

CASE STUDY

A Unique Cause of Asthma in a Baker

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Occupational asthma has been a recognized cause of respiratory disease for centuries, and bakers' asthma, in particular, has been well described. This case report identifies egg white protein as the trigger of asthma in an atopic baker. It highlights the clinical spectra of bakers' asthma, emphasizing the importance of occupational exposures in adult onset asthma.

Keywords occupational asthma, bakers' asthma, egg allergy

INTRODUCTION

The workplace has been recognized as a potential source of respiratory disease since the time of Hippocrates (1), and asthma is the most prevalent occupational respiratory disease. Prevalence of occupational asthma (OA) in the asthmatic population ranges from 2% to 6% (2). Bakers' asthma (BA) is a well-defined clinical entity with an estimated incidence of 5% to 24% according to www.asmanet.org. Asthma in bakers results principally from sensitization to the various components of the grains although a number of other allergens and contaminants have been implicated (3). This case report represents yet another, but unique, presentation of adult onset asthma in a baker.

CASE REPORT

B.T., 31 years of age, was referred by her primary care physician for allergy evaluation. She recalls having symptom of sneezing and nasal obstruction for the last 15 years or more. It was frequently associated with migraine headaches. These symptoms occurred throughout the year without obvious seasonal exacerbation and seemed to be worse at work. They were increasing in both frequency and severity. She had been treating the congestion with pseudoephedrine and the headaches with sumatriptan as needed.

During the course of the interview, B.T. described symptoms of chest heaviness and cough on at least one occasion while at work. She worked in a bakery for a local supermarket. Her primary job was decorating cakes except for once per month when she was responsible for the baking. After further questioning, it was learned that B.T. baked angel food cakes on the days in question.

She was in otherwise good general health. Hospitalization and surgery had never been necessary. She did not take any other medicines except for those described above and an occasional ibuprofen for headache. B.T. had never smoked. There was nothing notable on system review. When prompted, she did recall minor oral irritation on occasion when consuming eggs but could consume them without problem when cooked

in other products. Flour exposure did not appear to provoke symptoms.

Physical examination was unremarkable. Spirometry pre- and post-bronchodilator was normal. Prick skin testing (Greer) to a variety of inhaled and ingested antigens demonstrated reactivity to *Dermatophagoides farinae*, timothy grass, and egg when compared to the control. She was prick test negative to the other antigens, including wheat and soy.

She was to use fluticasone propionate 2 puffs per nostril daily with loratadine-pseudoephedrine 12 hours daily to twice daily as needed for symptom relief and before work when exposure was anticipated. Along with environmental controls for around the home, B.T. was encouraged to wear a mask when baking at work. A peak flow meter was dispensed at that first visit and the patient received instructions on its use and was to keep a record of the readings before and after work exposure. A chest radiograph, sinus series, and an immunocap RAST for egg yolk and white were requested. Follow-up for review of her diary/peak flow entries, the test results, and efficacy of the treatment was scheduled.

At that visit, B.T. reported feeling better in terms of her nasal symptoms and headache. Despite wearing a surgical mask, she experienced palatal itching while baking the angel food cakes and slight lower airway involvement. The ingredient list (Table 1) was reviewed. The radiographs were normal. CAP RAST results demonstrated reactivity to egg white (1.53kU/L, class 2) but the patient tested negative to egg yolk (<0.35, class 0). Serial peak flow readings were not performed on a regular basis but did appear to demonstrate a reduction of 12% to 15% from baseline when baking at work. There was improvement both subjectively and in peak flow following use of her bronchodilator.

B.T. conducted only limited environmental controls at home but left her baker position shortly after our visit. Since this case report she has done well and had an uneventful pregnancy.

DISCUSSION

The clinical features of this case report are consistent with the diagnosis of OA, which is defined as "a disease characterized by variable airflow limitation and/or hyperresponsiveness due to causes and conditions attributable to a particular

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TABLE 1.—Ingredient list for angel food cake mix.

Contains: Sugar, Bleached Enriched Flour (Bleached Flour, Niacin, iron, Thiamin Mononitrate, Riboflavin, Folic Acid), Egg Whites with Sodium Lauryl Sulfate, Wheat Starch, Baking Soda, Sodium Aluminum Phosphate, Fumaric Acid, Salt, Monocalcium Phosphate, Cream of Tartar, Cellulose Gum, Corn Starch, Artificial Flavor.

occupational environment” (4). Occupational asthma can be separated into non-immunologic and immunologic variants (2). Reactive airways dysfunction syndrome (RADS), reflex bronchoconstriction, pharmacologic bronchoconstriction are all manifestations of the non-immunologic form of the condition. Triggers of allergic/immunologic OA tend to be high molecular weight antigens and are predominantly animal, plant, or enzyme derived. These agents appear to induce specific T-helper type 2 lymphocyte and immunoglobulin (IgE) response (2). Examples of high and low molecular weight triggers of OA are listed in Table 2.

There is a low, but consistent, association with atopy and the development of OA (5). Rhinoconjunctivitis typically precedes the onset of asthma in the case of high molecular weight (HMW) exposures (6). Dust mite is clearly responsible for this patient’s perennial symptoms of rhinitis. Mite, including *D. pteronyssinus*, has been known to trigger bakers’ asthma by acting as an irritant or contaminant although the most common mechanism is IgE mediated (3). It could be argued that mite was also responsible this patient’s symptoms of asthma. Exposure to the offending antigen is essential for this sensitization to occur and is the single most important risk factor for OA (7). Generally, there exists a clear dose-response relationship between exposure to occupational allergens and subsequent sensitization. Distinct exposure to mite precipitating her symptoms at the worksite was lacking. Her immediate clinical symptoms and airflow obstruction corresponded with exposure to aerosolized egg white protein. The symptoms on exposure corresponded well with her skin test and specific RAST results, indicating it was not only the responsible agent but that it was provoking an allergic as opposed to irritant etiology. Wheat flour is the most common allergic cause followed by soy flour (3). B.T. lacked symptoms from flour exposure by history, and there was no corresponding evidence of IgE by skin test or RAST. There was no soy flour exposure or evidence of IgE when tested.

The clinical features are consistent with the natural history of OA as suggested by Malo (5): exposure, sensitization, asthma symptoms, cessation of exposure with persistence,

TABLE 2.—Potential causes of occupational asthma by molecular weight.

High molecular weight
 Animal proteins (avian, mammalian)
 Bean dust (castor bean, green coffee bean, etc.)
 Cereals
 Enzymes
 Fish/seafood proteins
 Grain dust
 Gums
 Latex
 Psyllium
 Seeds (flaxseed, linseed, etc.)
 Low molecular weight
 Acid anhydrides
 Amines
 Antibiotics (cephalosporins, penicillins, sulfonamides)
 Diisocyanates
 Dyes
 Flux
 Formaldehyde, glutaraldehyde
 Metals (platinum salts, nickel, vanadium)
 Persulfate
 Woods (Western red cedar/plicatic acid, etc.)

or possible cure. The patient’s improvement when removed from this exposure was not only consistent with diagnosis but an essential part of treatment.

Although asthma as a result of occupational exposure is nothing new, this case report identifies yet another potential trigger. More importantly, it emphasizes the necessity of suspecting occupational exposures in any patient with adult onset asthma. Early identification, rapid treatment, and removal from exposure are associated with significant improvement in prognosis.

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