
Correspondence

ALLERGY TO FUNGAL ALLERGENS IN NORTHERN ITALY

To the Editor:

An article published in the September issue of the *Annals*¹ concludes that IgE-mediated allergy to fungi is both rare and of minor clinical importance in Finland. Our data collected in 4,174 consecutive subjects attending our outpatient allergy clinic located in Bologna in northern Italy are only partly in line with this article. Skin prick test (SPT) with commercial extracts of *Alternaria alternata* and *Cladosporium herbarum* yielded a positive (at least 3-mm) reaction in 339 subjects (8.1%). Of these, 228 (67%) were reactive to *A alternata* only, 49 (14%) to *C herbarum* only, and the remaining to both. Only 21 (6%) of the 339 reactive subjects were sensitized to these molds only (18 to *A alternata*, 1 to *C herbarum*, and 2 to both), with the remaining displaying significant SPT positivity to other inhalant allergens (animal dander, house dust mites, pollens of both grass and trees). SPT positivity for other fungi (*Mucor racemosus*, *Penicillium notatum*, *Aspergillus fumigatus*) was detected in only 21 subjects (6%), most frequently with *M racemosus* (71%). Asthma was more frequently reported by the 318 polysensitized individuals (52%) than by the 21 subjects (38%) sensitized to fungi only. Interestingly, 75% of the latter were 18 years old or younger, and 95% were allergic to *A alternata*. Although in our series, allergy to *A alternata* and *C herbarum* is more frequent and more rarely associated with sensitization to other fungi than in the Finnish study, it seems similarly of low clinical importance, except probably in the youngest subjects sensitized to *A alternata*.

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PROLONGED SURVIVAL IN CHURG-STRAUSS SYNDROME

To the Editor:

We thoroughly enjoyed the case report by Vemuri et al¹ of a patient who survived with Churg-Strauss syndrome (CSS) for 26 years after diagnosis. Ironically, the journal in which this case appeared arrived on our desk on the 26th “anniversary” of the date of the lung biopsy in a still-living patient with CSS. Herein, we report the case of a 51-year-old man who has now survived for 27 years after the diagnosis of CSS.

Our patient was previously healthy, except for a prior septoplasty, when in November 1974 (at the age of 22 years) he developed severe nonatopic asthma. He had frequent exacerbations that required several courses of prednisone. In March 1976, he was admitted to our institution for 3 weeks with severe asthma. Symptoms gradually improved, although he was unable to discontinue prednisone therapy.

Six months later, in August 1976, he was transferred from his local hospital after a 12-day hospitalization for nonresolving pneumonia. He had a 15-kg weight loss, intermittent mild hemoptysis, purulent cough, and shortness of breath. Medications included beclomethasone inhaler, albuterol sulfate tablets and spray, theophylline, and prednisone, 5 mg 4 times daily. Physical examination revealed that he was afebrile, with a pulse of 88/min and blood pressure of 110/70 mm Hg. The results of cardiovascular and abdominal examinations were normal. Respiratory examination revealed a clear chest to auscultation. A pustular perioral rash was noted transiently. Chest radiograph demonstrated bilateral pulmonary infiltrates. Five days after his admission, an open lung biopsy specimen revealed a granulomatous vasculitis with interstitial pneumonitis and eosinophilic infiltrates consistent with CSS. The pulmonary infiltrates gradually resolved with high-dose steroids.

Despite improvement in his chest radiograph and constitutional symptoms, his asthma remained difficult to control, requiring baseline prednisone, 10 to 20 mg/d. In 1978, he started azathioprine therapy and temporarily discontinued prednisone use until his azathioprine supply ended. He resumed prednisone therapy thereafter, along with the use of various bronchodilators and inhaled corticosteroid medications. Unfortunately, he experienced numerous corticosteroid-related adverse effects, including weight gain, abdominal striae, bilateral cataracts, avascular necrosis of the right acetabular hip, osteoporosis with subsequent rib and thoracic vertebrae fractures, and recurrent infections, including pyelonephritis and cellulitis. He continued to have frequent exacerbations of his asthma but never with recurrence of the

infiltrates. His best forced expiratory volume in 1 second (FEV₁) ranged from 40% to 60% predicted.

In 1992, after the completion of his child bearing, he started cyclophosphamide therapy, 2 mg/kg, in hopes that the prednisone could be tapered. Despite 2 years of therapy, the lowest tolerated daily dose of prednisone was unchanged at 15 mg. In 1994, the dose of cyclophosphamide was increased with subsequent hematuria. Cyclophosphamide therapy was abandoned 8 months later due to adverse effects and no benefit.

In 1998, he developed a complex sinus infection that required 6 weeks of therapy with cefuroxime. He subsequently underwent a variety of therapeutic trials with montelukast, inhaled formoterol, high-dose inhaled budesonide, and finally combination therapy with inhaled budesonide and formoterol (Symbicort; AstraZeneca, Södertälje, Sweden). Despite this, his best FEV₁ remained in the 45% to 50% predicted range.

On his most recent visit in May 2003, his asthma remained severe but stable with the inhaled budesonide and formoterol therapy, as needed short-acting inhaled bronchodilators, prednisone (10 mg/d), and theophylline. His CSS (pulmonary infiltrates) has remained quiescent on the low-to-moderate doses of prednisone required to control his asthma.

We believe that this is the longest documented surviving patient after diagnosis of CSS. Since he has been under the care of a variety of different specialists throughout the last 27 years, his pathologic case was revisited by the pathology department at our institution in 1987, which reconfirmed the condition to be consistent with CSS. Unfortunately, the pathology slides have since been destroyed.

This patient fulfills the American College of Rheumatology criteria² for the diagnosis of CSS given the history of asthma, nonfixed pulmonary infiltrates, sinus abnormalities, and extravascular eosinophilic infiltrates on open lung biopsy. Unfortunately, it is uncertain whether he had a peripheral blood eosinophilia, because he was undergoing corticosteroid therapy before his initial presentation and biopsy. Interestingly, our patient may also have had associated skin involvement, although not as has been classically described previously.

In summary, we have described the case of a patient with CSS who has survived for 27 years after diagnosis and whose disease is now relatively quiescent. Similar to Vemuri and colleagues' patient, we suspect that there may be more patients with minimally active CSS who are surviving longer than 20 years while taking low-to-moderate doses of prednisone.

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ACUTE ANAPHYLAXIS AFTER PINE NUT SKIN TESTING

To the Editor:

We report a case of acute anaphylaxis following skin prick testing with pine nut in a 20-year-old woman. The patient collapsed minutes after eating a mixed salad and was referred to the Department of Dermatology and Allergology of our institution with complaints of generalized urticaria, swelling of the face, and dyspnea. We identified pine nuts as the ingredient most likely to have caused the anaphylaxis.

A prick-prick test with fresh pine nut was performed. Our patient developed a strong local skin reaction, acute dyspnea, angioedema, and circulatory collapse within minutes. She recovered rapidly after treatment with intramuscularly administered clemastine, epinephrine, and oxygen by mask.

In the literature, different test methods to evaluate pine nut allergy have been reported, including prick tests with a hydroalcoholic extract from raw pine nuts with a total protein content of 111 mg/L,¹ fresh pine nuts,² an aqueous suspension of fresh pine nuts crushed in 0.9% sodium chloride,^{3,4} and a filtered and sterilized mixture of pine nuts in Hollister-Stier diluent.⁵ To our knowledge, acute anaphylaxis to pine nut, the edible seed of *Pinus pinea*, has not yet been reported following skin testing. Severe systemic reactions following ingestion of pine nut have been described and are likely to be encountered more often, since pine nuts are increasingly used in all sorts of foods.

We stress the importance of a standardized skin test for pine nut hypersensitivity to minimize the risk of severe reactions. In our opinion, dilutions of an aqueous solution of pine nut should be used instead of prick-prick tests with fresh pine nuts.

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DRY VS AQUEOUS INHALERS

To the Editor:

With the removal of Nasacort (triamcinolone; Rhône-Poulenc Rorer, Collegeville, PA) dry inhaler from the US market in July 2003, only aqueous (AQ) formulations of nasal steroids are available for treatment of allergic and nonallergic rhinitis. Although AQ drugs are preferred by more people than dry forms, a significant minority of patients will not use any AQ formulation, whether or not it contains phenylethyl alcohol.

Necessity still being the mother of invention, we were reminded of altering the Atrovent metered-dose inhaler (ipratropium bromide; Boehringer Ingelheim, Ridgefield, CT) to treat gustatory rhinitis before nasal forms of Atrovent became available. Before 1995, many physicians attached a baby nipple to the opening of the Atrovent metered-dose inhaler and then cut off a small part of the tip of the nipple. This allowed ipratropium to be delivered to the nose.

We have been modifying QVAR inhalers (beclomethasone dipropionate; 3M Health Care Limited, Loughborough, UK) the same way, making a dry beclomethasone hydrofluoroalkane nasal spray. Until Nasacort hydrofluoroalkane becomes available, this simple modification should prove useful in continuing topical steroid treatment for rhinitis patients who prefer not to use an AQ form.

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THE MANAGEMENT OF LOCAL REACTIONS DUE TO ALLERGEN IMMUNOTHERAPY

To the Editor:

I have read with great pleasure the meticulously prepared article, "Allergen immunotherapy: a practice parameter."¹

This report covers the principles of allergen immunotherapy thoroughly. I have only one objection or contribution regarding the management of local reactions caused by injections of allergen vaccine: topical corticosteroid application for local reactions is a common practice in some allergy departments. We know that topical corticosteroids are effective mainly in T-cell-mediated diseases (eg, contact dermatitis, psoriasis, lichen planus). Both immediate- and late-phase skin response to cutaneous antigenic challenge are in the spectrum of type 1 hypersensitivity reaction.² From this point of view, it is clear that topical corticosteroids are ineffective in the management of immediate local reactions seen during skin tests and/or allergen vaccine injections. Their efficacy in late-phase skin response, which is much more complex in nature than the immediate phase, is also lacking or minimal. Only application of cool compresses and in some situations systemic antihistamine use seem reasonable for treating local reactions.

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