

Correspondence

Oral allergy syndrome?

To the Editor:

In the November 1994 issue of the JOURNAL, Liccardi and D'Amato (J ALLERGY CLIN IMMUNOL 1994; 94:931-2) describe a patient who does indeed have egg allergy as evidenced by a positive history of immediate-type allergy symptoms after ingestion of egg or egg-containing foods, positive immediate-type skin test results and IgE immunoassays for egg components, and even by a positive double-blind, placebo-controlled food challenge with egg. Because most of the reactions involved only oral symptoms, the authors have described this patient's egg allergy as oral allergy syndrome. However, the patient did have one episode that also involved hypotension.

I believe it is more appropriate to say that this is a patient with egg allergy (quite common) whose symptoms are usually confined to the mouth (somewhat unusual). I would not call this oral allergy syndrome because I believe that designation should be reserved for patients who have itching of the mouth and throat, and rarely other symptoms, after the ingestion of fruits and vegetables (usually fresh) and sometimes nuts, and who also have pollinosis. This phenomenon is almost certainly caused by cross-reacting allergens (e.g., profilins) found in the foods and pollens. Not only does limiting the use of the term *oral allergy syndrome* to this group of patients help with semantics, it has some impact on treatment. Because oral allergy syndrome symptoms are usually confined to oral itching, many patients with this condition can safely consume these foods. Egg allergy, which is much more often associated with potentially life-threatening reactions, warrants a more cautious approach to treatment, often involving strict avoidance and carrying self-injectable epinephrine.

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Reply

To the Editor:

This is in response to Dr. Kelso's letter (Oral allergy syndrome?) regarding our article published in the November 1994 issue of the JOURNAL.

Various considerations can be produced to demonstrate that our case report suggests the diagnosis of a "true" oral allergy syndrome (OAS) and not an egg allergy in which symptoms are confined to the mouth.

In our article we specified that the anamnestic term *hypotension* used by parents could be inappropriate, because they indicate with this term a nonspecific discomfort (which is commonly observed in early infancy also under different clinical conditions). No symptoms of

systemic involvement (cutaneous, gastroenteric, or respiratory) were demonstrated, either in this first episode or in others.

During the 3 years after the first observation, no changes in clinical data were observed: the symptoms of the allergy were always localized to only the mouth and throat, without any systemic manifestation.

Even though fresh fruits and vegetables are usually considered the most common causative agents in patients with pollinosis, theoretically, all foods may induce OAS.¹

Bircher et al.² demonstrated that IgE antibodies to food allergens (particularly fruits and vegetables) are highly prevalent in patients allergic to pollens. For this reason the occurrence of egg-related OAS without pollen sensitization can be considered a very unusual event. In other words, we think OAS is not exclusively associated with ingestion of fruits or vegetables in patients with pollinosis.

We agree with the concept that fruits and vegetables associated with OAS usually produce mild or moderate symptoms and, on the contrary, egg allergy may induce potentially life-threatening reactions. The local symptoms of our patient are in fact very severe, and when they are present, the ingestion of fresh or cooked eggs or foods containing eggs is not possible, even in small quantities.

In conclusion, because the mouth and throat represent the only "target" organ in our patient, we believe that this represents a true and unusual case of OAS.

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Risk of sting anaphylaxis

To the Editor:

The report by van der Linden et al. (J ALLERGY CLIN IMMUNOL 1994;94:151-9) and the accompanying editorial by Lichtenstein (J ALLERGY CLIN IMMUNOL 1994;

94:137-8) seem to raise new issues of "to whom we should offer venom immunotherapy." However, the debate is neither new nor settled. Concerns about "overtreatment" date to the introduction of commercial venom extracts.¹ Some consensus has been reached about "how much risk is too much risk" in that venom immunotherapy is not required for certain low-risk groups. Careful epidemiologic evaluation of the natural history of insect sting allergy has shown that children with cutaneous systemic reactions and all patients with large local reactions have less than 10% risk of sting anaphylaxis. Hopefully, this report does make it possible to counsel adults with limited cutaneous reactions that therapy is not required. However, it has never been possible to find any marker of reactivity that would predict the risk in individual patients, leading to the concept that if the risk is high enough (perhaps 30% to 50%), then therapy is warranted for the group as a whole because we cannot identify the reactors and we do know that we can reduce the risk to less than 2% with venom immunotherapy. Because patients (or parents) who have had one severe reaction are usually very concerned about a repeat reaction, the rule of "once stung, twice shy" has applied, and we recommend therapy to all patients with a history of sting anaphylaxis who continue to have positive skin test results. This recommendation has not really changed. We still, 17 years later, have no marker for systemic reactivity to any allergen (venom, food, penicillin) that has any better than 50% predictive value!

Although food challenge is now recommended as an accepted office procedure,² the clinician still relies most heavily on history and skin test (or RAST) results for diagnosis and prognosis.³ The suggestion that sting challenge be the required prognostic procedure is even less likely to be accepted in clinical practice in this country.⁴

The conclusions in the report by van der Linden et al. leave one major concern: will those who are unreactive to a single sting challenge remain unreactive to future stings? The conclusions of the authors may be overly optimistic, which raised concerns in the editorial. The observation has been made, in the literature, that the response to sting challenge (or field sting) is variable in occurrence but not so much in its pattern or severity.^{5,6} In fact, in a letter in the same issue of the JOURNAL as the report by van der Linden et al., the reaction rate to field stings in untreated patients was reported to be 50%.⁷ The authors present very little evidence on this question. I am reluctant to accept any conclusion without much

more extensive observations because of my ongoing experiences.

Most recently, for example, we have noted in our studies of discontinuing venom immunotherapy that half of the patients who did react to a challenge sting after discontinuing therapy had had no reaction to a previous posttherapy challenge sting 1 to 2 years earlier.⁸ I continue to believe that there are variable intrinsic determinants of clinical reactivity that make any single challenge sting unable to fully predict the risk over a period of years. Again, the interpretation of the results may be a matter of clinical judgment: if 30% to 50% will react and some will get worse, and we cannot tell who is really going to react, then it remains reasonable clinical policy to offer therapy to all patients with a significant history and positive skin test results.

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