
Case report

Atrial fibrillation associated with anaphylaxis during venom and pollen immunotherapy

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Background: Electrographic changes after insect stings have been reported both with and without systemic symptoms. To our knowledge, however, there have been no reported cases of arrhythmias in the maintenance phase of venom or pollen immunotherapy.

Objective: We report a case of a patient who developed paroxysmal atrial fibrillation after receiving venom and pollen immunotherapy. This patient was initially evaluated in an outpatient setting, and the events described occurred at an allergist's private office. We believe this is an atypical presentation and would be very pertinent to the practicing allergist.

Methods: This patient was under the care of a clinical allergist in a private practice with associations to a teaching hospital. Interventions were as described in the body of the paper, more specifically venom and pollen maintenance immunotherapy followed by two doses of 0.3 mL of 1:1,000 concentration epinephrine intramuscularly.

Results: Patient was found to be in atrial fibrillation along with systemic symptoms immediately after administration of immunotherapy. After epinephrine administration, patient converted back to normal sinus rhythm and systemic symptoms resolved.

Conclusions: Atrial fibrillation is an atypical presentation of a systemic reaction to immunotherapy.

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INTRODUCTION

Electrographic changes after insect stings have been reported both with and without systemic symptoms.^{1,2} To our knowledge, however, there have been no reported cases of arrhythmias in the maintenance phase of venom or pollen immunotherapy. We report a case of a patient who developed paroxysmal atrial fibrillation after receiving venom and pollen immunotherapy.

CASE REPORT

A 36-year-old man sought allergic evaluation after two episodes of anaphylaxis. Three years before his initial evaluation, within minutes of an insect sting occurring while mowing his lawn, he experienced acute angioedema of his lower extremities, nausea, generalized hives, and blurred vision. The patient called the paramedics, received diphenhydramine (Benadryl, Pfizer Consumer Healthcare, Morris Plains, NJ) in the field, and was immediately transported to the emergency room where his symptoms resolved without further treatment. Again, 1 month later while mowing the grass on his Illinois farm, he acutely developed similar symptoms including generalized pruritus, hives, laryngeal swelling, and dizziness after an insect sting. He again required diphenhydramine in the field and a subsequent visit to the emergency room for

observation. The patient was then referred for a complete allergic evaluation.

He had no history of serious illnesses or allergies. He was taking no medication and had no known drug allergies. He had no risk factors for cardiovascular disease. The patient did have an atopic family history. Radioallergosorbent testing performed by the primary care physician revealed elevated specific immunoglobulin (Ig)E titer to selected trees, grasses, molds, and ragweeds. Percutaneous skin testing was positive to yellow jacket, yellow hornet, and white-faced hornet venom. He was started on the corresponding immunotherapy to both inhalent allergens and mixed Vespid venom.

During the first 6 months of the build-up phase, he developed mild systemic symptoms to immunotherapy on two occasions. On each occasion, however, after subsequent reduction in extract concentrations, immunotherapy was resumed without incident.

On the day in question, the patient received a 0.05-mL injection of 1:1,000 concentration of mixed aeroallergen and 1.0 mL of 100 µg/mL/insect-mixed Vespid allergen. Within less than a minute, he became acutely diaphoretic, dyspneic, and complained of intestinal cramping and palpitations. Physical examination revealed a blood pressure of 70/palpable and an electrocardiogram at the time revealed the patient to be in atrial fibrillation with a rate of 98 beats per minute (Fig 1). There were occasional premature ventricular contractions with nonspecific ST-T wave changes in the inferior leads. The patient was immediately given two doses of 0.3 mL of 1:1,000 concentration epinephrine intramuscularly. Shortly

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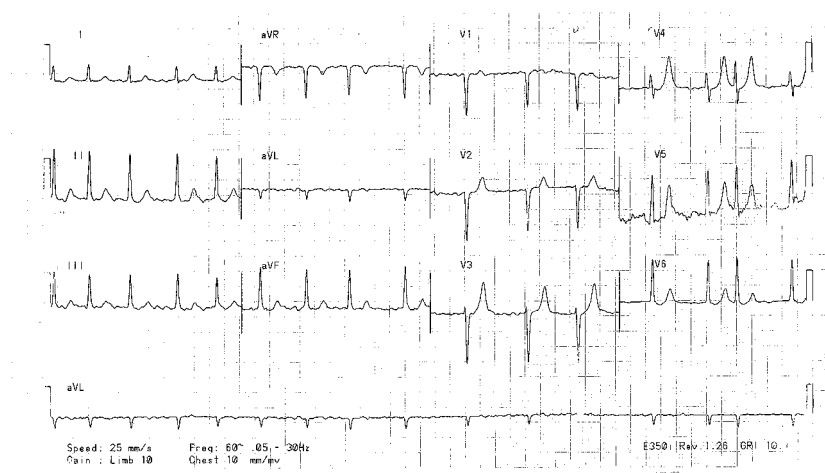


Figure 1. Electrocardiogram 1.

thereafter, the patient's symptoms resolved. He was no longer dyspneic, his blood pressure was 110/70 mm Hg, and a repeat electrocardiogram revealed the patient to be in normal sinus rhythm with a rate of 75 beats per minute (Fig 2). Subsequently, a complete evaluation for underlying predisposition for atrial fibrillation was ruled out. Specifically, an echocardiogram, thyroid function test, and an exercise stress test were all normal. Immunotherapy was not resumed. Followup for 4 years revealed no further episodes of atrial fibrillation. Repeat radioallergosorbent tests or skin tests were not performed.

DISCUSSION

Cardiac involvement during anaphylaxis is rare, especially in patients without underlying cardiac disease.³ As stated previously, there have been no reported cases of cardiac arrhythmias from specifically maintenance immunotherapy. However, there have been reported cases of atrial fibrillation in the course of an anaphylactic reaction to insect stings. Mueller⁴ reported one patient with atrial fibrillation in his review of 1,463 cases of systemic responses to insect stings. Brasher

and Sanchez⁵ reported a case of pulmonary edema, atrial fibrillation, and extreme ST-T segment depressions in a man after a wasp sting.

The immediate reversal of the cardiac arrhythmia after epinephrine administration in this case suggests a direct relationship between the atrial fibrillation and the anaphylactic response. Extensive followup cardiac testing in the patient, including repeat electrocardiograms, Holter monitor analysis, and exercise stress testing revealed no preexisting heart disease, nor clear demonstrable predisposition for atrial fibrillation. The patient has had no subsequent episodes of insect stings or atrial fibrillation after cessation of immunotherapy.

A review of the literature reveals references to arrhythmias, conduction abnormalities, and evidence of myocardial ischemia observed in the electrocardiogram in both human and experimental IgE-mediated anaphylaxis. The effects of histamine and leukotrienes on the heart remain unclear. Histamine and leukotrienes have been classically responsible for bronchospasm, capillary dilatation, urticaria, angioedema, and subsequent circulatory collapse during a systemic allergic response. However, these same chemicals may also contrib-

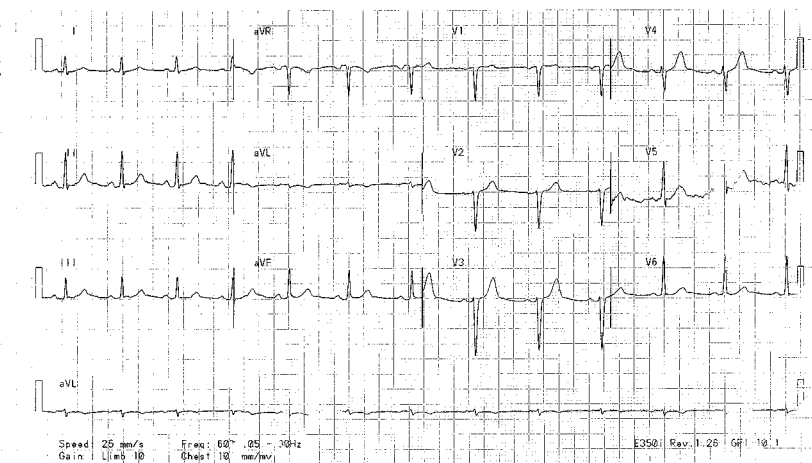


Figure 2. Electrocardiogram 2.

ute to cardiac conduction abnormalities. Aurep et al⁶ demonstrated in vitro the effects of histamine in an isolated perfused heart. They showed that histamine initiated a tachycardia, an increase in contractility, disturbances in conduction, and more specifically atrial fibrillation.

The effects on the heart during an anaphylactic response can be complex and numerous. Booth and Patterson⁷ reported ST-T wave changes suggestive of cardiac ischemia during anaphylaxis. They mentioned that possible etiologies include: 1) a direct antigen-antibody myocardial reaction; 2) a pharmacologic effect of mediators such as histamine released during anaphylaxis; 3) the effects of agents such as epinephrine used for treatment; 4) anoxia; 5) preexisting heart disease; and 6) other unknown factors or a combination of several factors. In several instances, electrocardiogram changes in anaphylaxis have occurred after administration of epinephrine. Levine⁸ reported electrocardiogram changes in two cases. In the first case, there was initial hypotension and respiratory compromise with a normal electrocardiogram. However, shortly after epinephrine was given, the electrocardiogram revealed significant ST-T segment elevations. In the second case, a patient first developed urticaria, hypotension, and loss of consciousness after an insect sting. An electrocardiogram revealed tachycardia with premature ventricular contractions. However, after two doses of epinephrine, the patient was found to have developed atrial fibrillation.

There are numerous mediators during anaphylaxis that can be potentially toxic to the human heart. Consequently, as Wasserman⁹ previously noted, the role of hypotension, hypoxia, epinephrine, and many substances released during an

Ig-E mediated anaphylactic reaction make direct cardiac anaphylaxis difficult to evaluate.

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