e-ISSN: 0976-822X, p-ISSN:2961-6042

Available online on http://www.ijcpr.com/

International Journal of Current Pharmaceutical Review and Research 2023; 15(10); 745-750

Original Research Article

A Hospital Based Observational Assessment of the Food Related Allergies in Children with Asthma

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Received: 09-08-2023 / Revised: 14-09-2023 / Accepted: 28-10-2023
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Conflict of interest: Nil

Abstract

Aim: The aim of the present study was to estimate the proportion of children with asthma who have food related respiratory symptoms and to correlate it with (a) skin prick test (SPT) results and (b) level of asthma control.

Material & Methods: This cross-sectional study involved children with asthma, aged ≥6 years attending the department of pediatrics at Jawaharlal Nehru medical College and Hospital, Bhagalpur, Bihar, India from February 2022 to December 2022.Basic demography and clinical details were recorded. In subjects with a history of food allergy, skin prick test (SPT) was done using Allergo SPT according to guidelines recommended by British Society of Allergy and Clinical Immunology (BSACI). Asthma control was assessed using asthma control test (ACT) and childhood ACT questionnaire.

Results: The onset of asthma in the first year of life was significantly associated with life-threatening asthma (OR, 6.48; 95% CI, 1.36-30.85; P = .016); cases had asthma develop at 10.8 compared with 28.8 months for the controls (P < .001). Furthermore, cases had been more frequently admitted with asthma than controls (P = .014). Cases and controls were sensitized to an average of 3.9 and 1.9 allergens, respectively. The presence of sensitization to 4 or more allergens was found to be a risk factor for life-threatening asthma. In the univariate analysis, only sensitization to dog or foods was significantly associated with life-threatening asthma. Food allergy was found to be a significant risk factor for life-threatening asthma; 50% of cases had food allergy compared with only 10% of the controls (P = .006).

Conclusion: Our findings have important implications for children with coexistent asthma and food allergies. Food allergy is seen in the first few years of life and is potentially a useful marker that would allow increased supervision of this group of high-risk children with asthma to reduce subsequent asthma morbidity and mortality.

Keywords: Food allergy, life-threatening asthma, lung function

Introduction

Bronchial asthma is a long-term disorder of respiratory tract characterized by airway inflammation and hyperactivity. [1,2] Significant surge in its prevalence is noted in recent time globally. [3,4] As per World Health Organization (WHO) 2005 estimate, [5] 300 million of world's population are suffering from asthma and 255,000 among them die of asthma. Majority of deaths due to asthma occur in poor and resource-limited countries. [6] In India, around 57,000 deaths in a year were attributed to asthma. [5]

Food allergen sensitization is recognized as an important modifiable risk factor for asthma exacerbation. [6] Food-induced anaphylaxis is also an important cause of acute severe/life-threatening asthma exacerbation. [7] Although food allergens can vary across regions depending upon socio-cultural characteristics and availability of particular food in the locality, globalization and increased social movement can bring people in contact with food from other countries or cultures and could be a reason for finding the increasing prevalence of food allergy in communities in which they had been

considered rare in the past. [8,9] Regional data regarding common food allergens and its effect on asthma symptom control are scarce and limited to a few geographic locations at present. [10] Allergy and allergic disorders result from a complex interaction between genetic, environmental, and multiple lifestyle factors; thus, a country such as India offers an excellent platform to investigate epidemiology and natural course of allergic diseases. The role of allergic inflammation in pediatric asthma has now been clearly established. [11] However, few studies have looked at atopy as a risk factor for life-threatening asthma in childhood. In eczema, increasing sensitization to aeroallergens correlates with disease severity. [12] A direct correlation exists between the number of allergens a child is sensitized to and the degree of bronchial hyperreactivity [13] and impaired lung function. [14] However, the authors did not define food allergy by history, skin testing, specific IgE testing, or challenge, because they were primarily interested in assessing frequent β-agonist use as a risk factor. Sensitization to inhalants and foods has also been shown to be a risk factor for brittle asthma in adults. [15] It has been estimated that 4% to 8% of children and teenagers with asthma have coexistent food allergy, making food allergy a potentially significant risk factor for life-threatening asthma. [16,17]

The primary objectives of our study were (a) to find out the proportion of children with asthma who have food-induced respiratory allergy symptoms and to correlate it with skin prick test (SPT) results (b) to study the correlation of food allergy with level of asthma control. The secondary objective was to describe the factors associated with food allergy in children with asthma.

Material & Methods

This cross-sectional study involved children with asthma, aged ≥6 years attending the department of pediatrics at Jawaharlal Nehru medical College and Hospital, Bhagalpur, Bihar, India from February 2022 to December 2022. Basic demography and clinical details were recorded. In subjects with a history of food allergy, skin prick test (SPT) was done using Allergo SPT according to guidelines recommended by British Society of Allergy and Clinical Immunology (BSACI). Asthma control was assessed using asthma control test (ACT) and childhood ACT questionnaire.

Inclusion Criteria

Children 6 years and above regularly attending the pediatrics department with clinical/ spirometry evidence of asthma were included for the study after obtaining informed written consent from parent and assent from child (8 years and above).

Exclusion Criteria

Children who had eczema, who were on drugs that can interfere with interpretation of skin prick test those with brittle asthma, or those with acute exacerbation were excluded from the study.

Methodology:

We were able to recruit 100 cases and 100 controls. Basic demographics, clinical and treatment details were collected from the parents. For the diagnosis of food allergy, a history of symptoms attributable to a particular type of food along with SPT positivity for that particular food was considered as food allergy. The procedure of SPT was carried out as per the guidelines laid down by the British Society of Allergy and Clinical Immunology (BSACI) and emergency drugs and equipment to deal with the rare possibility of anaphylaxis were kept ready. A drop (10 μL) of the suspected food allergen was placed on the forearm and a sterile lancet was used to prick the skin through the drop without causing bleeding. The allergen drops, including test allergens and positive and negative controls, were placed at a distance of 2 cm from each other to avoid cross reaction and were marked with an alphabet for identification. Twenty minutes after the prick, the site was examined for wheal and flare response and compared with positive and negative control. The test was considered as positive if a wheal greater than 3 mm, measured with a transparent scale, was produced, and reported as negative if there was no wheal and flare or if it was 3 mm or lesser. Children with reported food allergy symptoms and SPT positivity were diagnosed as having food allergy to that particular food. Antigens (Allergo SPT), procured from Merck, were used for SPT. Asthma control was defined based on childhood asthma control test C-ACT/ ACT scores. Children having a score of 20 or more were labeled as well controlled, those with 16 to 19 were labeled as partially controlled, and those with 15 or less were labeled as poorly controlled.

Statistical Analysis

Kolmogorov-Smirnov test was used to check the normality of data. Significance for continuous non-normal data was assessed using Mann-Whitney test and proportions using chi square test. For correlation, Spearman's correlation coefficient was used. SPSS version 23 was used for analysis.

Results

Table 1: Details of cases and controls

	Cases (100) N%	Controls (100)
Gender		
Male	65 (65)	68 (68)
Female	35 (35)	32 (32)
Average age at exacerbation (mo)	118 (range, 22-192)	110 (range, 24-192)
Parental occupation (%)		
Professional	32 (32)	22 (22)
Skilled	42 (42)	20 (20)
Semi-skilled/unskilled	6 (6)	30 (30)
No income	20 (20)	28 (28)
Mean interval between exacerbation and assessment for study (mo)	20.3 (range, 9-45)	18.0 (range, 5-41)

The time between the index asthma exacerbation and date of assessment was identical for cases and controls.

Table 2: Asthma history

	Cases: n = 100 (%)	Controls: n = 100 (%)	Odds ratio 95%	CI	P value
Asthma developed in first year of life	70 (70)	36 (36)	6.48	1.36-30.85	.016
No. with frequent (4 or more) previous admissions with asthma	58 (58)	22 (22)	14.20	1.77-113.59	.016
No. previously ventilated for asthma 3 months before presentation	15 (15)	0	-	-	-
Wheeze more than 3 times/wk	50 (50)	23 (23)	12.56	1.53-103.13	.014
Use of reliever more than twice/wk	60 (60)	64 (64)	2.45	0.78-7.76	.122
Daily use of inhaled steroids	70 (70)	25 (25)	6.15	1.70-22.30	.006
400 µg or more daily beclomethasone equivalent	48 (48)	40 (20)	3.791	0.980-14.674	.054
Frequent wheeze or cough with exercise	58 (58)	34 (34)	3.17	0.80-12.64	.105
Long-acting bronchodilator	35 (35)	12 (12)	4.22	1.08-16.54	.039

The onset of asthma in the first year of life was significantly associated with life-threatening asthma (OR, 6.48; 95% CI, 1.36-30.85; P = .016); cases had asthma develop at 10.8 compared with 28.8 months for the controls (P < .001). Furthermore, cases had been more frequently admitted with asthma than controls (P = .014).

Table 3: Allergen sensitization

Sensitization to	Cases (%)	Controls (%)	Odds ratio 95%	CI	P value
Grass pollen	54 (54)	32 (32)	4.00	0.80-20.02	.095
Tree pollen	20 (20)	15 (15)	2.17	0.36-12.94	.395
Alternaria	5 (5)	3 (3)	-	-	-
Cladosporium	5 (5)	2 (2)	2.00	0.13-31.98	.624
Aspergillus	5 (5)	5 (5)	1.00	0.053-18.92	1.000
Dog	38 (38)	11 (11)	6.34	1.29-30.74	.022
Cat	40 (40)	21 (21)	2.56	0.72-9.12	.147
D pteronyssinus	52 (52)	52 (52)	1.12	0.35-3.57	.845
D farinnae	28 (28)	25 (25)	1.12	0.36-3.49	.845

Any aeroallergens	72 (72)	62 (62)	1.90	0.46-7.85	.377
Any food allergens	52 (52)	12 (12)	6.90	1.45-32.78	.015
4 or more allergens	42 (42)	14 (14)	5.26	1.07-25.86	.041

Cases and controls were sensitized to an average of 3.9 and 1.9 allergens, respectively. The presence of sensitization to 4 or more allergens was found to be a risk factor for life-threatening asthma. In the univariate analysis, only sensitization to dog or foods was significantly associated with life-threatening asthma.

Table 4: Food allergy and other allergic diagnoses

	Cases (%)	Controls (%)	Odds ratio 95%	CI	P value
Food allergy	50 (50)	12 (12)	8.58	1.85-39.71	.006
Rhinitis	64 (64)	58 (58)	1.72	0.55- 5.41	.350
Eczema	70 (70)	72 (72)	0.86	0.23-3.19	.823
Pet allergy	52 (52)	18 (18)	2.82	0.97-8.19	.056
More than 3 allergic diagnoses	55 (55)	20 (20)	4.42	1.17-16.71	.028

Food allergy was found to be a significant risk factor for life-threatening asthma; 50% of cases had food allergy compared with only 10% of the controls (P = .006).

Discussion

Diseases including asthma, eczema, allergic rhinitis, and food allergy are typically considered as allergic diseases, although the exact association with atopy is frequently debated for eczema and asthma. Nonetheless, such diseases commonly coexist and are common in pediatric populations worldwide. Children affected with one allergic disease frequently develop other allergic diseases. The sequence of disease progression is often referred to as the "atopic march". [18] Food allergen sensitization is recognized as an important modifiable risk factor for asthma exacerbation. [19] Food-induced anaphylaxis is also an important cause of acute severe/life-threatening asthma exacerbation. [20] Although food allergens can vary across regions depending upon socio-cultural characteristics and availability of particular food in the locality, globalization and increased social movement can bring people in contact with food from other countries or cultures and could be a reason for finding the increasing prevalence of food allergy in communities in which they had been considered rare in the past. [21,22] Regional data regarding common food allergens and its effect on asthma symptom control are scarce and limited to a few geographic locations at present. [23]

The onset of asthma in the first year of life was significantly associated with life-threatening asthma (OR, 6.48; 95% CI, 1.36-30.85; P = .016); cases had asthma develop at 10.8 compared with 28.8 months for the controls (P < .001). Furthermore, cases had been more frequently admitted with asthma than controls (P = .014). Cases were significantly more likely to have indicators of severe asthma. Cases and controls were sensitized to an average of 3.9 and 1.9 allergens, respectively. The presence of sensitization to 4 or more allergens was found to be a risk factor for life-threatening asthma. In the univariate analysis, only sensitization to dog or foods was significantly associated with life-threatening asthma. Food allergy was found to be a significant risk factor for life-threatening asthma; 50% of cases had food allergy compared with only 10% of the controls (P = .006). Similar results have been found in the 1 adult study of life-threatening asthma in which the investigators found that a history of food-provoking asthma was the strongest risk factor (OR,5.1). [24] A number of explanations exist for the association between food allergy and life-threatening asthma. The first possibility is that anaphylaxis is misdiagnosed as asthma. This is plausible, because food-induced bronchospasm is often seen in anaphylaxis, and there is often a delay between allergen exposure and the development of respiratory symptoms. [25,26]

One prior study found the association of food allergy and asthma to be independent of aeroallergen sensitization. [27] This finding, while intriguing, has not been replicated. Studies of oral food challenges have found changes in bronchial hyper reactivity (BHR) or lung function [28,29] to be associated with clinical reactivity to food.

Conclusion

Our findings have important implications for children with coexistent asthma and food allergies. Food allergy is seen in the first few years of life and is potentially a useful marker that would allow increased supervision of this group of high-risk children with asthma to reduce subsequent asthma morbidity and mortality. Excellent control of coexistent asthma is an integral part of the management of food allergy in children. Similarly, the accurate diagnosis and management of food allergy must comprise an essential part of the management of skin prick test

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