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(11); 103-107

Original Research Article

A Hospital-Based Observational Study Assessing Food Allergies in Children Diagnosed with Asthma

Samir Kumar[1], Pankaj Kumar[2], Kumar Saurabh[3]

[1]Senior Resident, Department of Pediatrics, Government Medical College and Hospital, Bettiah, Bihar, India

[2]Senior Resident, Department of Pediatrics, Government Medical College and Hospital, Bettiah, Bihar, India

[3]Assistant Professor, Department of Pediatrics, Government Medical College and Hospital, Bettiah, Bihar, India

Received: 14-06-2023 Revised: 26-07-2023 / Accepted: 20-08-2023

Corresponding author: Dr. Pankaj Kumar

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 for one year. 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: 68% were male and 32% were females in cases and 70% were male and 30% were females in controls. Majority of the patients in both groups belonged to 6-10 years of age group. Cases had asthma develop at 10.8 compared with 28.8 months for the controls (P < .001). 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; 52% of cases had food allergy compared with only 10% of the controls (P = .006). Food allergy was found to be a significant risk factor for life-threatening asthma; 52% 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

Asthma is one of the most common long-term childhood conditions of which approximately 9% of children are affected by it. [1] Asthma is defined as a chronic respiratory disease characterized by recurrent attacks of wheeze and breathlessness. Exacerbations of asthma are the most common reason for hospital admission in the pediatric age group. A number of risk factors have been highlighted for life-threatening asthma, including markers of asthma severity, the frequent use of bronchodilators, poor compliance, low socioeconomic status, psychological problems, exposure to cigarette smoke, and previous life-threatening exacerbations. [2-5] A direct correlation exists between the number of allergens a child is sensitized to and the degree of bronchial hyper reactivity [6] and impaired lung function. [7] 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. [8,9] Food allergen sensitization is recognized as an important modifiable risk factor for asthma exacerbation. [10]

Food-induced anaphylaxis is also an important cause of acute severe/life-threatening asthma exacerbation. [11] Food allergy is a prevalent pediatric condition affecting 2–6% of children and 1 to 3.2% of adults. [12,13] Other studies have suggested that food allergen sensitization [14,15] is a risk factor for the development of asthma. The respiratory symptoms that occur in food allergic

reactions commonly include rhinitis, bronchospasm, cough, and laryngeal edema. [16] Asthma and food allergy have been commonly shown to coexist with each other, especially as they often share risk factors (family) history of allergy, atopic eczema, and asthma) but the way in which they interact and influence each other is yet to be fully understood. [1]

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 was a cross-sectional study done at Department of Pediatrics, Government Medical College and Hospital, Bettiah, Bihar, India for one year. Our study population consisted of people belonging predominantly to Bihar Region and a rural background.

Inclusion criteria

Children 6 years and above regularly attending the department of pediatrics 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 SPT, those with brittle asthma, or those with acute exacerbation were excluded from the study.

Methodology:

The sample size required was 83 in each group assuming α error of 5%, power of 80%, expected proportion of uncontrolled asthma as 20% and 40% among those without and with food allergy, respectively. However, we were able to recruit 100 cases and 200 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 (200)
Gender		
Male	68 (68)	140 (70)
Female	32 (32)	60 (30)
Age groups		
6-10	44 (44)	80 (40)
11-14	36 (36)	70 (35)
>14	20 (20)	50 (25)
Average age at exacerbation (mo)	118 (range, 22-192)	110 (range, 24-192)
Parental occupation (%)		
Professional	34 (34)	40 (20)
Skilled	40 (40)	44 (22)
Semi-skilled/unskilled	6 (6)	28 (28)
No income	20 (20)	68 (34)

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. 68% were male and 32% were females in cases and 70% were male and 30% were females in controls. Majority of the patients in both groups belonged to 6-10 years of age group.

Table 2: Asthma history

	Cases: n = 100 (%)	Controls: n = 200 (%)	Odds ratio 95%	CI	P value
Asthma developed in first year of life	72 (72)	70 (35)	6.48	1.36-30.85	0.016
No. with frequent (4 or more) previous admissions with asthma	58 (58)	40 (20)	14.20	1.77-113.59	0.016
No. previously ventilated for asthma 3 months before presentation	16 (16)	0	-	-	-
Wheeze more than 3 times/wk	50 (50)	40 (20)	12.56	1.53-103.13	0.014
Use of reliever more than twice/wk	60 (60)	124 (62)	2.45	0.78-7.76	0.122
Daily use of inhaled steroids	70 (70)	48 (24)	6.15	1.70-22.30	0.006
400 μg or more daily beclomethasone equivalent	48 (48)	40 (20)	3.791	0.980-14.674	0.054
Frequent wheeze or cough with exercise	58 (58)	70 (35)	3.17	0.80-12.64	0.105
Long-acting bronchodilator	35 (35)	20 (10)	4.22	1.08-16.54	0.039

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). 16 of the cases (16%) had been previously ventilated for asthma compared with none of the controls. Cases were significantly more likely to have indicators of severe asthma.

Table 3: Allergen sensitization

Sensitization to	Cases (%)	Controls (%)	Odds ratio 95%	CI	P value
Grass pollen	52 (52)	60 (30)	4.00	0.80-20.02	.095
Tree pollen	20 (20)	28 (14)	2.17	0.36-12.94	.395
Alternaria	5 (5)	4 (2)	-	-	-
Cladosporium	5 (5)	4 (2)	2.00	0.13-31.98	.624
Aspergillus	5 (5)	8 (4)	1.00	0.053-18.92	1.000
Dog	38 (38)	20 (10)	6.34	1.29-30.74	.022
Cat	40 (40)	40 (20)	2.56	0.72-9.12	.147
D pteronyssinus	52 (52)	100 (50)	1.12	0.35-3.57	.845
D farinnae	28 (28)	48 (24)	1.12	0.36-3.49	.845
Any aeroallergens	72 (72)	128 (64)	1.90	0.46-7.85	.377
Any food allergens	52 (52)	28 (14)	6.90	1.45-32.78	.015
4 or more allergens	42 (42)	30 (15)	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	52 (52)	20 (10)	8.58	1.85-39.71	0.006
Rhinitis	65 (65)	120 (60)	1.72	0.55- 5.41	0.350
Eczema	72 (72)	150 (75)	0.86	0.23-3.19	0.823
Pet allergy	50 (50)	40 (20)	2.82	0.97-8.19	0.056
More than 3 allergic diagnoses	54 (54)	42 (21)	4.42	1.17-16.71	0.028

Food allergy was found to be a significant risk factor for life-threatening asthma; 52% 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". [17] Food allergen sensitization is recognized as an important modifiable risk factor for asthma exacerbation. [18] Food-induced anaphylaxis is also an important cause of acute severe/life-threatening asthma exacerbation. [19] 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. [20,21] Regional data regarding common food allergens and its effect on asthma symptom control are scarce and limited to a few geographic locations at present. [22]

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 ($P = .014$). 16 of the cases (16%) had been previously ventilated for asthma compared with none of the controls. 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; 52% 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). [23] 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. [24,25]

One prior study found the association of food allergy and asthma to be independent of aeroallergen sensitization. [26] This finding, while intriguing, has not been replicated. Studies of oral food challenges have found changes in bronchial hyper reactivity (BHR) or lung function [27,28] to be associated with clinical reactivity to food. Carlos et al. in a report from Brazil on risk factor assessment in childhood asthma have shown association of prematurity, maternal asthma, exposure to pets during infancy, antibiotics use in first 6 months of life, current rhinitis, sharing bed room, history of atopy. [29] In a similar report from South Korea, risk factor analysis related to asthma severity showed significant association of tobacco smoke, exposure to dog dander's and absence of home air purifier in children. [30] Likewise, a Mexican study with 999 children have documented that association of exposure of smoking, common cold in early life, kitchen indoors, exposure to pets and mould were significant risk factors whereas breast feeding more than 3 months, caesarean section and having more than one sibling in the family were found to be protective. [31]

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 childhood asthma.

References

1. Caffarelli C, Garrubba M, Greco C, Mastrorilli C, Povesi Dascola C. Asthma and food allergy in children: is there a connection or interaction? Frontiers in pediatrics. 2016 Apr 5; 4:34.
2. Strunk RC, Nicklas RA, Milgrom H, Davis ML, Ikle DN. Risk factors for fatal asthma. In: Sheffer AL, editor. Fatal asthma. New York: Marcel Dekker; 1998.
3. Moore BB, Wagner R, Weiss KB. A community-based study of near-fatal asthma. Annals of Allergy, Asthma & Immunology. 2001 Feb 1;86(2):190-5.
4. Garrett JE, Lanes SF, Kolbe J, Rea HH. Risk of severe life threatening asthma and beta agonist type: an example of confounding by severity. Thorax. 1996 Nov 1;51(11):1093-9.
5. Kravis LP. An analysis of fifteen childhood asthma fatalities. Journal of Allergy and Clinical Immunology. 1987 Sep 1;80(3):467-72.
6. Sears MR, Burrows B, Herbison GP, Holdaway MD, Flannery EM. Atopy in childhood. II. Relationship to airway responsiveness, hay fever and asthma. Clinical & Experimental Allergy. 1993 Nov; 23(11): 949-56.
7. Schwartz J, Weiss ST. Relationship of skin test reactivity to decrements in pulmonary function in children with asthma or frequent wheezing. American journal of respiratory and critical care medicine. 1995 Dec;152(6):2176-80.

8. Bousquet J, Neukirch F, Noyola A, Michel F-B. Prevalence of food allergy in asthma. Pediatr Allergy Immunol 1992; 3:206-13.
9. Sicherer SH, Sampson HA. The role of food allergy in childhood asthma.Immunol Allergy Clin North Am 1998;18:49-60.
10. Global Initiative for Asthma [Internet]. Global Strategy for Asthma Management and Prevention. 2017
11. Roberts G, Patel N, Levi-Schaffer F, Habibi P, Lack G. Food allergy as a risk factor for life-threatening asthma in childhood: a case-controlled study. Journal of Allergy and Clinical Immunology. 2003 Jul 1;112(1):168-74.
12. Osterballe M, Hansen TK, Mortz CG, Høst A, Bindslev-Jensen C. The prevalence of food hypersensitivity in an unselected population of children and adults. Pediatric Allergy and Immunology. 2005 Nov;16(7):567-73.
13. Woods RK, Thien F, Raven J, Walters EH, Abramson M. Prevalence of food allergies in young adults and their relationship to asthma, nasal allergies, and eczema. Annals of Allergy, Asthma & Immunology. 2002 Feb 1;88(2): 183-9.
14. Kulig M, Bergmann R, Tacke U, Wahn U, Guggenmoos-Holzmann I. Long-lasting sensitization to food during the first two years precedes allergic airway disease. The MAS Study Group. Germany Pediatr Allergy Immunol. 1998; 9:61–7.
15. Penard-Morand C, Raherison C, Kopferschmitt C, Caillaud D, Lavaud F, Charpin D, Bousquet J, Annesi-Maesano I. Prevalence of food allergy and its relationship to asthma and allergic rhinitis in schoolchildren. Allergy. 2005; 60:1165–71.
16. . Leung TF, Lam CW, Chan IH, Li AM, Tang NL. Sensitization to common food allergens is a risk factor for asthma in young Chinese children in Hong Kong. J Asthma. 2002; 39:523–9.
17. Shaker M. New insights into the allergic march. Current opinion in pediatrics. 2014 Aug 1;26(4):516-20.
18. Global Initiative for Asthma [Internet]. Global Strategy for Asthma Management and Prevention. 2017.
19. Roberts G, Patel N, Levi-Schaffer F, Habibi P, Lack G. Food allergy as a risk factor for life-threatening asthma in childhood: a case-controlled study. Journal of Allergy and Clinical Immunology. 2003 Jul 1;112(1):168-74.
20. Castelain M. Globalisation and allergy. European Journal of Dermatology. 2011 Sep 1;21(4):472-8.
21. Tang ML, Mullins RJ. Food allergy: is prevalence increasing? Internal medicine journal. 2017 Mar;47(3):256-61.
22. Devdas JM, Mckie C, Fox AT, Ratageri VH. Food allergy in children: an overview. The Indian Journal of Pediatrics. 2018 May;85: 369-74.
23. Ernst P, Habbick B, Suissa S, Hemmelgarn B, Cockcroft D, Buist AS, Horwitz RI, Mcnutt M, Spitzer WO. Is the association between inhaled beta-agonist use and life-threatening asthma because of confounding by severity? American Review of Respiratory Disease. 1993 Jul 1; 148:75.
24. Pumphrey RS. Lessons for management of anaphylaxis from a study of fatal reactions. Clinical and experimental allergy. 2000 Aug 30;30(8):1144-50.
25. Sampson HA, Mendelson L, Rosen JP. Fatal and near-fatal anaphylactic reactions to food in children and adolescents. New England Journal of Medicine. 1992 Aug 6;327(6):380-4.
26. Penard-Morand C, Raherison C, Kopferschmitt C, Caillaud D, Lavaud F, Charpin D, Bousquet J, Annesi-Maesano I. Prevalence of food allergy and its relationship to asthma and allergic rhinitis in schoolchildren. Allergy. 2005 Sep;60(9):1165-71.
27. James JM, Bernhisel-Broadbent J, Sampson HA. Respiratory reactions provoked by double-blind food challenges in children. American journal of respiratory and critical care medicine. 1994 Jan;149(1):59-64.
28. James JM, Eigenmann PA, Eggleston PA, Sampson HA. Airway reactivity changes in asthmatic patients undergoing blinded food challenges. American journal of respiratory and critical care medicine. 1996 Feb;153(2):597-603.
29. Neto AC, Solé D, Hirakata V, Schmid LS, Klock C, Barreto SS. Risk factors for asthma in schoolchildren in Southern Brazil. Allergologia et Immunopathologia. 2020 May 1;48(3):237-43.
30. Lee E, Song DJ, Kim WK, Suh DI, Baek HS, Shin M, Yoo Y, Kim JT, Kwon JW, Jang GC, Lim DH. Associated factors for asthma severity in Korean children: a Korean childhood asthma study. Allergy, asthma & immunology research. 2020 Jan 1;12(1):86-98.
31. Larenas-Linnemann D, Romero-Tapia SJ, Virgen C, Mallol J, Bacab MA, García-Marcos L. Risk factors for wheezing in primary health care settings in the tropics. Annals of Allergy, Asthma & Immunology. 2020 Feb 1;124(2): 179-84.