

Paroxysmal atrial fibrillation after insect sting

Sergio Ferrari, MD,^a Antonio Pietroiusti, MD,^b Andrea Galanti, MD,^a
Mario Compagnucci, MD,^a and Luigi Fontana, MD^a *Rome, Italy*

Cardiac involvement during anaphylaxis after insect sting is an exceedingly rare event, although major complications such as arrhythmias, myocardial infarction, and angina have been reported.¹ Because these complications are almost exclusively found in patients with preexisting cardiac disease or after the administration of drugs with potentially adverse effects on the heart, it is still unclear whether cardiac function may be affected as a consequence of conditions associated with anaphylaxis or whether cardiac anaphylaxis may have a primary role.¹

We report the case of a young man, free of any cardiac disease, who experienced paroxysmal atrial fibrillation after insect sting. No factor other than cardiac anaphylaxis was convincingly associated with the cardiac arrhythmia.

CASE REPORT

A 38-year-old man without risk factors for cardiovascular disease experienced within 5 minutes of a wasp sting to the ear lobe vomiting, loss of consciousness, sphincter incontinence, and generalized seizures. In the past he had had two wasp stings with large local reactions (according to Mueller²). Fifteen minutes after the sting, he was visited by a general practitioner who found marked hypotension (systolic, 60 mm Hg; diastolic, undetectable), a faint radial pulse, a chaotic cardiac rhythm with a frequency of 110 beats/min, dyspnea, cyanosis, and loss of consciousness. Because epinephrine was not available to the general practitioner, he opted for the rapid administration of 500 ml of saline solution and 1 gm of hydrocortisone intravenously, maintaining the patient in Trendelenburg's position. After 15 minutes, vital signs improved, the patient became alert and oriented, and systolic pressure rose to

80 mm Hg and diastolic to 40 mm Hg, although cardiac arrhythmia persisted. At the time of hospitalization, the cardiac arrhythmia was documented as being due to atrial fibrillation and persisted unmodified during the first 4 hours of hospitalization in spite of the normalization of all vital signs. During the first 4 hours of hospitalization, systolic arterial pressure ranged between 100 and 120 mm Hg; diastolic pressure, between 65 and 80 mm Hg; cardiac frequency, between 90 and 115 beats/min; and breath frequency, between 16 and 20 per minute. At this time, the administration of chlorpheniramine maleate was planned. Other therapeutic options, such as epinephrine, were discarded because of the absence of any signs of generalized anaphylaxis and because of some concern for the potential arrhythmogenic effect of the drug. Other reasonable alternatives, such as the administration of classic antiarrhythmic drugs, seemed less attractive than the administration of an H1-receptor antagonist, given the peculiar clinical context in which atrial fibrillation developed. After 10 mg of chlorpheniramine maleate was administered intravenously, a prompt return to sinus rhythm was observed (Fig. 1). During hospitalization, results of biochemical analyses (including tests of thyroid function) were in the normal range, with the exception of muscle enzymes (creatinine kinase, 2005 IU/L [normal, 22 to 270 IU/L]; lactate dehydrogenase, 594 IU/L [normal, 266 to 500 IU/L]; aspartate transaminase, 98 IU/L [normal, 10 to 42 IU/L], which returned to normal within 5 days. The CK-MB fraction was repeatedly normal.

The assessment of cardiac function and structure by means of Holter examination, Doppler echocardiography, exercise electrocardiogram, and chest roentgenogram did not reveal any abnormality. High IgE level for wasp venom and a marked positive reaction to the prick test were observed. For this reason, the patient underwent a desensitization program.

DISCUSSION

Cardiac involvement during anaphylaxis is rare and is detected almost exclusively in patients with cardiac disease.¹ In particular, atrial fibrillation in the course of an allergic reaction to insect sting is very unusual. This complication has not been reported in a recent extensive review on this subject.³ To our knowledge, only three cases have been reported. Mueller,² in his review of 1463 patients with allergic reactions to insect sting, reports only one case with atrial fibrillation. In the

From ^aCattedra di Immunologia clinica ed Allergologia Università degli Studi di Roma, "Tor Vergata"; and ^bCattedra di Semeiotica e Metodologia clinica Università degli Studi di Roma, "Tor Vergata".

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Reprint requests: Luigi Fontana, MD, Cattedra di Immunologia clinica ed Allergologia, Ospedale S. Eugenio, Piazzale dell'Umanesimo 10, 00144 Rome, Italy.

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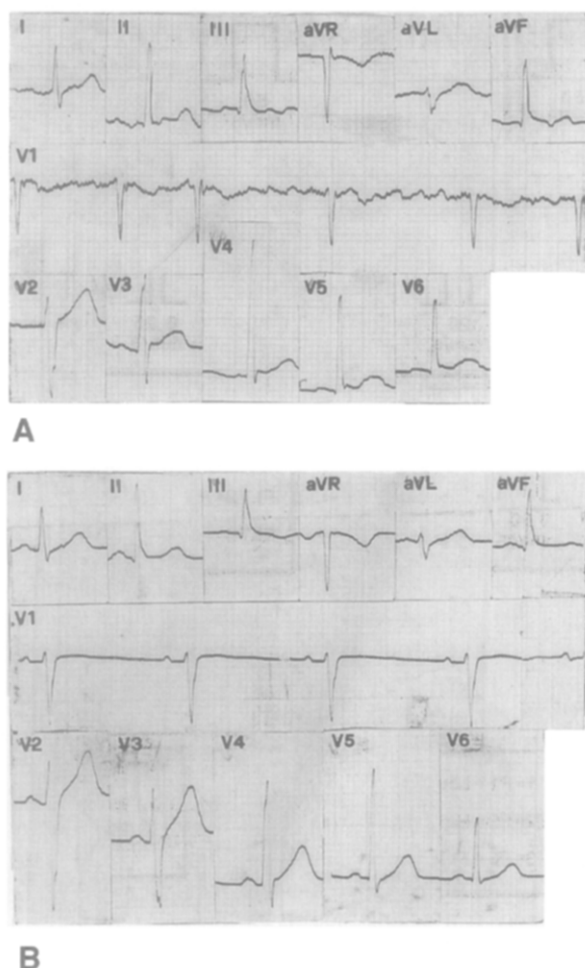


FIG. 1. Electrocardiographic changes after wasp sting. **A**, Tracing obtained on arrival at the hospital: rhythm is atrial fibrillation. **B**, Tracing obtained approximately 4 hours later, immediately after the intravenous injection of 10 mg of chlorpheniramine maleate, shows reversion to normal sinus rhythm.

other two cases reported by other authors, atrial fibrillation followed the administration of potentially arrhythmogenic drugs.¹ It is still a matter of controversy whether cardiac involvement is due to the mediators of cardiac anaphylaxis or to factors associated with anaphylaxis (e.g., hypoxia, hypotension, the administration of drugs for treatment of shock) or to a direct cardiac effect of venom components. Our case is peculiar because many potential arrhythmogenic factors can be safely excluded. In fact, atrial fibrillation was observed after only one sting, involving a quantity of venom insufficient for a direct arrhythmogenic effect, in the absence of a history of cardiac disease or other predisposing factors for arrhythmia (alcohol, coffee, cocaine, hyperthyroidism, drugs). Furthermore, the presence of any cardiac disease, and in

particular of myocardial ischemia, was excluded by clinical examination, biochemical analyses, and instrumental evaluation. The appearance of atrial fibrillation before any medical treatment and its persistence after stabilization of vital signs suggest that cardiac anaphylaxis played a major role in the development of arrhythmia in this case. It is possible that a direct arrhythmogenic effect was induced by the release of histamine, leukotriene, platelet activating factor, prostaglandins, and other mediators by activated cardiac mast cells.² Such an adverse effect on cardiac rhythm may be amplified by endogenous catecholamines, which are released in response to hypotension.

In spite of a strict temporal association, the reversal of atrial fibrillation after the administration of chlorpheniramine does not necessarily imply a cause-effect relationship. It is in fact difficult to postulate ongoing histamine-induced anaphylaxis more than 4 hours after insect sting, although it is possible that cardiac anaphylaxis caused by histamine triggered other mechanisms (e.g., fine regional alterations in coronary blood flow), which in turn may be responsible for the sustained release of histamine by the cardiac mast cells. In fact, in addition to the classic IgE-mediated stimulation of mast cells, several histamine-releasing factors have been recently described.⁴ In this light, it is of interest that in a recent experimental model of ischemia-reperfusion cardiac injury, the administration of chlorpheniramine totally inhibited reperfusion arrhythmias.⁵ The administration of H₂-receptor antagonists was not considered in the patient under discussion. In retrospect, however, it would have been interesting to have tried the administration of these drugs in this case because H₂ antagonism is, theoretically, more attractive than H₁ antihistaminic drugs for the treatment of atrial fibrillation in the context of cardiac anaphylaxis. In fact, H₂ antagonism may affect sinus and other tachyarrhythmias, ectopic beats, re-entrant rhythms, and some instances of atrioventricular conduction delay; whereas H₁ antagonists prevent slowing of atrioventricular conduction and inhibit ventricular fibrillation.¹

In conclusion, this case report suggests that cardiac anaphylaxis may develop during an allergic reaction after insect sting. Its contribution to the development and persistence of cardiac reactions may be separated from that of hypoxia, hypotension, and adverse drug reactions. In these cases a rationale exists for the early administration of antihistamine drugs. However, the drug of choice (chlorpheniramine, cimetidine, or antiarrhyth-

mics) for the treatment of atrial fibrillation in our patient was not determined by this study.

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