freeze attending the onset of their dysrhythmia symptoms.

We suspect that the true number of cases of cold-induced AF is under-reported in the ED since patients might not make the causal connection between the temperature of their food or beverage and the onset of rhythm-related symptoms. Even if patients were to report to their emergency physicians that cold ingestion was the probable AF precipitant, this fact may go under-documented in the health record since emergency physicians might not believe the association or think it relevant.

One prospective study asked 100 randomly selected patients presenting for hospital care for idiopathic paroxysmal AF to identify their dysrhythmogenic precipitant [33]. The investigators found that 8% of their cohort reported cold beverage ingestion to be their AF trigger. Others studies have found similar rates [21]. Combined with our preliminary study results, these data suggest that the prevalence of cold-induced AF may be higher than suspected.

Interestingly, the emergency physician in our case documented that the AF developed at the time of the rapid ingestion of the icy drink, but failed to caution the patient at the time of discharge to avoid further exposure. Rather, the physician advised, not against cold drinks, but against coffee, although this was not the precipitant of the patient's headache or dysrhythmia. Though it might be prudent for a patient with paroxysmal AF to limit caffeine ingestion, the relationship between coffee ingestion and incident AF has been called into question by a number of studies [34]. Just as in our case report, none of the five emergency physicians caring for the patients with cold-induced AF from our Pharm CAFÉ Study reported advising their patients to avoid re-exposure to ice-cold drinks. Instead, they all recommended a reduction in caffeine intake.

Conclusions

This case report illustrates that rapid ingestion of cold beverages can have serious medical implications. In our case, a

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slushy ice drink induced the simultaneous development of brain freeze headache and paroxysmal AF. Although cold-stimulus headache subsides quickly after the removal of the inciting substance, paroxysmal AF can be longer lasting, sometimes requiring medical intervention. Unless the treating physician is aware of the association between cold ingestion and paroxysmal AF, the patient may not be given the best avoidance advice on discharge. Ongoing research may help us better understand the prevalence of cold-induced AF in patients seeking medical care.

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References:

1. Headache Classification Committee of the International Headache S. The International Classification of Headache Disorders, 3rd ed. (beta version). Cephalalgia, 2013; 33: 629–808
2. Hulihan J: Ice cream headache. BMJ, 1997; 314: 1364
3. Al-Farsi Y, Tariq O, Siddiqui A, Sharef YW: Atrial fibrillation with a rare cause of initiation. Case report. Med J Babylon, 2014; 11: 482–86
4. Brodsky MA, Orlov MV, Allen BJ, Selvan A: Frozen yogurt near deep-freeze. Am J Cardiol, 1994; 73: 617–18
5. Tan CW, Gerry JL, Glancy DL: Atrial fibrillation in father and son after ingestion of cold substances. Am J Med Sci, 2001; 321: 355–57
6. Wilmshurst P: Tachyarrhythmias triggered by swallowing and belching. Heart, 1999; 81: 313–15
7. Fuh JL, Wang SJ, Lu SR, Juang KD: Ice-cream headache – a large survey of 8359 adolescents. Cephalalgia, 2003; 23: 977–81
8. Mattsson P: Headache caused by drinking cold water is common and related to active migraine. Cephalalgia, 2001; 21: 230–35
9. Selekler HM, Erdogan MS, Budak F: Prevalence and clinical characteristics of an experimental model of 'ice-cream headache' in migraine and episodic tension-type headache patients. Cephalalgia, 2004; 24: 293–97
10. de Oliveira DA, Valenca MM: The characteristics of head pain in response to an experimental cold stimulus to the palate: An observational study of 414 volunteers. Cephalalgia, 2012; 32: 1123–30
11. Kaczorowski M, Kaczorowski J: Ice cream evoked headaches (ICE-H) study: randomised trial of accelerated versus cautious ice cream eating regimen. BMJ, 2002; 325: 1445–46
12. Blatt MM, Falvo M, Jasien J et al: Cerebral vascular blood flow changes during 'brain freeze'. FASEB Journal, 2012; 26: 685.4.
13. Cohen L, Larson D, Strandjord N: Swallowing-induced atrial fibrillation. Circulation, 1970; 42: 145
14. Jervell O, Lodoen O: The gastrocardiac syndrome. Acta Med Scand Suppl, 1952; 266: 595–99
15. Malik A, Ali SS, Rahmatullah A: Deglutition-induced atrial fibrillation. Tex Heart Inst J, 2005; 32: 602–4
16. 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, 2007; 30: 1224–32
17. Kanjwal Y, Imran N, Grubb B: Deglutition induced atrial tachycardia and atrial fibrillation. Pacing Clin Electrophysiol, 2007; 30: 1575–78
18. Roman C, Bruley des Varannes S, Muresan L et al: Atrial fibrillation in patients with gastroesophageal reflux disease: a comprehensive review. World J Gastroenterol, 2014; 20: 9592–99
19. Shen MJ, Zipes DP: Role of the autonomic nervous system in modulating cardiac arrhythmias. Circ Res, 2014; 114: 1004–21
20. Oral H, Chugh A, Scharf C et al: Pulmonary vein isolation for vagotonic, adrenergic, and random episodes of paroxysmal atrial fibrillation. J Cardiovasc Electrophysiol, 2004; 15: 402–6
21. Rosso R, Sparks PB, Morton JB et al: Vagal paroxysmal atrial fibrillation: prevalence and ablation outcome in patients without structural heart disease. J Cardiovasc Electrophysiol, 2010; 21: 489–93
22. Carpenter A, Frontera A, Bond R et al: Vagal atrial fibrillation: What is it and should we treat it? Int J Cardiol, 2015; 201: 415–21
23. Efremidis M, Letsas KP, Lioni L et al: The impact of vagotonic, adrenergic, and random type of paroxysmal atrial fibrillation on left atrial ablation outcomes. Int J Cardiol, 2013; 168: 4015–18
24. Aliyev F, Celiker C, Turkoglu C, Uzunhasan I: Successful use of ivabradine in a case of exaggerated autonomic dysfunction. Turk Kardiyol Dern Ars, 2010; 38: 285–89
25. Lemola K, Chartier D, Yeh YH et al: Pulmonary vein region ablation in experimental vagal atrial fibrillation: role of pulmonary veins versus autonomic ganglia. Circulation, 2008; 117: 470–77
26. de Vos CB, Nieuwlaat R, Crijns HJ et al: Autonomic trigger patterns and anti-arrhythmic treatment of paroxysmal atrial fibrillation: data from the Euro Heart Survey. Eur Heart J, 2008; 29: 632–39
27. Mitra S, Ludka T, Rezkalla SH et al: Swallow syncope: a case report and review of the literature. Clin Med Res, 2011; 9: 125–29
28. Tandeter H, Kobal S, Katz A: Swallowing-induced atrial tachyarrhythmia triggered by salbutamol: case report and review of the literature. Clin Cardiol, 2010; 33: E116–20
29. Coumel P, Friocourt P, Mugica J et al: Long-term prevention of vagal atrial arrhythmias by atrial pacing at 90/minute: experience with 6 cases. Pacing Clin Electrophysiol, 1983; 6: 552–60
30. Ringdahl EN: Vagally mediated atrial fibrillation in a young man. Arch Fam Med, 2000; 9: 389–90
31. Chock JG, Gill MR: Paroxysmal supraventricular tachycardia induced by oral stimulation: a case report and review of swallow-induced dysrhythmias. J Emerg Med, 2002; 22: 389–92
32. Borchard R, van Bracht M, Wickenbrock I et al: [Inhibition of the muscarinic potassium current by KB130015, a new antiarrhythmic agent to treat atrial fibrillation]. Med Klin (Munich), 2005; 100: 697–703 [in German]
33. Hansson A, Madsen-Hardig B, Olsson SB: Arrhythmia-provoking factors and symptoms at the onset of paroxysmal atrial fibrillation: a study based on interviews with 100 patients seeking hospital assistance. BMC Cardiovasc Disord, 2004; 4: 13
34. Turagam MK, Velagapudi P, Kocheril AG, Alpert MA: Commonly consumed beverages in daily life: do they cause atrial fibrillation? Clin Cardiol, 2015; 38: 317–22

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