Association Between Peanut Allergy and Asthma Morbidity

Alyson B. Simpson, MD, Ejaz Yousef, MD, and Jobayer Hossain, PhD

Objective To evaluate the relationship between peanut allergy and asthma morbidity in school-age children.
Study design The study involved a medical chart review to assess the association of peanut allergy with asthma morbidity in children beyond age 3 years. Peanut allergy was assessed by specific and validated criteria. A Poisson regression model was used to compare the frequency of systemic steroid use and of hospitalization for asthma beyond age 3 years in children with asthma with and without peanut allergy.
Results Children with peanut allergy had a 2.32-times greater rate of hospitalization ($P = .03$) and a 1.59-times greater rate of systemic steroid use ($P < .001$) after controlling for covariates.
Conclusions Peanut allergy serves as an early marker for asthma morbidity. Early prevention and intervention can improve quality of care. *(J Pediatr 2010;156:777-81)*.

Asthma is a highly prevalent disease, affecting approximately 9 million children under age 18.[1,2] Asthma exacerbations are the leading cause of childhood inpatient admissions, causing 2 million emergency department visits and 500 000 hospitalizations among all patients with asthma in 2005.[3] Asthma also is a major risk factor for fatal and near-fatal food-induced anaphylaxis.[4-6] More than one-third of children with food allergies are currently living with asthma.[7,8]

Morbidity from asthma has been increasing in the United States. Reducing asthma morbidity is a key focus of research and public health initiatives. A better understanding of the underlying mechanisms of asthma and risk factors for poor asthma control is therefore essential to improve the outcomes of these patients.

Several risk factors for increased asthma morbidity have been identified, including the presence of food allergy.[4,9-11] In a previous study, we established a link between allergy to milk and peanut and asthma morbidity in children in the early childhood years.[11] As more children are entering school with a dual diagnosis of peanut allergy and asthma,[12-14] we now need to focus on the effect of peanut allergy on asthma symptoms beyond age 3 years. Because children often present with food allergy in early childhood, peanut allergy could serve as an early marker for asthma morbidity. In the present study, we assess the effect of peanut allergy on asthma morbidity beyond age 3 years.

Methods

The study population comprised 410 children age 5-18 years with asthma and a food allergy who received care at a large tertiary care children's hospital at any time between their third birthday up to September 2008. A central computerized patient database was used to identify potential subjects. Information in the database included hospitalizations, discharge summaries, outpatient and emergency department records, prescribed medications (including systemic steroids), laboratory tests, and medical imaging.

A list of patients with *International Classification of Diseases, Ninth Revision* codes 493.90 (asthma, unspecified), 493.91 (asthma with status asthmaticus), and/or 493.92 (asthma with acute exacerbation), was obtained. The medical records of 410 patients age 5-18 years with a diagnosis of asthma were reviewed. Study subjects were identified by collecting data on those patients age ≥5 years who had developed persistent asthma symptoms by age 3 years. The study protocol was approved by the hospital's Institutional Review Board.

Definition and Characteristics of Children With Asthma with and without Peanut Allergy

Asthma was diagnosed based on the following criteria: (1) symptoms of recurrent (ie, more than 2) episodes of wheezing, cough, shortness of breath, or a combination of these; and (2) documented reversibility with bronchodilators.[15] All of the patients were prescribed inhaled corticosteroids. Patients in the peanut allergy group were diagnosed based on the following criteria: (1) adverse reaction to food;[16] (2) food-specific IgE level >95% positive predictive value (PPV) to peanut (IgE ≥15)

CI	Confidence interval
ICAP RAST	ImmunoCAP radioallergosorbent test
NAEPP	National Asthma Education and Prevention Program
PPV	Positive predictive value
RR	Risk ratio
SPT	Skin prick test

From the Department of Pediatrics, Alfred I. duPont Hospital for Children, Wilmington, DE (A.S., E.Y., J.H.) and Division of Allergy and Immunology, Thomas Jefferson University, Philadelphia, PA (A.S., E.Y.)

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(Table I; available at www.jpeds.com);[17,18] (3) and a skin prick test (SPT) 3 mm greater than negative control.[19-21]

Exclusion Criteria

Patients were excluded from the study if: (1) their clinical history did not support the diagnosis of asthma; (2) were not being treated according to National Asthma Education and Prevention Program (NAEPP) guidelines;[15] (3) did not attend >75% of their scheduled outpatient visits, as documented in our computerized outpatient database; (4) were lost to follow-up before September 2008; (5) were followed for <2 years beyond age 3 years; or (6) had cystic fibrosis or immunodeficiency. Patients with peanut allergy were excluded if they had a clinical history suggestive of peanut allergy but did not have a confirmatory ImmunoCAP radioallergosorbent test (ICAP RAST; Pharmacia Diagnostics AB, Portage, Michigan) value >15, the 95% PPV to peanut,[16-18] and an SPT 3 mm greater than negative control.[19-21] Children diagnosed with egg, milk, and shellfish allergy also fulfilled the strict inclusion criteria for food allergy, including a suggestive clinical history, IgE >95% PPV for each specific food,[16-18] and SPT 3 mm greater than negative control.[19-21]

Predictors

Demographic information was gathered, including race, sex, age (documented in months), history of atopic dermatitis, family history of asthma, gastroesophageal reflux, history of food allergy, history of aeroallergen sensitivity, and passive smoke exposure in the home. Atopic dermatitis was defined according to the diagnostic criteria of Hannifin and Rajka.[22] Gastroesophageal reflux was defined based on clinical signs and symptoms as well as diagnostic test results (ie, pH probe, gastric emptying scan, endoscopy with biopsy, or upper gastrointestinal series). Most of the covariates were organized into categorical variables, including sex (male or female), race (Caucasian, African American, or others), and the presence or absence of the following: atopic dermatitis; family history of asthma; gastroesophageal reflux; adenoidal hypertrophy; passive smoke exposure in the home; allergy to milk, egg, or shellfish; and the presence or absence of sensitization to dust mite, aspergillus, alternaria, grass, weed, cat, dog, and tree. Age was recorded at 3 points in time: at the first visit to the food allergy clinic, at the onset of persistent asthma symptoms, and at the end of the review period in September 2008. Chronologic age was included in the regression as a continuous variable.

IgE Level and Skin Testing

The results of previous skin tests and food-specific IgE levels (ICAP RAST) to peanuts, egg, milk, and shellfish were obtained in addition to the results of skin testing to aeroallergens. All SPTs were performed using the Greer*Pick* method (Greer Lab, Lenoir, North Carolina) and allergen extract (provided by Hollister-Stier Laboratories, Spokane, Washington). A SPT was considered positive when the wheal diameter was 3 mm and 50% larger than negative control, and the negative control remained negative.

Table II. Univariate Poisson regression model of the effect of peanut allergy and other factors/covariates on the hospitalization of pediatric patients with asthma in a sample from a large tertiary hospital

Variables	RR	95% CI	*P**
Peanut allergy			
No	1.0	Reference	Reference
Yes	2.88	1.86-4.47	<.001
Race			
Caucasian	1.0	Reference	Reference
African American	2.36	1.47-3.81	<.001
Others	3.24	1.69-6.22	<.001
Sex			
Female	1.0	Reference	Reference
Male	1.73	1.03-2.89	.037
Family history of atopy			
No	1.0	Reference	Reference
Yes	2.55	0.35-18.32	.353
Smokers at home			
No	1.0	Reference	Reference
Yes	2.01	1.29-3.13	.002
Atopic dermatitis			
No	1.0	Reference	Reference
Yes	1.56	0.98-2.47	.058
Gastroesophageal reflux			
No	1.0	Reference	Reference
Yes	1.79	1.16-2.78	.009
Adenoidal hypertrophy			
No	1.0	Reference	Reference
Yes	0.47	0.26-0.83	.01
Aspergillus sensitization			
No	1.0	Reference	Reference
Yes	2.93	1.86-4.62	<.001
Alternaria sensitization			
No	1.0	Reference	Reference
Yes	1.43	0.91-2.23	.12
Dust mite sensitization			
No	1.0	Reference	Reference
Yes	2.91	1.78-4.76	<.001
Milk allergy			
No	1.0	Reference	Reference
Yes	0.92	0.53-1.62	.780
Egg allergy			
No	1.0	Reference	Reference
Yes	1.53	0.97-2.41	.064
Shellfish allergy			
No	1.0	Reference	Reference
Yes	3.41	2.09-5.58	<.001
Grass sensitization			
No	1.0	Reference	Reference
Yes	1.57	1.01-2.44	.043
Weed sensitization			
No	1.0	Reference	Reference
Yes	0.73	0.42-1.26	.251
Tree sensitization			
No	1.0	Reference	Reference
Yes	2.02	1.31-3.15	.002
Cat sensitization			
No	1.0	Reference	Reference
Yes	2.61	1.61-4.23	<.001
Dog sensitization			
No	1.0	Reference	Reference
Yes	1.20	0.75-1.92	.453
Age, months	1.01	1.00-1.01	.120

*P <.05.

All children with a history suggestive of allergic rhinitis or atopic dermatitis underwent skin testing with the same panel of aeroallergens (Table II). Data were recorded in a Microsoft