
Case report

Buckwheat pillow-induced asthma and allergic rhinitis

Stephen B. Fritz, MD and Bernard L. Gold, MD

Background: Immunoglobulin (Ig)E-mediated hypersensitivity is a mechanism suggested to explain adverse reactions to buckwheat. This is the first reported case in the United States of a person who developed asthma and worsening allergic rhinitis after exposure to a buckwheat pillow.

Objective: To describe a patient who developed asthma and worsening allergic rhinitis after exposure to a buckwheat pillow and to provide evidence that the adverse reaction was IgE-mediated.

Methods: The patient underwent skin prick and ImmunoCAP testing (Pharmacia Diagnostics, Kalamazoo, MI) to buckwheat as well as skin prick testing to several environmental allergens.

Results: The patient showed a 4+ skin prick test response to buckwheat. He also showed 4+ positive skin prick responses to multiple trees, grasses, and weeds, *Alternaria*, *Helminthosporium*, dog, and histamine control and was 3+ positive to house-dust mites, *Penicillium*, *Aspergillus*, cat, and feather mix. His negative control was negative. His ImmunoCAP test for buckwheat-specific IgE was class 4, or strongly positive. He had normal spirometry values. Performance of house-dust mite avoidance measures did not result in improvement of the patient's symptoms. Removal of the patient's two buckwheat pillows resulted in resolution of his asthma and improvement of rhinitis symptoms.

Conclusions: The positive skin prick and ImmunoCAP test to buckwheat along with the positive clinical response to buckwheat pillow elimination support an IgE-mediated mechanism in explaining our patient's buckwheat pillow-induced asthma and allergic rhinitis.

Ann Allergy Asthma Immunol 2003;90:355–358.

INTRODUCTION

Adverse reactions to buckwheat have been reported in the medical literature since 1909 when Henry L. Smith¹ reported the case of a man who developed repetitive emesis and angioedema caused by "buckwheat poisoning." In fact, it was a scarification test performed by Rufus I. Cole on a buckwheat allergy patient that launched the field of diagnostic allergy testing nearly 100 years ago.² Since this time, buckwheat ingestion has been well documented to be responsible for a host of medical conditions including allergic rhinoconjunctivitis, asthma, urticaria, angioedema, and anaphylaxis.^{3–6} Buckwheat has also been demonstrated to cause occupational allergic disease. Bakers have been shown to develop contact urticaria, allergic rhinoconjunctivitis, and asthma related to skin contact and inhalation of buckwheat flour.^{7,8}

We describe a patient with a new diagnosis of asthma and worsening allergic rhinitis after exposure to a buckwheat pillow. The patient exhibited immediate skin test reaction and had strongly positive, ImmunoCAP-specific immunoglobulin (Ig)E (Pharmacia Diagnostics, Kalamazoo, MI) to buckwheat. This patient's allergic asthma and rhinitis resolved

with removal of his buckwheat pillow. We believe this to be the first case report of a person in the United States who developed asthma and worsening allergic rhinitis after exposure to a buckwheat pillow.

CASE REPORT

The patient is a 24-year-old male who originally presented in mid-January, complaining of a persistent nonproductive cough, worsening sneezing, nasal congestion, and rhinorrhea over the preceding 3 months. The patient had previously been seen by his primary care physician, who prescribed a 5-day course of azithromycin, which did not result in improvement in his symptoms. He went on to have a normal chest x-ray and was subsequently evaluated by this same primary care physician with "allergic" rhinitis and asthma, based on his clinical history, physical examination, and the reversibility of his pulmonary symptoms after inhaled bronchodilator treatment. He was treated with loratadine 10 mg orally, daily, triamcinolone acetonide metered-dose inhaler four puffs inhaled, twice daily, and albuterol metered-dose inhaler for as-needed use. On this medical regimen the patient required albuterol use two to three times per week to control his cough. His cough was most pronounced in the nighttime where he would require albuterol six to eight times per month. He noted that his coughing and rhinitis symptoms would improve during long periods away from his apartment.

Division of Allergy and Clinical Immunology, The University of Michigan Medical Center, Ann Arbor, Michigan.

Received for publication September 5, 2002.

Accepted for publication in revised form October 18, 2002.

When he left his apartment for 10 days at Christmas time and again for 10 days during a skiing trip, his albuterol use would decline to zero. Each time he returned from a vacation, his coughing and rhinitis symptoms would return, usually within a few hours of entering his apartment. His cough and rhinitis symptoms also improved during the day while he was at school or at work and away from his apartment.

He denied having consumed any new foods including buckwheat in his diet. He also denied using any new colognes or hairsprays and was on no medications before the beginning of his cough. He did not take any herbal or nontraditional remedies. He also denied having any pets, household mold problems, or leaking water fixtures in his apartment. He resided in an apartment free of insect infestation. He had a dehumidifier present in his apartment. He slept on an inner-spring cotton mattress and a buckwheat pillow, which he had purchased 3 years earlier. He had recently purchased a second identical buckwheat pillow for his girlfriend; the second pillow was also kept in his apartment.

His past medical history was significant for mild perennial rhinitis symptoms which were particularly severe during the spring, summer, and fall seasons. His symptoms were well controlled on as-needed loratadine use, which the patient rarely required during the winter. He denied known triggers for his rhinitis complaints. He denied a history of eczema, bee-sting allergy, conjunctivitis, or previous pulmonary complaints. He also denied a history of food or latex allergies. His family history was significant for a father with allergic rhinitis and eczema and a mother with allergic rhinitis. His social history revealed no current or past alcohol or tobacco use. He worked as a graduate student and denied any new stressors in his life. He denied any current or previous occupational exposure to chemicals or inhalants.

On physical examination he had normal vital signs and pale, mildly edematous nasal mucosa without nasal septal deviation, septal perforation, or nasal polyps. His posterior pharynx demonstrated mild cobblestoning and his sclera was mildly injected. His sinuses were nontender to palpation and his neck was without lymphadenopathy, jugular venous distention, or thyromegaly. His lungs were clear on examination, as was his skin. His heart had a regular rate and rhythm and was free of murmurs, rubs, and gallops. He had no cyanosis, lower extremity edema, or clubbing detected.

METHODS

Allergen extracts for aeroallergens as well as buckwheat were obtained from Greer Labs (Lenoir, NC). Histamine base (1 mg/mL) positive control was obtained from Center Laboratories (Port Washington, NY), and negative control (50% vol/vol glycerin) was supplied by Miles Inc (Elkhart, IN). Skin prick testing was performed using Duotip-test applicators obtained from Lincoln Diagnostics (Decatur, IL). Skin prick tests were performed after providing informed consent and graded using a standard 0 to 4+ rating system used at our institution. Specifically, prick tests were performed using a 1:20 wt/vol extract with a 0 rating equal to reaction no

different from saline; 1+ equal to erythema <21 mm but greater than control; 2+ equal to erythema $\geq$ 21 mm with wheal <3 mm; 3+ equal to erythema >21 mm with wheal >3 mm without pseudopod formation; and 4+ equal to erythema and wheal with pseudopod formation. Intradermal testing was performed only for inhalant allergens and graded as follows: 0 equal to no different from saline, 1+ equal to 3 to 4 mm wheal with erythema, 2+ equal to 4 to 8 mm wheal with erythema, 3+ equal to wheal greater than 8 mm without pseudopod formation with erythema, and 4+ equal to wheal with erythema and pseudopod formation. ImmunoCAP for allergen specific IgE was performed at the Mayo Medical Laboratories (Rochester, MN) using the ImmunoCAP assay provided by Pharmacia Diagnostics. Specific IgE was reported in kU/L, with a class 0 equal to less than 0.38 kU/L or undetectable, class 1 equal to 0.35 to 0.69 kU/L or of uncertain clinical relevance, class 2 = 0.7 to 3.4 kU/L or probably clinically relevant, class 3 = 3.5 to 17.49 kU/L or clinically relevant, and class 4 equal to >17.49 kU/L or highly clinically relevant. The patient also underwent basic spirometry testing using a Multispiro spirometer (Irvine, CA) with spirometry values calculated based on the patient's height, weight, sex, and age compared with standardized controls, using the best of three trials.

RESULTS

The patient had a 4+ (erythema and wheal with pseudopod formation) skin prick test response to buckwheat. He also had 4+ skin reactions to several trees, grasses and weeds, dog, *Alternaria*, and *Helminthosporium*. He was 3+ positive to *Dermatophagoides pteronyssinus*, *Dermatophagoides farinae*, *Penicillium*, and *Aspergillus*. His histamine control was 4+ positive, and negative control was nonreactive. The patient's ImmunoCAP testing for buckwheat specific IgE had a value of 27.7 kU/L which was a class 4, or strongly positive result. The patient's spirometry showed a normal inspiratory and expiratory flow/volume loop. His forced vital capacity was 97% predicted and his forced expiratory volume in 1 second was 91% predicted with a forced expiratory volume in 1 second/forced vital capacity ratio of 93% predicted. His forced expiratory flow between 25 and 75% was 82% predicted.

The patient performed a 6-week trial of house-dust mite avoidance measures using dust mite encasings for his pillow and mattress and washing his bedding in hot water greater than 140° F weekly. Despite these efforts he had no improvement in his asthma or rhinitis symptoms. After removing both buckwheat pillows from his apartment, his cough and rhinitis symptoms completely resolved within 4 days. On 2-month followup evaluation, he had no significant decline of his spirometry values despite having discontinued all medications. At 6-month followup he had no complaints other than his previous seasonal rhinitis symptoms despite taking no medicines and without performing house-dust mite avoidance.

DISCUSSION

Buckwheat pillows are filled with buckwheat hulls, which are the outer husks of the buckwheat kernel. Interestingly, buck-

wheat (*Fagopyrum esculentum*) is actually a fruit related to the rhubarb plant. It is not taxonomically related to wheat but is sometimes used as a substitute. Buckwheat can be cultivated under poorer circumstances than rice, and both grains and leaves can be used as food. In Japan it is commonly added to foods such as buckwheat noodles and buckwheat dumplings. In other countries it is often used as a baking material for cakes, cereals, and pancakes.

Buckwheat allergy is quite rare in the United States, but it is not uncommon in Japan and other Asian countries where buckwheat consumption is more widespread.⁶ Two proteins with molecular masses approximately 14 and 18 kDa have been identified as the major allergic proteins in buckwheat-allergic patients.⁹ It is interesting to note that these proteins apparently share some structural homology and cross-allergenicity with the proteins implicated in rice allergies.⁹

We could only find one case report in the literature where buckwheat pillows were implicated in causing asthma.¹⁰ This case report documented the presence of buckwheat flour/dust in the buckwheat pillows of three South Korean children. Each of these children had positive skin prick tests as well as positive radioallergosorbent testing for buckwheat specific IgE. They were also observed to have resolution of their asthma within 7 days of buckwheat pillow elimination and positive buckwheat flour bronchial provocation tests. We could find no case reports of asthma or allergic rhinitis attributable to buckwheat pillow exposure in the United States, where allergy to buckwheat is much less common.

In the case presented here, positive skin prick and ImmunoCAP testing to buckwheat, along with the resolution of symptoms with buckwheat pillow elimination, support a buckwheat-specific, IgE-mediated mechanism for our patient's asthma and worsening allergic rhinitis. One might question whether our patient's clinical improvement might also be explained by his skin test positivity to house-dust mites and molds. We believe this to not be the case, given the patient's lack of response to house-dust mite avoidance measures and the absence of visible mold, both in his buckwheat pillows and elsewhere in his apartment. It is also important to note that, at 6-month followup, he did not have a recurrence of his pulmonary or rhinitis symptoms while using a synthetic pillow without house-dust mite encasings.

Our patient's only exposure to buckwheat was presumably through dust in his pillow. He denies having ever knowingly consumed any foods containing buckwheat. It is interesting to note that dust mite encasings with a pore size of 10 μm /L did not improve the asthma and perennial rhinitis symptoms of our patient. Scientists in Japan¹¹ studied buckwheat flour particles using scanning electron microscopy. Their research demonstrated buckwheat flour particles to be round in shape with sizes varying from 1 to 7 μm /L. These particles can easily penetrate our patient's dust mite encasings and may be aerosolized, especially with pillow perturbation. Without the ability to contain buckwheat flour in his pillows, our patient's dust mite avoidance measures were therefore ineffective at

removing buckwheat from his environment, likely resulting in his sensitization to buckwheat.

A recent study¹² of two patients with latex and buckwheat allergies in the Netherlands demonstrated that latex contains epitopes which show cross-reactivity with buckwheat based on latex inhibition of buckwheat radioallergosorbent testing.¹² Our patient denied any history of latex allergy. He also denied a history of food allergy to banana, kiwi, avocado, or chestnut. We were unable to find any published reports of an oral allergy syndrome in patients who react to buckwheat flour. Our patient denied the presence of oropharyngeal pruritus and angioedema with the consumption of foods. Our patient now purposely avoids all buckwheat-containing foods because of fear of a potential systemic reaction.

CONCLUSION

Manufacturers and distributors of buckwheat pillows market them as useful treatments for a host of ailments including headaches, snoring, and lower back pain. One marketer described buckwheat hulls as being the pillow filler of choice of kings and emperors for centuries. Another mentioned that their pillows are the choice of physical therapists, rehabilitation clinics, pediatricians, and allergists. Manufacturers frequently describe buckwheat pillows as being nonallergenic, chemical- and pesticide-free, all natural, and longer-lasting than synthetic or feather/down-stuffed pillows. Over the past decade in the United States, there has been marked increase in the popularity of all-natural/organic foods and medicines. In the future it is likely that many of our patients will try buckwheat pillows as an alternative to poorly perceived synthetic pillows. Allergists as a group need to be aware of the potential risk for sensitization to buckwheat from contact with buckwheat pillows. We need to inquire about the presence of buckwheat pillows in our patients' homes and consider buckwheat allergy in the differential diagnosis for our allergic asthma and rhinitis patients.

REFERENCES

1. Smith HL. Buckwheat poisoning with report of a case in man. *Arch Intern Med* 1909;3:350–359.
2. Dolen WK. Skin testing. *Immunol Allergy Clin North Am* 2001; 21:11a–12a.
3. Obase Y, Shimoda T, Mitsuta K, et al. Two patients with occupational asthma who returned to work with dust respirators. *Occup Environ Med* 2000;57:62–64.
4. Wieslander G, Norback D. Buckwheat allergy. *Allergy* 2001; 56:703–704.
5. Davidson AE, Passero MA, Settignano GA. Buckwheat-induced anaphylaxis: a case report. *Ann Allergy* 1992;69:439–440.
6. Burks AW, Jones SM, Wheeler JG, et al. Anaphylaxis and food hypersensitivity. *Immunol Allergy Clin North Am* 1999;19: 533–552.
7. Valdivieso R, Moneo I, Pola J, et al. Occupational asthma and contact urticaria caused by buckwheat flour. *Ann Allergy* 1989; 63:149–152.
8. Gohte CJ, Wieslander G, Ancker K, et al. Buckwheat allergy: health food, an inhalation health risk. *Allergy* 1983;38:155–159.

-
9. Yoshimasu MA, Zhang JW, Hayakawa S, Mine Y. Electrophoretic and immunochemical characterization of allergenic proteins in buckwheat. *Int Arch Allergy Immunol* 2000;123:130–136.
 10. Lee SY, Lee KS, Hong CH, Lee KY. Three cases of childhood nocturnal asthma due to buckwheat allergy. *Allergy* 2001;56:763–766.
 11. Kusano T, Imaoka S, Segawa S. The structure of buckwheat subcellular particles: research with scanning electron microscopy. *Current Advances in Buckwheat Research. Sixth International Symposium on Buckwheat. Vol. 4.* 1995;881–892.

12. De Maat-Bleeker F, Stapel SO. Cross-reactivity between buckwheat and latex. *Allergy* 1998;53:538–539.

Requests for reprints should be addressed to:
Stephen B. Fritz, MD
Division of Allergy and Clinical Immunology
The University of Michigan Medical Center
3918 Taubman Center, Box 0380
Ann Arbor, MI 48109-0380
E-mail: sbfritz@umich.edu
