
Bee pollen-induced anaphylactic reaction in an unknowingly sensitized subject

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Background: The food supplement bee pollen has been previously found to cause anaphylactic reactions. It has been proposed as useful for “everything from bronchitis to hemorrhoids.”

Objective: This study describes an atopic patient who experienced a non-life-threatening anaphylactic reaction upon her initial ingestion of bee pollen. Microscopic examination of the pollen sample and ELISA inhibition assays were performed.

Results: The patient had a 7 mm/28 mm wheal/erythema reaction to bee pollen at 1 mg/mL concentration. Bee pollen caused 52% inhibition of IgE binding to short ragweed and 55% to ryegrass. Microscopic analysis revealed ragweed, *Alternaria*, *Cladosporium*, honeysuckle (*Lonicera sp*), privet shrub (*Ligustrum sp*), and vetch (*Vicia sativa*).

Conclusions: An unknowingly sensitized atopic patient experienced an anaphylactic reaction after ingestion of a small quantity of bee pollen that contained pollens and fungi. Previously administered allergen immunotherapy that had reduced rhinitis symptoms did not prevent this allergic reaction.

Ann Allergy Asthma Immunol 2001;86:239–242.

INTRODUCTION

Bee pollen is a dietary supplement that has been advertised as a natural nutrient for increased athletic endurance, prevention of memory loss, and treatment of “everything from bronchitis to hemorrhoids.” Proponents refer to its anti-aging properties, because in murine studies, very large doses of bee pollen reduced the presence of lipofuscin in cardiac muscle of aged mice.¹ Bees inadvertently collect pollens as they make contact with flowers or other vegetation. The pollens are carried in a pollen sac located by the bee’s hind legs. Bee keepers can collect this pollen by use of a screen at the entry to the hive.

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Supported by the Ernest S. Bazley Grant to Northwestern Memorial Hospital and Northwestern University Medical School.

Received for publication March 9, 2000.

Accepted for publication in revised form July 27, 2000.

The literature contains two previous reports of four patients who had developed allergic symptoms after oral ingestion of bee pollen.^{2,3} Symptoms began within 15 to 45 minutes with doses from 5 to 15 mL, and ranged from acute conjunctival pruritus and limited urticaria with dyspnea² to flushing, hypotension, and a grand mal seizure.³ Microscopic analysis of two commercial sources of bee pollen contained dandelion (a member of the *Compositae* family), buttercup, and legume pollen, either sweet clover or alfalfa.² A report from Spain noted pollens in 93% and fungi in 6% of the sample.³

The purpose of this report is to describe a mild anaphylactic reaction that occurred after ingestion of bee pollen, results of ELISA inhibition assays, and microscopic analysis of the incriminated bee pollen product.

CASE REPORT

A 56-year-old female patient with perennial allergic rhinitis developed acute-onset palmar pruritus, generalized urticaria, acute dyspnea with throat tightness, and fatigue within 20

minutes of drinking a “smoothie” milk shake. The patient used hydroxyzine 25 mg when she realized an allergic reaction was beginning and the throat tightness resolved completely within 1 hour. The smoothie was made with yogurt, bananas, strawberries, a nut mix, and bee pollen. (The patient stated that she needed extra energy.) Previously, on multiple occasions, the same smoothie without bee pollen had been consumed without adverse reaction. Bee pollen had not been consumed previously. Urticaria lasted 9 hours despite an additional 50 mg of hydroxyzine.

The patient had no known food or nut allergy, or hymenoptera sensitivity. She reported experiencing mild voice change (raspiness) lasting 2 hours after eating fresh but not cooked pineapple. She denied pruritus or urticaria from pineapple. At the time of the reaction, she was receiving maintenance allergen immunotherapy for allergic rhinitis and conjunctivitis with olive and mesquite pollens. Two years before, she had completed a 3-year course of allergen immunotherapy for pollens, molds, and dust mites (Allergy Labs of Ohio, Columbus, OH) with a marked reduction in rhinitis. She had received 1:10,000, 1:1,000, and 1:100 dilutions of a tree mix (cottonwood, white ash, beech, elm, oak, maple, hickory), grass mix (100,000 bioequivalent allergy units/mL of juniper, redtop, orchard, timothy and 10,000 bioequivalent allergy units/mL Bermuda), ragweed (giant, short), dust mites (5000 allergy units (AU)/mL *Dermatophagoides pteronyssinus* and 5000 AU/mL of *Dermatophagoides farinae*), and molds (40% *Alternaria*, 20% *Penicillium*, 20% *Aspergillus*, 20% *Cladosporium*). However, whereas allergic rhinitis and conjunctivitis symptoms decreased

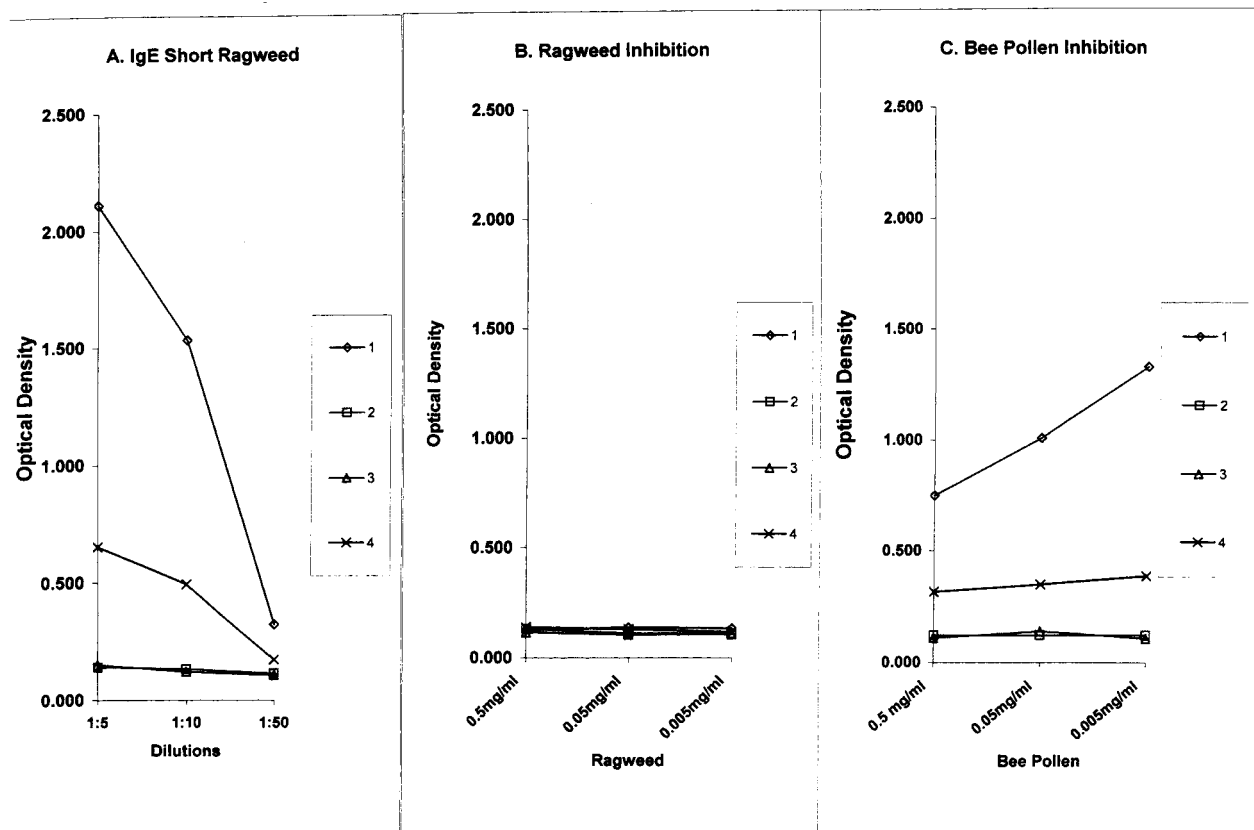


Figure 1. 1A Serum IgE antibodies to short ragweed. Serum 1 is the index patient. Sera 2 and 3 are from nonatopic subjects. Serum 4 is pooled sera from patients with asthma who skin-tested positive to *Aspergillus*. 1B. Inhibition experiment with short ragweed using the serum dilution of 1:10. 1C. Inhibition experiment with bee pollen.

markedly while she was in the Chicago area, she noticed increasing symptoms of rhinitis and conjunctival pruritus while in Tucson, AZ from January to April. Skin testing was carried out for olive and mesquite (Meridian Bio-Medical, Round Rock, TX) and was 3+ positive on prick skin testing. Allergen immunotherapy for olive and mesquite was begun and within a year she was able to tolerate the tree pollination in Tucson with little need for medications. She was at 1:100 wt/vol concentration for each tree.

She had mild asthma that necessitated pharmacotherapy infrequently. Her medication allergies included penicillin (generalized urticaria and dyspnea), sulfonamides (angioedema), and benzalkonium chloride (conjunctival pruritus and injection).

Skin testing revealed prick positive responses to the same bee pollen (1

mg/mL) 7 mm/28 mm wheal/erythema and to elm, blue grass, orchard grass, short and giant ragweed, and cat. She was positive on intradermal testing to

dog dander but negative to mites, molds (*Alternaria*, *Cladosporium*, *Aspergillus*, *Penicillium*), and cockroach. The supplier is Allergy Labs of Co-

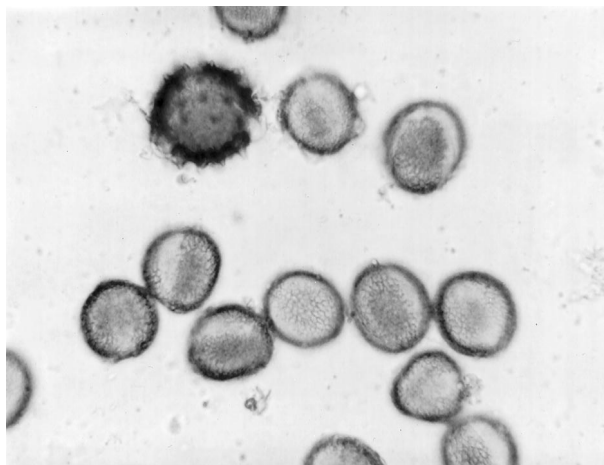


Figure 2. Microscopic analysis of bee pollen showing ragweed pollen.



Figure 3. Microscopic analysis of bee pollen showing an *Alternaria* spore.

lumbus, Ohio except for the bee pollen that was the same batch that the patient ingested and was used in ELISA studies.

She has not experienced urticaria or manifestations of anaphylaxis since the single episode and tolerates milk, strawberries, blueberries, bananas, and various nuts without allergic symptomatology.

METHODS

Inhibition Experiments

ELISAs were established to detect serum IgE to grass and ragweed extracts. Microtiter wells were sensitized overnight at 4° C with either 1000 AU of

short ragweed (ALK America, Milford, CT) or 1000 AU of perennial ryegrass (Miles, Inc., Spokane, WA). After washing three times with PBS 0.05%/Tween, serum dilutions of 1:5, 1:10, and 1:50 were incubated for 60 minutes at 37° C. Next, rabbit antihuman IgE (Accurate Chemical Co., Westbury, NY), 1:5000 dilution, was incubated for 45 minutes, followed by goat antirabbit IgG-alkaline phosphatase (Sigma Chemical Co., St. Louis, MO) at 1:1000 dilution for 60 minutes. *p*-Nitrophenylphosphate (Sigma Diagnostics Inc., St. Louis, MO), 1 mg/mL, was incubated for approximately 30 minutes, and color change was de-

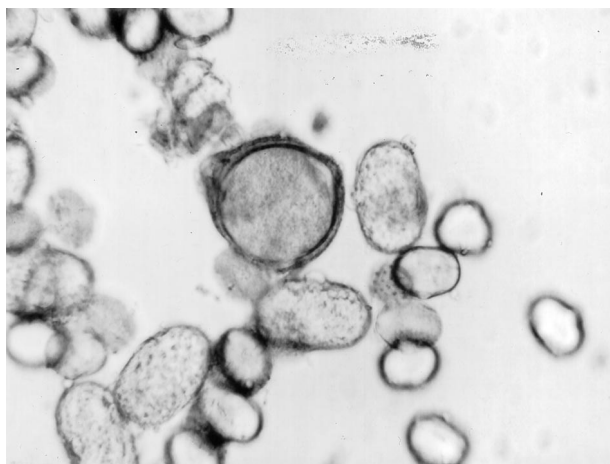


Figure 4. Microscopic analysis of bee pollen showing honeysuckle (*Lonicera* sp), 62.5 μ m at 400 $\times$ magnification.

tected by an ELISA microplate reader (Dynex Technologies, Chantilly, VA). With both the serum IgE-ragweed and IgE-ryegrass assays, a serum dilution of 1:10 was selected for inhibition experiments. Bee pollen (obtained from the same commercially purchased supply that was used in the smoothie) at 0.01, 0.1, and 1 mg/mL was incubated with equal parts of serum diluted 1:10 so that effective inhibitor concentrations were 0.005, 0.05, and 0.5 mg/mL. Similarly, ragweed and ryegrass at the same initial concentrations were incubated with serum in respective experiments. Each assay was performed on four separate determinations.

Microscopic Analysis

The same bee pollen that was ingested by the patient and used in the ELISAs was analyzed microscopically at Greer Laboratories (Lenoir, NC) courtesy of John Richardson.

RESULTS

Figure 1 demonstrates detection of serum IgE-short ragweed and inhibition by bee pollen (52%) and short ragweed (92%). Similar experiments with serum IgE-ryegrass resulted in inhibition by bee pollen (55%) and ryegrass (82%) (results not shown).

Microscopic analysis revealed ragweed (Fig 2), *Alternaria* (Fig 3), honeysuckle (*Lonicera* sp) (Fig 4), *Cladosporium*, privet shrub (*Ligustrum* sp), and vetch (*Vicia sativa*).

DISCUSSION

The first dose of bee pollen in a patient with allergic rhinitis and mild asthma caused a non-life-threatening anaphylactic reaction. This report is the first to demonstrate that the bee pollen sample contained ragweed, grasses, tree pollens, pollen from a vine, and the fungi *Alternaria* and *Cladosporium*. Previously, dandelion pollen was identified in bee pollen and serum IgE antibodies were demonstrated by RAST.² Because dandelion is a member of the *Compositae* family, it was considered a likely cause of anaphylactic reactions in three ragweed-allergic patients.² Patients allergic to ragweed could be ex-

pected to have cross-reacting IgE antibodies to some other members of the *Compositae* family, such as dandelion, chrysanthemum, or sunflower seed that might be ingested as a dietary supplement.² In addition, a report of anaphylaxis occurred after a patient allergic to ragweed drank chamomile tea.⁴

The bee pollen-induced anaphylaxis that occurred in the current patient with allergic rhinitis and intermittent asthma was not prevented by a 3-year course of grass and ragweed immunotherapy that was completed 2 years previously. Because studies of oral immunotherapy as a treatment for allergic diseases continue to be attempted, it is worth noting that oral immunotherapy was administered from 1900 to 1930 and then abandoned by many allergists after a 1940 study of oral versus subcutaneous treatment.^{5,6} Injection therapy was found to reduce symptoms far better than oral pollen immunotherapy, and many patients had no clinical improvement of asthma or allergic rhinitis at all.^{5,6} Pollen injections had a more favorable side effect profile in that, with oral immunotherapy, 33% of

patients developed gastrointestinal symptoms, 7.8% allergic rhinitis, and 3.6% acute asthma.⁶

CONCLUSION

Bee pollen ingestion caused a non-life-threatening anaphylactic reaction in an atopic patient who had responded successfully to a previous course of allergen immunotherapy. The specific bee pollen preparation inhibited IgE binding to short ragweed and ryegrass and, microscopically, was found to contain ragweed, grasses, tree pollens, pollen from a vine, and the fungi *Alternaria* and *Cladosporium*. Although bee pollen has been touted "for everything from bronchitis to hemorrhoids," it can induce anaphylaxis in atopic subjects. We are unable to state which allergen was most responsible for the reaction. This report serves to reemphasize that oral ingestion of seemingly small quantities of pollens or fungi can cause anaphylaxis in unsuspecting but sensitized atopic subjects.

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