ASIAN PACIFIC JOURNAL OF ALLERGY AND IMMUNOLOGY (1999) 17: 203-206

SPECIAL ARTICLE

Food Allergy in Children-The Singapore Story

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SUMMARY **The study of food allergy in Singaporean children is still in its infancy. Confusion and misunderstanding is common among the public. Even so, we have found certain unique features regarding food allergy among Singaporean children. "Bird's nest" has been shown to be the most common cause of anaphylaxis requiring medical attention. This allergen has not been described before. Peanuts and tree nuts are extremely uncommon causes of anaphylaxis, unlike the West. However, the pattern of sensitization to foods in children as shown by skin prick test is similar to other Western populations. The reasons for the difference between the profile foods responsible for anaphylactic reactions in our population and those of the western population, despite the similarity in sensitization profiles, are still unclear.**

There are many types of adverse reactions to food. Food allergy implies an underlying immunologic reaction mechanism. It is a term that is often misunderstood and misused by the public in that many diseases and symptoms are attributed to food. Public perception of the prevalence of food allergy is also higher than the true prevalence as confirmed by appropriate blinded food challenges. The situation in Singapore is no different, with parents blaming different foods for a variety of clinical manifestations. This review will discuss the studies that have been carried out in Singapore.

Definitions

Food allergy denotes an immunologically mediated reaction to food. This may be IgE mediated-the classic immediate type hypersensitivity response. There is also some evidence for the role of cell-mediated allergy in food-allergic patients with atopic dermatitis.[1] The evidence for other possible immunological mechanisms in food allergy is scant. Thus, IgE-mediated reactions, which have the potential for life-threatening anaphylaxis, are the best understood of the immune mediated reactions.

Food intolerance describes an abnormal physiological response to food. This may not be immunologically mediated. Some foods contain chemicals that provoke a pharmacological response. For example, monosodium glutamate in cooking may cause headache and flushing.[2] In others, there may be a metabolic cause for the food intolerance. Lactase deficiency, which is common amongst orientals, may manifest as diarrhea and bloating after the ingestion of milk, and is also relatively common in our population.[3] Finally, food intolerance may also be due to toxic reactions if bacterial toxins or chemicals have contaminated the food.

Prevalence

There is limited data on the prevalence of food allergy in Singapore. A population-based questionnaire of 6,404 Singapore chil-

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dren aged 5-12 years estimated the prevalence to be 4-5%.[4,5] The actual figure may be lower as questionnaire surveys tend to overestimate the true prevalence. The prevalence of cow's milk allergy was found to be 2.2% in Danish infants.[6] The true prevalence is probably not more than 5% during the first few years of life, and declines thereafter. It is estimated to be about 1% in adults.[7]

Table 1 Foods triggering anaphylaxis and the age of presentation

Food allergen	N (%)	[1]Median age (range)
Egg and milk	14 (11)	0.7 (0.1-4.0)
Bird's nest	34 (27)	4.5 (2.0-14.0)
Chinese herbs	9 (7)	5.0 (2.0-11.0)
Others[2]	37 (30)	7.0 (0-15.0)
Crustacean seafood	30 (24)	11.0 (2.0-15.0)
Total	124 (100)	5.0 (0-15.0)

[1]Age refers to age in years of presentation.
[2]Others include a wide spectrum of food types including chicken, duck, ham, fruits (banana, rambutan), cereals, gelatin (in the form of jelly dessert) and spices.
Adapted from published data[8]

Table 2 IgE sensitization to food as measured by skin prick test or specific IgE antibodies in atopic children less than 5 years old

Food	No. with positive test	Rank
Shellfish	50	1
Milk	23	2
Egg	22	3
Wheat	19	4
Peanut	14	5
Soy	11	6
Total	139	

Adapted from Hill *et al.*[4]

Immediate reactions to food

The majority of immediate hypersensitivity reactions to food are IgE-mediated. These reactions can involve the skin, respiratory tract, gastrointestinal tract or cause systemic anaphylaxis. They occur within minutes of ingestion of the offending food. A retrospective study was conducted to evaluate the pattern of food-induced anaphylaxis in Singapore.[8] Out of 868 children that presented with acute allergic reactions (angioedema, urticaria, generalized flushing, wheezing and/or hypotension), 14.3% was suspected to be due to food. This was based on clinical history alone with few diagnostic tests done. This figure was lower than a previous review conducted in the United States of 266 children and adults presenting with anaphylaxis.[9] In that study, food was the offending agent in 34%.

Interestingly, the foods that caused the symptoms in our population were very different from those described by the western population. In the study by Kemp, crustaceans accounted for 29% and peanuts for 22% of the reactions. In our study, we found that the single most common trigger was "bird's nest". This is a Chinese delicacy made with the edible nests of the swiftlet, *Collocalia* spp. This was followed by crustaceans (24%), egg and cow's milk (11%), and others (Table 1). "Bird's nest" is made from the saliva of the swiftlets and we have documented that the reaction is IgE mediated and the major allergen is a 66 kD glycoprotein.[10] Allergy to "bird's nest" has not been previously described and shows the importance of cultural practices on the development of disease.

The other point of interest was that peanuts and tree nuts were not found to cause anaphylaxis in our population. This is stark contrast to the 'epidemic' of peanut anaphylaxis/allergy that is being documented in the American and British populations.[11] In a recent nation-wide survey in the United States, the overall prevalence of symptomatic peanut allergy in their population was estimated at 1.1%.[12] The reasons for this marked difference in prevalence of peanut allergy between the West and our community is uncertain. The lack of exposure to peanuts is not likely to be a significant factor. A study of the pattern of food sensitization in our atopic children less than 5 years of age shows that sensitization to shellfish and peanut was most common, followed by milk

and egg (Table 2), thus indicating exposure to these allergens at an early age.[4] This pattern of sensitization is similar to other studies where the foods most commonly incriminated in children are eggs, milk, peanuts, tree nuts, shrimp, soy, fish and wheat.[13]

It is tempting to speculate that the absence of a peanut allergy 'epidemic' in our population may be related to similar reasons responsible for the rising prevalence of allergy and asthma observed with increasing affluence and westernization. If this is so, then we should anticipate an 'epidemic' of peanut allergy occurring in our population in the next one or two generations.

Role of food allergy in eczema

For the individual patient, the evaluation of food allergy in a patient with eczema may be difficult, as there is a high prevalence of food-specific IgE, which may not be clinically relevant. In the absence of a convincing history of acute reactions, or significantly high value of specific IgE level (by CAP system FEIA; above 6 kU(A)/l for egg, 32 kU(A)/l for peanut and 20kU(A)/l for fish),[14] there is a need to rely on food challenges. With these criteria, it has been shown that approximately only 20% of children with atopic dermatitis have clinically significant food allergy.[15] In those with refractory and moderately severe atopic dermatitis, this figure may be up to 37%.[16] Our studies on children with eczema also showed a high rate of sensitization to foods (50%), however, food challenge positivity was documented in only one patient.[17]

Treatment

Elimination of the offending allergen is the only proven therapy. In Singapore, the food control department of the Ministry of Health requires that a complete list of the ingredients used in the food should be stated on the label. However, there is always the lurking hazard of inadvertently consuming the offending food that may be hidden in the hugely varied Asian diet. With the likelihood of an increase in prevalence of food allergy along with the other atopic disorders, there is also a pressing need to improve public awareness of food allergy. As the consumption of food is an integral part of our daily activities, school teachers and all those involved in the food industry should be aware of the importance of avoidance of the incriminating food allergen in individuals with food allergy problems.

In addition, it is crucial to educate our patients and parents of younger patients on the importance of strict allergen avoidance. This includes the recognition of chemical names on food labels, eg. casein and whey for cow's milk. Especially for those with life threatening anaphylactic reactions, they should be provided with a management plan in case of accidental ingestion of the offending allergen. In these situations, the availability of self-injectable adrenalin have been proven to be life saving.

Conclusion

Much work still needs to be done in the field of food allergy in Singapore. Of importance will be understanding the role of specific allergens such as "bird's nest". We also need to develop more sophisticated techniques of diagnosis. This will help not only in the understanding of the pathophysiology of the allergy, and will aid in managing what may be a potentially life-threatening reaction. With the recent investigations into novel immunotherapeutic modalities for the prevention and management of food allergy such as the use of DNA vaccines,[17] the prospects of more effective strategies for the management of food allergy appear to be realistic possibility in the not too distant future.

REFERENCES

1. Kondo N, Fukutomi O, Agata H, *et al.* The role of T lymphocytes in patients with food-sensitive atopic dermatitis. J Allergy Clin Immunol 1993; 91: 658-8.
2. Yang WH, Drouin MA, Herbert M, Mao Y, Karsh J. The monosodium glutamate symptom complex: assessment in a double-blind, placebo-controlled, randomized study. J Allergy Clin Immunol 1997; 99: 757-62.
3. Quak SH. Lactose intolerance in Asian children. J Paediatr Child Health 1994; 30: 91-2.
4. Hill DJ, Hoskings CS, Chen YZ, *et al.* The frequency of food allergy in Australia and Asia. Environ Toxicol Pharmacol 1997; 4: 101-10.
5. Lee BW, Chew FT, Goh DYT. Changing prevalence of childhood allergic diseases in Singapore. Proceedings of the 5th West-Pacific Allergy Symposium & 7th Korea-Japan Joint Allergy Symposium. 1997; pp. 17-22.
6. Host A, Halken SA. A prospective study of cow's milk allergy in Danish infants during the first 3 years of life. Allergy 1990; 45: 587-96.
7. Sampson HA, Burks AW. Mechanisms of food allergy. Am Rev Nuts 1996; 16: 161-77.
8. Goh DLM, Lau YN, Chew FT, Shek LPC, Lee BW. Pattern of food-induced anaphylaxis in children of an Asian community. Allergy 1999; 54: 84-6.
9. Kemp SF, Lockey RF, Wolf BL, Lieberman P. Anaphylaxis: A review of 266 cases. Arch Intern Med 1995; 155: 1749-54.
10. Goh DLM, Chew FT, Chua KY, Chay OM, Lee BW. Edible 'Bird's Nest'-induced anaphylaxis-an under recognized entity? Lancet (submitted).
11. Emmett SE, Angus FJ, Fry JS, Lee PN. Perceived prevalence of peanut allergy in Great Britain and its association with

other atopic conditions and with peanut allergy in other household members. Allergy 1999; 54: 380-5.
12. Sicherer SH, Munoz-Furlong A, Burks AW, Sampson HA. Prevalence of peanut and tree nut allergy in the US determined by a random digit dial telephone survey. J Allergy Clin Immunol 1999; 103: 559-62.
13. Bock SA. A critical evaluation of clinical trials in adverse reactions to foods in children. J Allergy Clin Immunol 1986; 78: 165-74.
14. Sampson HA, Ho DG. Relationship between food-specific IgE concentrations and the risk of positive food challenges in children and adolescents. J Allergy Clin Immunol 1997; 100: 444-51.
15. Hanifin JM. Atopic dermatitis. J Allergy Clin Immunol 1984; 73: 211-26.
16. Eigenmann PA, Sicherer SH, Borkowski TA, Cohen BA, Sampson HA. Prevalence of IgE-mediated food allergy among children with atopic dermatitis. Pediatrics 1997; 463-4.
17. Lee BW, Giam YC, Seah CC, Tan KC. Pattern of food-specific IgE in children with atopic eczema. In Proceedings of the 24th Singapore-Malaysia Congress of Medicine, 1990; p. 180.
18. Krishnendu R, Mao HQ, Huang SK, Leong KW. Oral gene delivery with chitosan-DNA nanoparticles generates immunologic protection in a murine model of peanut allergy. Nature Med 1999; 5: 387-91.