

Food Allergy and Asthma Morbidity in Children

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Summary. Background: Coexisting food allergy and asthma is a significant problem in the pediatric population. Studies have looked at the association between food sensitization and asthma severity. It is unknown whether specific food allergies are associated with increased asthma morbidity. Objective: We studied the independent effect that allergy to egg, milk, fish, and peanut has on the number of hospitalizations and courses of systemic steroids in children with asthma. Methods: We performed a medical record review to evaluate the effect food allergy to egg, fish, peanut, and milk has on asthma morbidity. We reviewed the records of 201 children aged 3 months to 14 years with the diagnosis of asthma (ICD-9 codes 493.90, 493.91, and 493.92), of which 88 had coexistent food allergy. All children in the food allergy group had food-specific IgE concentrations greater than the 95% positive predictive value. We compared the rate of hospitalizations and use of systemic steroids between children with asthma and food allergies and those without coexisting food allergy using direct-entry, multiple regression analysis. Patients were adjusted for the severity of their asthma based on symptoms documented at their first visit to the allergist according to the National Asthma Education and Prevention Program guidelines and presence of environmental allergy, eczema, smoke exposure, and gastroesophageal reflux. Results: Peanut and milk allergies were both associated with increased number of hospitalizations ($P=0.009$, 0.016), and milk allergy was associated with increased use of systemic steroids ($P=0.001$). Conclusion: Peanut and milk allergies were associated with increased hospitalization and steroid use and may serve as early markers for increased asthma morbidity.

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Key words: asthma morbidity; egg allergy; fish allergy; food allergy; food-specific IgE levels; milk allergy; peanut allergy.

INTRODUCTION

The prevalence of asthma has increased by 75% in the past two decades despite improvements in prevention and management.^{1,2} It continues to be the most common reason for hospitalizations in the pediatric age group, causing over 200 deaths in the United States every year.¹ Several risk factors for asthma morbidity have already been described in the literature including early onset of wheezing, atopy, smoke exposure, allergic rhinitis, and poor access to health care,^{2,3} but little has been studied on the association between asthma and food allergy.

Coexistent food allergy and asthma is an increasing burden on the pediatric population. Four to eight percent of children with asthma have food allergy, and more than a third of children with food allergy have asthma.^{4,5} Despite the strong association between food allergy and asthma, it is rare that food sensitization would cause isolated asthma symptoms.⁶ Longitudinal studies have indicated that food allergy to egg and wheat in early infancy increases the risk for developing asthma later in life.^{7,8} Studies have not shown that milk and peanut allergy are predictors of persistent asthma symptoms and increased morbidity even as a child's sensitivity to the food wanes.

Allergies to egg, milk, peanut, and fish may serve as early markers for persistent asthma symptoms and increased asthma morbidity even as children outgrow their food allergies. Wang et al.³ found that food sensitization was associated with higher rates of hospitalizations and steroid use among inner-city children with asthma. Roberts et al.⁹ found an association between food allergy and life-threatening asthma. With the increasing prevalence of food allergies and asthma

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and the advancement in their management, it is important to identify those at risk and the need for closer follow-up.

The purpose of this study was to determine whether IgE-mediated allergy to egg, milk, fish, or peanuts is associated with increased number of hospitalizations and use of systemic steroids in children with asthma. Because food allergies are usually diagnosed in children at a very young age, early food avoidance and closer follow-up may improve clinical outcome and reduce asthma morbidity. Interventions like early avoidance of allergenic foods may prevent the onset of asthma or decrease its severity.

METHODS

We performed a retrospective chart review study approved by the Institutional Review Board at the Alfred I. duPont Hospital for Children. We first identified patients with asthma presenting to the outpatient allergy department at our hospital from January 1995 to March 2006 through the Health Information Management Department by using the International Classification of Diseases, Ninth Revision (ICD-9) codes 493.90, 493.91, and 493.92. Medical records of 835 patients, aged 3 months to 14 years, with the diagnosis of asthma were reviewed.

We used EPIC, our central computerized patient database, to attain all demographic, clinical, and historical information. This information includes all of the discharge summaries from our hospital and documentation of all medications prescribed including systemic steroids and reviews all emergency department visits, telephone encounters, laboratory tests, and medical imaging.

Study patients were divided into two groups. Group A consisted of 88 patients with asthma and food allergy. Group B consisted of 113 patients with asthma alone. Asthma was diagnosed based on having both of the following criteria: (1) symptoms of recurrent (>2) episodes of wheezing, cough, or shortness of breath, or a combination of the three and (2) documented reversibility with bronchodilators. Additionally, 78 (85%) of the children in Group A and 85 (75%) of the children in Group B had a family history of asthma. All the patients in the food allergy group (Group A) must have fulfilled all three of the following criteria for food allergy: (1) adverse reaction to food;¹⁰ (2) food-specific IgE levels

greater than 95% positive predictive value (PPV) to egg, milk, peanut, or fish¹⁰⁻¹² (Table 1); and (3) a skin prick test (SPT) 3 mm greater than control.¹³⁻¹⁵ Some children with food allergy were enrolled prior to developing asthma symptoms, but all children developed asthma symptoms within 6 months of their initial allergy visit.

Patients were excluded from the study if (1) their clinical history did not support the diagnosis of asthma, (2) they were not being treated according to the National Asthma Education and Prevention Program (NAEPP) guidelines,¹⁶ (3) they did not attend over 75% of their scheduled outpatient visits as documented in our computerized outpatient database, (4) they were followed for less than 2 years, or (5) they had cystic fibrosis or immunodeficiency.

Patients with food allergy were excluded if they had clinical history suggestive of food allergy but did not have a confirmatory ImmunoCAP radioallergosorbent test (ICAP RAST) (Pharmacia Diagnostics AB, Portage, MI) level greater than 95% PPV to egg, milk, peanut, or fish¹⁰⁻¹² and SPT 3 mm greater than control.¹³⁻¹⁵

Patients with asthma were classified according to the NAEPP guidelines¹⁶ based on the history and physical examination documented at their first visit to the allergy practice. Patients were separated into two groups: one group combined patients with mild intermittent and mild persistent asthma, and the second group combined patients with moderate and severe persistent asthma.

Children in both groups were managed according to the NAEPP guidelines,¹⁶ and all children were eventually placed on inhaled steroids for control of persistent symptoms. Twenty children (22.7%) in Group A and 40 children in Group B (35.3%) had been on a combination of inhaled corticosteroid and a long-acting beta agonist (Advair[®]) once they were older than 5 years. None of the study subjects used long acting beta agonists as monotherapy.

Demographic information including race and age (documented in months), presence or absence of gastroesophageal reflux, history of eczema, family history of asthma, and passive smoke exposure in the home was gathered from the history documented in the first outpatient allergy visit. We defined eczema/atopic dermatitis according to the diagnostic criteria by Hanifin and Rajka.¹⁷ Gastroesophageal reflux was defined by clinical signs and symptoms plus one of the diagnostic tests (pH probe, gastric emptying scan, endoscopy with biopsy, or upper gastrointestinal series).

We reviewed the number of hospitalizations and courses of systemic steroids from the first documented allergy visit and added on all hospitalizations and courses of systemic steroids until the end of the study period, March 2006. All hospitalizations including those at our institutions and those at any other outside facility were accounted for and documented in the patient's medical record. This record also included all courses of systemic steroids prescribed

TABLE 1—Food-Specific IgE Levels Greater Than 95% Positive Predictive Value (PPV)¹¹

Food	Value (kU/L)	PPV (%)
Egg	7	98
Egg (infants ≤ 2 years)	2	95
Milk	15	95
Milk (infants ≤ 2 years)	5	95
Peanut	14	100
Fish	20	100

TABLE 2—Distribution of Environmental Allergens

Allergen	Food allergy (Group A)	No food allergy (Group B)
Dust	37 (42)	41 (36.3)
Cat	43 (48.9)	41 (36.3)
Cockroach	19 (21.6)	26 (23)
Mold	22 (25.0)	35 (31)
Dog	28 (31.8)	30 (26.5)
Weed	36 (40.9)	32 (28.3)
Tree	25 (28.4)	27 (23.9)
Alternaria	32 (36.4)	28 (24.8)
Aspergillus	19 (21.6)	20 (17.7)
Grass	30 (34.1)	38 (33.6)
Hormod	23 (26.1)	14 (12.4)
Penicillin	14 (15.9)	6 (5.3)

Number in parentheses is percentage.

for an asthma exacerbation by any physician caring for the patient including those prescribed by their primary care physician. We did not include visits where children were admitted for an immediate reaction to food ingestion including anaphylaxis, urticaria, or gastrointestinal symptoms or were treated with epinephrine and/or benadryl for concern of food-induced wheezing.

The results of previous skin tests and food-specific IgEs (ICAP RAST) to fish, egg, milk, and peanuts were also obtained from the records in EPIC in addition to the results of skin testing to aeroallergens. Skin testing was performed by the prick method using commercial extract (Hollister-Stier Laboratories, LLC, Spokane, WA). The SPTs were considered positive when the wheal diameter was 3 mm and 50% larger than positive control and the negative control remained negative. The possibility of dermatographism was eliminated by having a negative reaction to the negative control.¹⁸ All children with histories suggestive of allergic rhinitis or eczema had been skin tested to the same panel of aeroallergens as recorded in Table 2. Data were recorded in a Microsoft Excel file and they were later transferred to an SPSS dataset (SPSS version 14.0, Chicago, IL, 2005).

All study patients were followed by the same providers and were provided complete asthma education during their routine clinic visits. Likewise, the allergists also educated all patients with food allergy on food avoidance and treatment of accidental exposure.

Data Analysis

Two outcomes/criteria were examined: number of hospitalizations and courses of systemic steroids. Both variables were on the interval scale of measurement. Therefore, data were analyzed using four direct-entry multiple regression analyses. A separate multiple regression analysis was performed for each criterion using SPSS version 14.0. Predictors for the first two analysis were identical and comprised eczema; gastroesophageal reflux; smoke exposure in the home; presence of environmental allergens; classification of asthma at first documented allergy visit; and presence of egg, peanut, milk, or fish allergy. We then replaced specific food allergies for the number of food allergies to determine whether two or more food allergies has more of an impact in predicting number of hospitalizations and courses of systemic steroids. Number of food allergies was quantified on a scale from 1 to 4 as follows: 1 = one food allergy, 2 = two food allergies, 3 = three food allergies, and 4 for all children with four food allergies or more. All of the other predictors were binary and dummy coded before analysis. The relative importance and utility of the predictors were determined through the interpretation of squared semi-partial coefficients.^{19,20}

RESULTS

Group Characteristics

The 201 children who met the inclusion criteria for the study were divided into two groups. Group A (N = 88) consisted of patients with diagnosis of asthma and food allergies. Group B (N = 113) consisted of patients with

TABLE 3—Demographic Comparisons Between Asthmatic Patients With and Without Food Allergy

Demographic	Food allergy (Group A)	No food allergy (Group B)
Female	22 (23.9)	51 (45.1)
Male	66 (75)	62 (54.9)
Caucasian	46 (52.3)	86 (76.1)
African American	20 (22.7)	16 (14.2)
Hispanic	2 (2.3)	6 (5.3)
Asian	4 (4.5)	2 (1.8)
Unknown race	16 (18)	3 (2.7)
Atopic dermatitis/eczema	62 (70.5)	28 (24.8)
Smoke exposure	35 (39.8)	35 (31)
History of reflux	16 (18.2)	36 (31.9)
Mean age at first documented visit	31.5	57.8
Mean age at last documented visit	75.4	109.3
Mean time that elapsed from first documented visit to the last visit	45	52

Number in parentheses is percentage. Age and time documented in months.

TABLE 4—Classification of Asthma According to the National Asthma Education and Prevention Program Guidelines¹⁶

Classification	Food allergy (Group A)	No food allergy (Group B)
Mild intermittent	58 (65.9)	18 (15.9)
Mild persistent	20 (22.7)	54 (47.8)
Moderate persistent	10 (11.4)	38 (37.6)
Severe persistent	0 (0)	3 (2.7)

Number in parentheses is percentage.

asthma without food allergy. Demographic data are shown in Table 3. Patients ranged in age from 3 months to 14 years. There were more females in Group B and more African Americans in Group A. However, both groups had a majority of Caucasian children, and race was unknown in 16 (18%) of those with food allergy. There were more cases of gastroesophageal reflux (GERD) in Group B and more cases of atopic dermatitis in Group A. However, we controlled for both of these variables in our regression. Children in Group A presented at a younger age and, on average, were followed for a shorter period of time. Patients in both groups had a similar distribution of environmental allergens (Table 2).

More patients in Group A had mild asthma symptoms at their first documented allergy visit (Table 4), which we controlled for in the regression. Patients were evaluated for the markers of morbidity, and the results are outlined in Table 5.

Specific Characteristics of Children With Food Allergy (Group A)

Patients, on average, showed signs of food allergy at 9 months of age. Distribution data on food allergy showed that 52 (59.1%) children had egg allergy, 54 (61.4%) had

TABLE 5—Markers for Morbidity in Those With and Without Food Allergy

Marker for morbidity	Food allergy	Mean	Standard deviation	P-value
Number of ED visits	Yes	1.95	2.59	0.083
	No	1.36	2.17	
Courses of systemic steroids	Yes	2.67	2.88	0.155
	No	2.12	2.543	
Number of hospitalizations	Yes	0.89	1.57	0.038
	No	0.48	1.196	
Number of PICU stays	Yes	0.11	0.416	0.117
	No	0.04	0.207	

ED = emergency department; PICU = pediatric intensive care unit; # = number.

P-values reported in the table detect significant differences in the mean number of emergency department visits, courses of systemic steroids, hospitalizations, and PICU stays between those with food allergy and those without food allergy derived from a *t*-test detecting differences in mean variables.

peanut allergy, 33 (37.5%) had milk allergy, and 8 (9.1%) had fish allergy. Thirty-four (38.6%) had 1 food allergy, 24 (27.3%) had 2 food allergies, 19 (21.6%) had 3 food allergies, 6 (6.8%) had 4 food allergies, 4 (4.5%) had 5 food allergies, and 1 (1.1%) had 6 food allergies. All patients had food-specific IgE levels greater than 95% PPV.^{10–12}

Effect of Food Allergy on Markers for Morbidity

Table 6 shows the results for the multiple regression analysis focusing on the effect that food allergy to egg, milk, peanut, and fish has on number of hospitalizations. Peanut allergy ($P = 0.009$), milk allergy ($P = 0.016$), and asthma classification based on symptoms at first documented allergy visit ($P = 0.001$) were independently

TABLE 6—Direct Regression of Predictors on Number of Hospitalizations

Predictor	b	β	P	95% confidence limit for β		
				sr_i^2	Lower	Upper
Reflux	0.079	0.024	0.733	0.001	−0.379	0.537
Atopic dermatitis	−0.150	−0.054	0.508	0.002	−0.597	0.296
Smoke exposure at home	0.316	0.107	0.108	0.014	−0.069	0.702
Environmental allergens	0.017	0.037	0.586	0.002	−0.044	0.078
Asthma classification	1.277	0.401	0.001	0.142	0.825	1.730
Egg allergy	0.154	0.048	0.616	0.001	−0.450	0.757
Peanut allergy	0.593	0.190	0.009	0.036	0.152	1.035
Milk allergy	0.765	0.200	0.016	0.031	0.144	1.387
Fish allergy	−0.359	0.200	0.733	0.002	−1.561	0.842
Constant	−0.108					

b = unstandardized regression coefficients; β = standardized regression coefficients (where mean = 0 and standard deviation = 1); P = statistical significance level; sr_i^2 = squared semipartial coefficients, and asthma classification controls for those with moderate to severe asthma at first documented visit to the allergist.

TABLE 7—Direct Regression of Predictors on Number of Hospitalizations

Predictor	b	β	P	95% confidence limit for β		
				sr_i^2	Lower	Upper
Reflux	0.022	0.007	0.924	0.000	−0.434	0.478
Atopic dermatitis	−0.060	−0.022	0.786	0.000	−0.499	0.379
Smoke exposure at home	0.067	0.023	0.740	0.001	−0.330	0.463
Environmental allergens	0.017	0.032	0.596	0.001	−0.046	0.079
Asthma classification	0.699	0.412	0.000	0.136	0.446	0.952
Number of food allergies	0.364	0.327	0.000	0.073	0.177	0.550
Constant	−0.36					

b, unstandardized regression coefficients; β , standardized regression coefficients (where mean = 0 and standard deviation = 1); P, statistical significance level; sr_i^2 , semipartial coefficients, and asthma classification controls for those with moderate to severe asthma at first documented visit to the allergist.

associated with hospitalization rates for asthma exacerbations. Table 7 shows the results for a similar regression looking at the effect the number of food allergies has on number of hospitalizations taking out specific food allergies, which are covariants. The number of food allergies ($P = 0.000$) and asthma classification at first documented allergy visit ($P = 0.000$) were independently associated with number of hospitalizations for asthma exacerbations. Egg allergy, fish allergy, eczema, gastroesophageal reflux, smoke exposure in the home, and presence of environmental allergens were not significant predictors.

Table 8 shows the results for the multiple regression analysis focusing on the effect that food-specific allergy has on courses of systemic steroids. Milk allergy ($P = 0.001$) and asthma classification based on symptoms at first documented allergy visit ($P = 0.001$) were independently associated with systemic steroid use. Table 9 shows the results for a similar regression run looking at the effect the number of food allergies had on courses of systemic steroids. The number of food allergies

($P = 0.000$), asthma classification based on symptoms at first documented allergy visit ($P = 0.000$), and atopic dermatitis ($P = 0.043$) was independently associated with increased courses of systemic steroids. Allergy to egg, fish, or peanut; gastroesophageal reflux; smoke exposure; and presence of environmental allergens were not significant predictors.

DISCUSSION

Coexistent food allergy and asthma is a significant burden in the pediatric population, and the incidences of both have increased dramatically over the past two decades. Our study compared the number of hospitalizations and courses of systemic steroids in children with asthma and coexisting food allergy with asthmatic children without food allergy. Our study included only children with food-specific IgE levels greater than 95% PPV to evaluate the association clinical food allergy has on asthma morbidity. Milk allergy ($P = 0.016$) and peanut allergy ($P = 0.009$) were associated with increased

TABLE 8—Direct Regression of Predictors on Courses of Systemic Steroids

Predictor	b	β	P	95% confidence limit for β		
				sr_i^2	Lower	Upper
Reflux	−0.083	−0.013	0.850	0.001	−0.950	0.784
Atopic dermatitis	−0.475	−0.088	0.269	0.007	−1.321	0.370
Smoke exposure at home	0.478	0.084	0.198	0.009	−0.251	1.208
Environmental allergy	0.087	0.098	0.138	0.012	−0.028	0.203
Asthma classification	2.666	0.435	0.001	0.168	1.810	3.522
Egg allergy	0.155	0.025	0.789	0.001	−0.987	1.297
Peanut allergy	0.742	0.123	0.082	0.016	−0.095	1.578
Milk allergy	2.020	0.274	0.001	0.058	0.843	3.197
Fish allergy	−0.723	−0.042	0.531	0.002	0.997	1.550
Constant	0.910					

b, unstandardized regression coefficients; β , standardized regression coefficients (where mean = 0 and standard deviation = 1); P, statistical significance level; sr_i^2 , semipartial coefficients, and asthma classification controls for those with moderate to severe asthma at first documented visit to the allergist.

TABLE 9—Direct Regression of Predictors on Courses of Systemic Steroids

Predictor	b	β	P	95% confidence limit for β		
				sr_1^2	Lower	Upper
Reflux	0.489	0.078	0.289	0.005	−0.420	1.396
Atopic dermatitis	−0.904	−0.167	0.043	0.022	−1.777	−0.030
Smoke exposure at home	0.325	0.057	0.418	0.003	−0.464	1.114
Environmental allergy	0.074	0.083	0.241	0.007	−0.050	0.198
Asthma classification	0.706	0.216	0.006	0.039	0.202	1.209
Number of food allergies	0.779	0.363	0.000	0.083	0.409	1.150
Constant	0.947					

b, unstandardized regression coefficients; β , standardized regression coefficients (where mean = 0 and standard deviation = 1); P, statistical significance level; sr_1^2 , semipartial coefficients, and asthma classification controls for those with moderate to severe asthma at first documented visit to the allergist.

number of hospitalizations. Milk allergy ($P = 0.001$) was also associated with more courses of systemic steroids. The presence of more than one food allergy was also associated with increased number of hospitalizations ($P = 0.000$) and courses of systemic steroids ($P = 0.000$). Asthma severity upon presentation to the clinic was also a predictor of asthma morbidity. Allergy to egg or fish, passive smoke exposure in the home, gastroesophageal reflux, and environmental allergens were not found to be statistically significant predictors of asthma morbidity. This was the first study to find an association between clinical food allergy to milk and peanut and asthma morbidity.

More children in the food allergy group presented with atopic dermatitis, which worsened immediately after eating certain foods with egg, milk, or peanuts. Previous studies have found an association between atopic dermatitis and food allergy,^{21,22} and an association between atopic dermatitis and persistent wheezing in childhood.²³ Our study suggests that food allergy may be the link between atopic dermatitis and asthma morbidity. Atopic dermatitis was found to be significant only in predicting the courses of systemic steroids ($P = 0.043$) when measuring the effect that the number of food allergies had on steroid use. It was not found to be significant in predicting number of hospitalizations in any of our regressions and was not a significant predictor for steroid use when controlling for specific food allergies. Our findings suggest that children with specific food allergies often present with worsening atopic dermatitis and that their food allergy may be the link between atopic dermatitis with asthma morbidity. However, a future study needs to follow these children over a longer period of time to better assess the role of atopic dermatitis in predicting wheezing through later childhood.

In our study, children with food allergies, on average, presented to the allergy clinic prior to the development of persistent asthma symptoms but later went on to develop persistent asthma symptoms even when their food allergies waned. More than 65% (58) of the children in

the food allergy group had mild intermittent symptoms at their first documented allergy visit, and all eventually went on to develop persistent asthma symptoms requiring inhaled steroids by the last documented visit.

Our study is unique in that it focuses on children followed by an outpatient allergy practice who attended at least 75% of their appointments for 2 years or more. This allowed us to control for appropriate asthma education, compliance, and access to health care. Additionally, our study included children with a diagnosis of food allergy in the first years of life when food allergy can serve as an early marker for asthma morbidity. Most of the children also had mild asthma symptoms at presentation, which differs from previous studies looking at the relationship between food allergy and life-threatening asthma.⁶ It further expands upon Wang et al.'s³ association between food sensitization and asthma morbidity and captures the effect that clinically significant food allergy supported by history and IgE levels greater than 95% PPV has on steroid use and hospital admissions.

There are some potential limitations to this study. First, the diagnosis of food allergy was made in the absence of a double-blind, placebo-controlled food challenge. Because it would have been unethical and impractical to do a double-blind, placebo-controlled food challenge, all patients had to have a food-specific IgE level greater than 95% PPV and a positive SPT, which is a specific definition accepted by many food allergists⁷ in the literature. With this specific definition, we were only able to include five children with fish allergy. Perhaps, future studies will include more children with fish allergy and broaden the study to include children with wheat and soy allergy.

Additionally, many children with food allergy (Group A) presented to the allergy clinic at a younger age with mild symptoms and were followed for shorter periods of time, yet they were compared with a majority of children without food allergy (Group B) who were referred to manage their asthma after developing persistent or more severe symptoms at an older age. A future prospective study could include more children with food

allergy who have already developed persistent asthma symptoms and follow both study patients and controls for the same period of time to assess the effect early food allergy has on asthma morbidity throughout later childhood.

Another limitation to a retrospective study is that there may be some bias in the symptoms recorded. We wanted to control for medical compliance by including only those children who attended more than 75% of their documented outpatient follow-up visits. We also included subjects whose medical records revealed a satisfactory compliance with food avoidance and adherence to daily-inhaled corticosteroids for asthma prevention, but these subjective criteria very well could be biased.

To our knowledge, this is the first time that a study has found an association between milk and peanut allergy and asthma morbidity in an outpatient practice. Because patients are often diagnosed with food allergy before the onset of persistent asthma symptoms, food allergy can help serve as an early marker for asthma severity. Asthma specialists can focus on early prevention and anticipatory guidance to reduce asthma morbidity and mortality in an outpatient setting. Future studies are still needed to assess the effect food allergy has on the pathophysiology of airway reactivity and whether early food avoidance would decrease asthma morbidity and persistent symptoms throughout later childhood.

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