

Vagally Mediated Atrial Fibrillation in a Young Man

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Atrial fibrillation may be provoked by either vagal or sympathetic stimulation. Sympathetic effects are common in middle-aged and elderly patients with underlying heart disease. However, in the young, nondiseased heart, vagal influences are more likely to predominate. Recognition of vagally mediated atrial fibrillation in young adults as a unique clinical entity has diagnostic and therapeutic implications.

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The atria are subject to both vagal and sympathetic influences. In the diseased heart, increased sympathetic tone (physical exertion, emotional stress, caffeine, alcohol, or hyperthyroidism) may precipitate arrhythmias. However, in the absence of heart disease, vagal factors are more likely to be responsible for arrhythmias.¹ Paroxysmal atrial fibrillation in the young healthy adult more likely reflects intense vagal tone and warrants treatment that is different from that appropriate for the older patient with underlying cardiac abnormalities and heightened sympathetic influences.

REPORT OF A CASE

A 28-year-old, otherwise healthy man presented to the emergency room on July 1991 complaining of palpitations and an inability to catch his breath. Earlier in the day he had played golf and basketball, had ingested several caffeinated beverages, and had taken 60 mg of pseudoephedrine for allergic rhinitis. His symptoms developed later that evening, approximately 10 minutes after relaxing in a recliner and ingesting a chocolate milk shake.

His heart rate was 100 beats per minute (compared with a documented baseline of 45 beats per minute) and an electrocardiogram demonstrated atrial fibrillation. He underwent conversion to sinus rhythm the next morning after receiving an intravenous infusion of 1 g of procainamide. Findings from electrolyte and thyroid studies were normal, and re-

sults of an echocardiogram were normal except for mild dilation of the right ventricle. The patient was given 25 mg of atenolol per day for 6 months. Subsequent echocardiograms over the next 5 years demonstrated stability of the shape and size of the right ventricle.

The patient was asymptomatic until February 16, 1997. Shortly after 10 PM, while ingesting a chocolate milk shake, he again experienced palpitations and mild chest tightness. An electrocardiogram obtained in the emergency room showed atrial fibrillation with a ventricular response of 90 beats per minute. After no response that night to intravenous procainamide, he was given 1 mg of ibutilide twice the next morning. Sinus rhythm was re-established and the patient was discharged and prescribed 1 aspirin per day.

The patient was again asymptomatic until August 7, 1997, when he had recurrence of his symptoms after taking several bites of a chocolate sundae one evening. When procainamide did not alter his arrhythmia, the patient underwent electrical cardioversion to sinus rhythm.

COMMENT

A good patient history can help distinguish between vagally mediated and sympathetically induced atrial fibrillation. Vagally mediated atrial fibrillation occurs more frequently in young healthy men than sympathetically induced atrial fibrillation. Rather than being precipitated by physical or emotional stress, the arrhythmia is more likely to occur in the period of relaxation after these events, and most

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frequently in the evening. In fact, conversion to sinus rhythm frequently occurs in the morning when sympathetic drive is higher.¹ Other inciting events (reflecting increased vagal tone) include cough, nausea, and the postprandial state.² Conversely, adrenergically mediated paroxysmal atrial fibrillation occurs more commonly in patients older than 50 years, during the daytime, and during times of increased physical or emotional stress. The diseased myocardium is more sensitive to sympathetic stimulation evoked by these stressors.

Common denominators in this patient's paroxysms of atrial fibrillation include ingestion of cold solid food and chocolate and late evening occurrence. A review of the English-language literature did not find any association between chocolate and arrhythmias, but in this case it may have been a contributing factor. However, the time course suggests that ingestion of cold food rather than the caffeine in chocolate was a precipitant, as caffeine would not have been absorbed at the time of the atrial fibrillation. A case report in the *American Journal of Cardiology*³ describes a healthy 43-year-old woman who developed atrial fibrillation after ingesting frozen yogurt. The authors hypothesized that the atrial fibrillation was the result of intense vagal stimulation caused by the extremely cold temperature of the dessert. In many intensive care units, iced beverages are proscribed for patients with heart problems because of a possible association of cold liquids with electrocardiographic changes involving the ST segment, T wave, rate, and rhythm.^{4,5} Possibly, persons who are susceptible to vagally mediated atrial fibrillation are more sensitive to frozen solids than liquids owing to a bolus effect and/or the longer transit times through the esophagus seen with solids.

The autonomic cause of atrial fibrillation determines treatment. Patients with sympathetically mediated atrial fibrillation respond to β -blockers and/or digoxin. Digoxin works in part by increasing parasympathetic influences on the atrioventricular node.⁶ β -Adrenergic blockade of sympathetic influences similarly shifts the balance toward vagal effects on the atria. These medications, which are highly effective in sympathetically mediated atrial fibrillation, are less effective in the treatment of vagally mediated atrial fibrillation, and may even prolong the dysrhythmia.⁷ Calcium channel blockers will not terminate the arrhythmia, but are used to control the ventricular rate. One study suggests that calcium channel blockers may sustain paroxysmal atrial fibrillation.⁸ Further studies are needed to determine the role of calcium antagonists in the treatment of vagally mediated atrial fibrillation.

The published experience with the treatment of vagally mediated atrial fibrillation is limited. Currently, several clinical trials with class III antiarrhythmic drugs are under way.⁹ Either electrical cardioversion or pharmacologic cardioversion with antiarrhythmics such as procainamide or ibutilide is appropriate for termination of the acute event. Early conversion may prevent atrial electrical remodeling that may predispose to chronic atrial fibrillation.¹⁰ The risk of immediate recurrence is theoretically lower with pharmacologic conversion than

electrical cardioversion as the antiarrhythmic properties of the drug persist until blood levels decline.¹⁰ In the healthy young patient with paroxysmal atrial fibrillation, prophylaxis is not warranted after the first episode. Prophylactic treatment is not indicated unless recurrence is frequent enough to allow assessment of the efficacy of any intervention. Paroxysms that remain few and far between and relatively asymptomatic may not warrant prophylaxis. When episodes are more frequent or symptoms become intolerable, long-term use of antiarrhythmics has been shown to maintain sinus rhythm at 1 year 50% to 60% of the time, but these medications carry proarrhythmic risks.⁹ Flecainide may be more effective than propafenone in the treatment of vagally mediated atrial fibrillation because it has more significant vagolytic actions.¹¹ For patients with quite frequent recurrences, atrial pacing or ablative therapy may be indicated.

CONCLUSIONS

This case illustrates the effect of vagal influences on the young healthy heart. The late-night occurrences, the relaxed state following physical exertion, and ingestion of a frozen dessert tipped the autonomic scales sufficiently to precipitate vagally mediated atrial fibrillation. An awareness of the vagal influence in young healthy patients with paroxysmal atrial fibrillation may alter treatment decisions. Patients who are susceptible should be counseled to avoid vagally predominant situations. Traditionally used β -blockers and digoxin should be avoided, with consideration given to other antiarrhythmic agents.

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