

Brief communications

Occupational allergy caused by urticating hair of Brazilian spider

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In 1812, Von Langsdorf, a Russian diplomat in Brazil and a naturalist, described a strange inflammatory reaction caused by the manipulation of a spider of the *Theraphosidae* genus.¹ Almost 160 years later, Cooke et al.^{2,3} identified the so-called "urticating hair" as being responsible for the inflammatory manifestation.^{2,4} Today it is known that the urticating hair is located on a plate on the back of the spider's abdomen at a density of around 10,000 hairs per square millimeter. Four types of hair have been described, varying from 0.06 to 1.5 mm² in length.^{2,4} When threatened, these spiders raise their forelegs and provoke a rapid vibration in their bodies, thereby scattering the hair into the environment. When inhaled by small mammals, the hair causes a severe inflammatory reaction of the respiratory tract, leading to asphyxiation.²

Similar symptoms have been observed recently in employees of the Arthropods Laboratory of the São Paulo Butantan Institute, who manipulate these spiders almost daily. The clinical manifestations ranged from local skin eruptions to systemic symptoms. After continuous contact with the spiders for about 1½ years, the clinical conditions of most patients deteriorated, suggesting the existence of a period of sensitization. In this study we also investigated the possible involvement of an IgE-mediated hypersensitivity reaction in the spider-induced inflammation.

METHODS

Patients

Nine female patients, aged 28 to 56 years, working in the Poisonous Arthropod Laboratory of the Butantan

Abbreviation used

PBS: Phosphate-buffered saline

Institute in São Paulo, Brazil, were included in the study. The period of exposure to the spiders' urticating hair ranged from 1.5 to 25 years (Table I). Ten individuals with no history of exposure to spiders served as a control group. The study was approved by the Institutional Review Board, and all subjects gave informed consent to participate.

Preparation of urticating hair extracts

Urticating hair (100 mg) was collected from *Theraphosidae* spiders by shaving, suspended in buffer, centrifuged twice for 10 minutes at 2500 g, and sonicated. After filtration through a 0.22 µm Millipore filter (Millipore Corp., Bedford, Mass.), protein concentration was determined by the method of Lowry.⁵

Skin tests

Prick and intradermal tests were performed with urticating hair extracts at concentrations of 2 mg/ml and 1 mg/ml, respectively. Histamine phosphate (1 mg/ml) was used as a positive control, and the diluent buffer of the extract was used as a negative control. The tests were evaluated after 20 minutes for wheal size and the presence of erythema.

Determination of total and allergen-specific IgE

Total IgE was measured by ELISA Phadezym IgE PRIST (Pharmacia, Upsala, Sweden). The assay for urticating hair-specific IgE was performed as described above. A polystyrene 96-well microtiter plate (Nunc, Roskilde, Denmark) was coated with allergen extract (50 µg) at a protein concentration of 200 µg/ml in phosphate-buffered saline (PBS). After overnight incubation at 4° C, the plates were washed (PBS, 0.05% Tween-20) and blocked with PBS, 1% bovine serum albumin (Sigma, St. Louis, Mo.) for 1 hour at room temperature. After washing, 50 µl serum (1:5 in PBS, 0.5% Tween) was added to the wells, and the plates were incubated with peroxidase-conjugated anti-IgE antibody (Organon Teknika, West Chester, Pa.) overnight at 4° C. O-phe-

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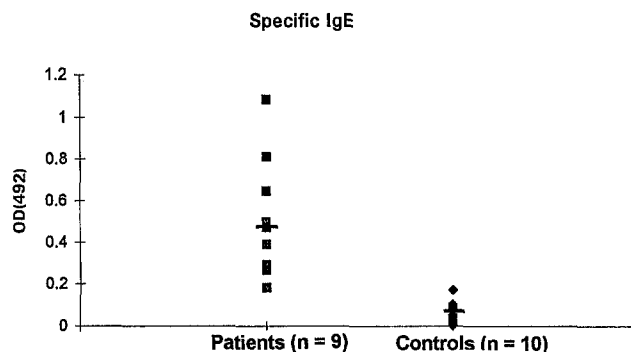


FIG. 1. Specific IgE. Patient group ($n = 9$) compared with control group ($n = 10$). The titers of allergen-specific IgE were significantly higher ($p < 0.01$) in the patient group.

TABLE I. Patient characterization

Patient No.	Age (yr)	Duration of exposure (yr)	Intensity of symptoms	Prick test result	Total IgE (KU/L)
1	28	1.5	Moderate	+++	53.9
2	41	3	Minor	(-)	23.1
3	36	4	Severe	++++	220.0
4	33	8	Severe	++++	192.0
5	46	25	Severe	++++	137.0
6	45	22	Minor	(-)	>1000.0
7	31	8	Moderate	++	26.4
8	56	6	Minor	(-)	22.0
9	41	6	Minor	(-)	302.5

nylenediamine was used as substrate. The enzymatic reaction was allowed to develop for 15 minutes and then stopped with 50 μ l of 2 mmol/L H_2SO_4 , and absorbance was measured at 492 nm (Microelisa Reader, Organon Teknika).

IgE inhibition test

To further determine the presence of specific IgE to urticating hair, patient sera with a high level of specific IgE were incubated (30 minutes at 37° C) with different concentrations of the allergen (0, 1.0, 2.0, 4.0, 8.0, and 10.0 μ g/ml). Nonbound IgE was determined by ELISA, as described above.

Statistical analysis

Data were analyzed statistically by the Mann-Whitney U test. Differences were considered to be significant at a p value of less than 0.05.

RESULTS

The patients had the following symptoms in chronologic order: skin irritation, pruritus, coryza, sneezing, conjunctivitis, urticaria, and angioedema. Five patients experienced moderate or

severe symptoms on exposure to the spiders. The average duration of exposure to the allergen (urticating hair) was 9 years, ranging from 1.5 to 25 years. The results of the skin and intradermal tests of all patients were related to the intensity of the clinical symptoms. Patient 4 experienced urticaria immediately after intradermal application of the allergen and was treated with antihistamine drugs.

Total IgE was slightly elevated in the patients with severe symptoms. As shown in Fig. 1, the titers of allergen-specific IgE were significantly higher in the patient group than those in the control group ($p < 0.001$). This was confirmed by the IgE-inhibition test preincubation of patient serum with increasing concentrations of allergen, which led to a reduction of specific IgE levels to almost normal values (Fig. 2).

DISCUSSION

Little is known about the clinical manifestations of allergy after contact with spiders. The only published information available is restricted to clin-

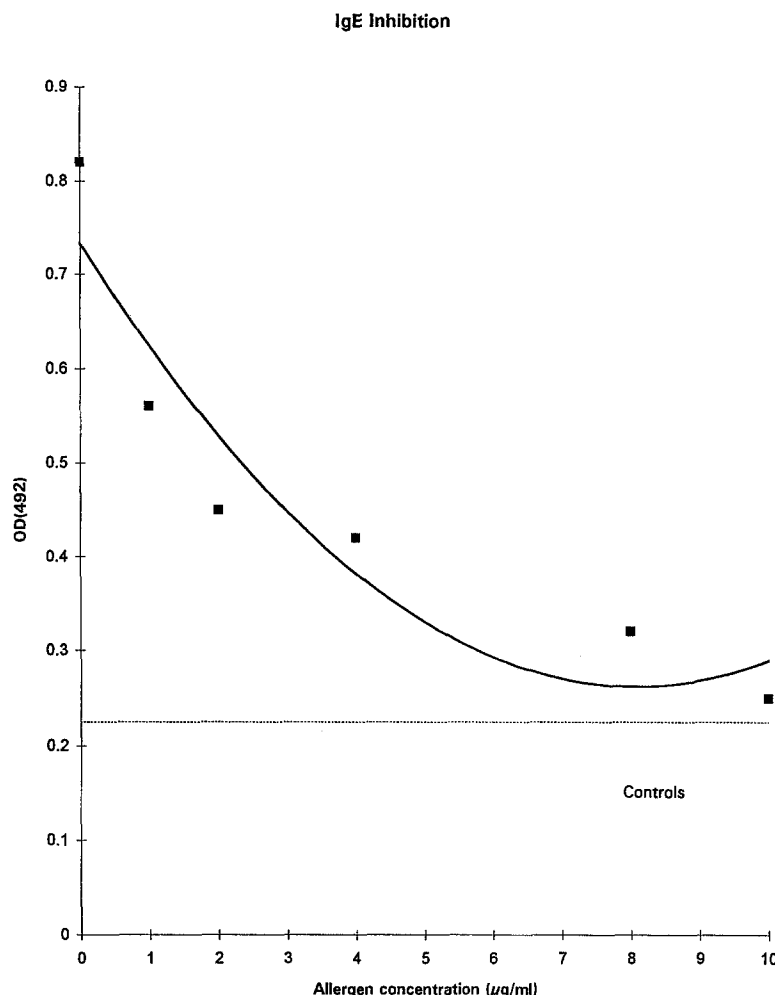


FIG. 2. IgE inhibition. Patient sera with high levels of specific IgE were incubated with different concentrations of the allergen (0, 1.0, 2.0, 4.0, 8.0, and 10.0 µg/ml).

ical symptoms and the classification of different types of hair from these spiders.²⁻⁴ Toxic components and mechanical irritation have been implicated in provoking local inflammation. In our studies we observed that most individuals had a slight local skin reaction on the first contact with the spiders. Because this reaction intensified after a period of approximately 1 year of direct or indirect exposure to these spiders, we suggest the existence of a sensitization period, characteristic of hypersensitivity reactions.

The typical symptoms, varying only in intensity, argue in favor of a hypersensitivity reaction mediated by IgE. The latency period until the onset and the variation in the intensity of the clinical manifestations conflict with the mechanical-irritating phenomenon or a toxic reaction as underlying mechanisms.

Because the results of skin tests performed in the control group were negative, the presence of a

skin-irritating toxic substance in the hair extract was excluded.

The significant difference in plasma concentrations of specific IgE antibodies between patients and control subjects and the correlation of high specific IgE titers with clinical symptoms confirmed the involvement of the IgE-mediated hypersensitivity reaction. The dose-dependent absorbance of allergen-specific IgE proved the specificity of the newly developed ELISA.

Conclusion

For the first time it was demonstrated that the urticating hair of *Theraphosidae* spiders is a potent allergen if it is inhaled or comes in contact with the skin. Our results suggest that besides the mechanical-irritating phenomenon, an IgE-mediated mechanism of hypersensitivity is also involved in the inflammatory process.

Although the allergic reaction to urticating hair may be of little significance for the general population, it is a subject of great importance to the researchers and personnel dealing with these spiders.

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Fatal asthma attack during an inhalation challenge with ultrasonically nebulized distilled water

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CASE REPORT

A 22-year-old man had a history of asthma that was well controlled by regular treatment with inhaled steroids and β -agonists, until a fatal attack occurred during an inhalation challenge in the laboratory with ultrasonically nebulized distilled water.

The medical history included bronchial asthma from the age of 10 years and positive skin test results for mites. The family history was positive for allergic asthma. Immunotherapy for mites was performed from the age of 16 to the age of 20 but did not provide significant benefits. The patient had no known drug sensitivities, history of aspirin-induced asthma, or food

allergy. He had never been hospitalized for treatment of severe asthma attacks. He had been treated irregularly with inhaled beclomethasone and fenoterol. In April 1992 he started regular therapy with inhaled beclomethasone 500 μ g twice daily, inhaled salmeterol 50 μ g twice daily, and fenoterol on an as-needed basis.

In June 1992, he was referred to the pulmonary function laboratory for assessment of the degree of airway responsiveness to ultrasonically nebulized distilled water. At that time he was free of symptoms, and his baseline spirometry demonstrated normal lung volumes (forced expiratory volume in 1 second = 85% of predicted value and forced expiratory volume in 1 second/vital capacity = 88%). After spirometry was performed, he was invited to inhale ultrasonically nebulized distilled water through a rubber face mask. One minute after the beginning of the inhalation, a severe asthma attack occurred and progressed so rapidly that it could not be reversed by prompt treatment with salbutamol 10 puffs, oxygen therapy, methylprednisolone 200 mg administered intravenously, epinephrine 2 mg administered subcutaneously, and mechanical ventilation. During the treatment he also had an episode of vomiting. Within 15 minutes of the beginning of the attack the patient was dead.

At autopsy, macroscopic examination excluded cerebral or cardiovascular causes of death. The lungs were overinflated, and the cut surface showed mucus plugs in

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