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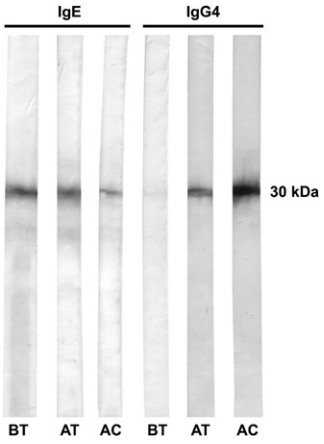

FIG 1. Western blotting of the patients' sera before *(BT)* and immediately after *(AT)* sublingual immunotherapy compared with a sample taken after 5 years of continuous kiwi consumption and a subsequent cessation for 4 months *(AC)*. Shown are the IgE reactivity *(left)* and the IgG^4 reactivity *(right)* to the major kiwi allergen Act c 1 (30 kd).

Life-threatening anaphylaxis to kiwi fruit: Protective sublingual allergen immunotherapy effect persists even after discontinuation

To the Editor:

We previously reported the case of a 29-year-old woman with several episodes of severe anaphylaxis after consumption of kiwi fruit, including 3 episodes of allergic shock with loss of consciousness and hospitalization, who was subsequently treated with a modified sublingual immunotherapy (SLIT) protocol enabling her to ingest substantial amounts of kiwi without symptoms.[1]

Sublingual immunotherapy has been shown to reduce clinical symptoms in a variety of IgE-mediated respiratory allergic diseases (see review[2]),[3,4] but its therapeutic value in anaphylactic food allergy is repeatedly questioned because of the lack of standardized protocols and the great variability in allergen uptake by custom-made protocols. While information is already scarce concerning possible indication for this treatment, almost no information exists on the possible duration of the protective/beneficial effect in patients undergoing SLIT in food-associated anaphylaxis.[5]

We had initiated the successful SLIT in our patient in 2001 by starting with a dose of 100 μL 10^{-4} diluted stock solution of fresh kiwi pulps (concentration of the stock solution, 1 mg/mL) and a gradual increase (100-500-1000 μL) until the next concentration was reached. The patient was advised to keep the kiwi extract under her tongue for 1 minute before swallowing. This regimen was kept until 1 mL undiluted kiwi extract was tolerated. Thereafter, a 1 cm^3 cube of fresh or frozen kiwi was given without symptoms.

The patient was then followed at regular intervals and was advised to consume this dose (1 cm^3 kiwi) using an identical technique (sublingual application for 1 minute before swallowing) every day. No further immunotherapies against concomitantly diagnosed sensitizations to grass pollen, birch pollen, latex, or crab meat[1] were performed.

Because allergic reactions to food in adults (best documented for peanut allergy), unlike reactions to milk and egg in children, do not tend to undergo spontaneous resolution,[6] we decided to recommend a lifelong continuation of this treatment in an identical manner.

However, because of a severe tonsillitis with pharyngeal abscess and subsequent tonsillectomy in February 2006, the patient stopped the SLIT for a period of 4 months on her ear, nose, and throat doctor's advice. After that, the patient wished to resume the immunotherapy, because she had experienced a high degree of safety under SLIT when consuming food of unknown source. At this point, skin prick test, a labial application test with diluted fresh kiwi extract, and an oral provocation with fresh kiwi were performed. The skin prick test still showed positive results, which were, however, decreased compared with pretreatment values (5/14 instead of 10/30 mm wheal/mm flare by using a 1:10 dilution of the kiwi extract stock solution). The labial application test elicited negative results, and the oral provocation was tolerated well without any adverse reactions at a dose of a 1 cm^3 cube of fresh kiwi. Thus, our findings at this point were still in accordance with a persisting state of tolerance, most probably because of the ongoing effect of a successful SLIT.

The clinical results were also mirrored in specific IgE testing by the Immulite system (DPC Biermann, Bad Nauheim, Germany) and by Western blotting with kiwi extract. The amount of kiwi specific IgE had dropped from70.4 kU/L after treatment to 34.6 kU/L. In Western blots with kiwi extracts, we found a clearly diminished IgE-reactivity to the dominant kiwi allergen Act c 1 (30 kd), which was strongly positive before and immediately after SLIT, together with a substantial increase in IgG_4 reactivity to the same protein (Fig 1). Thus, the patient's

serum showed all criteria for a persisting state of tolerance even after the cessation of kiwi intake.

In consequence of our findings and the patient's personal intention for a high degree of safety, we advised her to resume the SLIT protocol without modifications (1 cm^3 kiwi cube sublingually for 1 minute before swallowing).

In summary, this case should encourage the consideration of SLIT protocols in patients with severe food allergies, especially in cases in which the foods are difficult to avoid. Even after accidental interruptions, the beneficial effects seem to endure. Experiences in larger collectives such as reported for hazelnut allergens[7] should help to evaluate the findings in our patients and to design protocols for immunotherapy strategies in patients with food allergy.

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Severe refractory atopic dermatitis in adults is highly atopic

To the Editor:

Atopic dermatitis (AD) is a chronic, relapsing inflammatory skin disease affecting both children and adults. Most children have a substantial reduction in the severity of their AD over time or outgrow it altogether. Others continue to have severe disease as teenagers and adults, or experience a relapse during adulthood. The factors that contribute to the persistent severe skin inflammation in these individuals are unknown.

We performed a retrospective chart review of all patients older than 18 years who were treated for AD at National Jewish Medical and Research Center in Denver between 2000 and 2006. Patients were assigned a severity score based on the Rajka and Langeland Criteria[1] evaluating the extent of involvement, the amount of time of remission per year, and the intensity of itch.

Forty adults were found to have severe AD by these criteria. These patients had poorly controlled eczema despite the use of topical and oral corticosteroids, topical calcineurin inhibitors, antibiotics, and antihistamines. They were compared with adults in 2 other groups: those with quiescent AD or moderate AD. The quiescent AD group consisted of patients with a history of AD in the past, but no recent skin disease. The moderate AD group had active disease responsive to topical treatment.

As shown in Table I, the median serum IgE value in the severe AD group was 3922 IU/mL, compared with 44 IU/mL for quiescent AD and 358 IU/mL for moderate AD ($P < .0001$ and .005, respectively.)

As shown in Table II, the severe AD group had increased perennial allergen sensitivity compared with the moderate AD group (dust mite, $P = .005$; molds, $P = .004$). The majority of patients in the severe AD group lived outside Colorado (28/38), most frequently in humid climates such as Florida (6/38), Texas, Oklahoma, California, and Massachusetts (each 2/38). The majority of patients with moderate AD lived in Colorado (16/19). There was also a significantly higher rate of food allergen sensitization by skin testing in the severe AD group compared with the moderate AD group ($P = .007$). The most common positive foods, by skin prick testing, in the severe AD group were peanut (11/35), egg (9/35), shellfish (7/35), soy (7/35), and milk (6/35).

Of the patients in the severe refractory AD group, 25 of 36 (69%) reported a history of asthma, compared with 16 of 23 (70%) in the moderate AD group ($P = 1.0$). In addition, 34 of 37 (92%) in the severe AD group had a history of allergic rhinitis, as did 22 of 23 (97%) in the moderate AD group ($P = 1.0$) There was no difference in family history of atopic disease ($P = .74$) between these 2 groups. The age of onset of AD was not significantly different in the 2 groups, with most having early childhood onset. Of the severe AD group, 4 of 38 (10%) had onset of AD during adolescence or adulthood, compared with 4 of 23 (17%) in the moderate group ($P = .46$).

In our population, severe refractory AD is primarily an atopic disease characterized by increased levels of total serum IgE and positive skin tests to dust mites, molds, and food allergens, compared with patients with more moderate disease. Further studies are needed to determine whether severe, poorly controlled AD may be a result of polyallergen sensitivities, and the inability of these individuals to remove allergenic triggers from their indoor