### Some aspects of food allergy

### Sanath P Lamabadusuriya<sup>1</sup>

### Sri Lanka Journal of Child Health, 2004; 33: 3-5

### (Key words: Food allergy)

An adverse reaction to food could be defined as an abnormal clinical reaction related to ingestion of food<sup>1</sup>. It could either be a true food allergy or hypersensitivity due to an underlying immunological reaction or a pseudo-allergy or food intolerance with which has no underlying immunological basis<sup>1</sup>. The incidence of food allergies in adults is 1-2% whilst in children under 3 years of age it is between  $5-8\%^1$ . This is because some children "outgrow" their food allergies after 3 years. Incidence of cow milk protein allergy in children under 2 years is about  $2.5\%^2$ .

# Challenge to gastrointestinal tract and immune system

The small intestine confronts an enormous amount of foreign protein, after the onset of weaning. It has to distinguish between nutrients required for growth and energy needs and foreign pathogens, which are rejected. The intestinal barrier against foreign antigens are either non-immunologic or immunologic<sup>3</sup>. Digestive enzymes, intestinal motility, surface mucus layer and brush-border cell membranes are components of the nonimmunologic barrier<sup>3</sup>. The immunologic barrier consists of gut associated lymphold tissue (GALT), secretory immunoglobulin A (SIgA) and effector cells (e.g. macrophages, mast cells and lymphocytes)<sup>3</sup>. Foreign antigens are cleared by a variety of mechanisms. SIgA antibodies prevent adherence of micro-organisms on to mucosal surface. In addition, specific antibodies produced in the submucosa activate clearance of antigens. Sometimes acquired tolerance to specific antigens prevents activation of immune responses<sup>3</sup>.

### Predisposing factors for allergy

Many factors predispose to food allergy such as genetic factors, levels of IgE in cord blood, maternal tobacco smoking and maternal ingestion of highly allergenic food during the last trimester<sup>3</sup>. The risk of developing atopy based on family history of allergy is shown in Table 1.

<sup>1</sup>Dean and Senior Professor of Paediatrics, Faculty of Medicine, University of Colombo.

### Table 1 Risk of developing atopy based on family history of allergy

Family history Risk

Both parents atopic							
40 - 60%							
Both	parents	atopic	with	same	ne manifestation		
50 - 80%							
One parent atopic						20	-
40%							
One sibling atopic						25	-
35%							
Neither	par	ent	nor	sibliı	ıg	atoj	pic
5 - 15%	)						

Genetic factors are more important in determining allergy in early life, since the allergic phenotype is expressed soon after the interactions between immune system and allergens; in adults allergen-independent environmental factors are more important. Prematurity and low birth weight do not affect the development of allergy<sup>3</sup>.

### Pathogenesis of food allergies

There are 3 major players in this process; the allergen, GI barrier and digestive components and the immune system<sup>3</sup>. The common food allergens incriminated are shown in Table 2.

#### Table 2 Common food allergens

- 1. Cow milk proteins
  - Caseins Whey proteins Beta\_lactoglobulin, Alpha lacto globulin, Bovine serum albumin, Bovine immunoglobulin
- 2. Egg proteins Ovomucoid

Ovalbumin

- 3. Tree nuts and ground nuts Walnut Hazelnut Almonds Brazil nut Peanuts
- 4. Soy Proteins
- 5. Fish and sea food proteins Shrimp Crab Lobster Cod fish Oyster Mussels Cockles etc.
- 6. Fruits
- Apples Pears Cherries Apricots Peaches Pineapple
- 7. Vegetables
  - Spinach Tomatoes Celery

GI food allergies could be exclusively IgE mediated, partially IgE mediated or exclusively cell mediated (non IgE mediated)<sup>1</sup>. In IgE mediated disorders symptoms develop within minutes to 2 hours of ingestion<sup>1</sup>. It is an example of immediate GI hyper-sensitivity and common symptoms are nausea, abdominal pain, colic, vomiting, flatulence and diarrhoea. Other target organs such as the skin and lungs may be affected. Nut allergies, including peanut allergy, are usually life-long and may result in anaphylaxis.

### IgE mediated disorders

The oral allergy syndrome is IgE mediated<sup>1</sup>. Fresh fruits, raw vegetables, tree nuts, peanuts, egg, milk and fish are associated. Symptoms are almost exclusively confined to the oropharynx and consist of pruritus, tingling, angio-oedema of lips, tongue, palate and throat, of rapid onset<sup>1</sup>.

### Mixed IgE and non-IgE mediated disorders

These consist of many disease entities such as eosinophilic oesophagitis, gastritis and gastroenteritis, which may manifest from infancy up to adolescence<sup>4</sup>. Symptoms include intermittent vomiting, food refusal, abdominal pain, dysphagia, irritability, sleep disturbance

and gastro oesophageal reflux. There is peripheral eosinophilla in 50% of patients and on biopsy eosinophilic infiltration of the oesophagus, stomach and intestinal walls becomes evident<sup>4</sup>.

## Exclusively cell-mediated disorders (non-IgE mediated)

There is a variety of exclusively cell-mediated disorders. These are dietary protein enterocolitis syndrome, dietary protein proctitis/ proctocolitis syndrome, dietary protein enteropathy and the well-documented coeliac disease<sup>5</sup>. In proctitis / proctocolitis syndrome, manifestations are seen in early infancy and includes blood streaked stools and anaemia<sup>1</sup>. Such babies may be breast-fed, or formula fed with cow milk or soya protein based formulae.

In dietary protein enteropathy, infants are predominantly affected and clinical manifestations include protracted diarrhoea, vomiting, failure to thrive, abdominal distension, malabsorption, anaemia, oedema and hypoproteinaemia<sup>1</sup>. Coeliac disease due to gluten sensitivity is a classic example<sup>6</sup>. Other food allergens include cow milk, soya, egg, rice, fish and chicken.

### **Coeliac disease**

Ingestion of gluten (found in wheat) results in subtotal or total villous atrophy and hyperplasia of crypts of small intestine. Protracted diarrhoea, malabsorption, steatorrhoea, abdominal distension and failure to thrive are common clinical manifestations<sup>6</sup>. Antigliadin and antiendomysium antibodies are detected in the serum in 90% of patients<sup>6</sup>. A gluten-free diet should be prescribed life-long. It is associated with HLA – DQ2(&DQ8) haplotype<sup>6</sup>.

### Cow-milk protein allergy

It is due to  $\beta 2$  lactoglobulin and is associated with consumption of pasteurized cow milk. Manifestations are seen usually below 6 months of age and include gastrointestinal blood loss, anaemia and chronic constipation. Usually children grow out of it by 2 to 3 years of age<sup>2</sup>.

## Differential diagnosis of gastro intestinal food allergies

As symptoms of food allergies are non specific, differential diagnosis includes other diseases with similar symptoms such as poisoning, acute gastroenteritis, gastro-oesophageal reflux, and ascariasis. Therefore a high degree of suspicion plays an important role in diagnosis coupled with a detailed, relevant history.

### **Diagnosis of food allergy**

In the past, a variety of investigations such as skin prick tests, atopy patch tests, RAST and serum IgE levels have been used<sup>7</sup>. These tests are not very helpful. Double blind, placebo controlled food challenge (DBPCFC) is considered to be the gold standard for diagnosis<sup>8</sup>. However DBPCFC is very tedious in practice and carried out only in a few specialized centres. As mentioned before, a detailed relevant history coupled with a high degree of suspicion plays an important role in clinical situations.

### Management of food allergies

If the allergen is identified, it should be withdrawn. If not, a vegetarian diet, free of animal proteins and artificial food additive should be prescribed. Symptomatic relief would be obtained with antihistamines and steroids. Once patient is free of symptoms, the withdrawn dietary items should be reintroduced gradually one at a time. If symptoms recur on re-introduction, the offending dietary item should be withdrawn<sup>9</sup>.

### **Preventive measures**

These include promotion of breast feeding, avoiding weaning during the first 4 to 6 months, avoidance of aeroallergens, air pollution and passive smoking.

### Food intolerance or pseudo - food allergies

Pseudo allergies have no immunological basis even through they may display similar clinical features<sup>1</sup>. The offending items are shown in Table 3.

The code for food additives is usually displayed on the label; in susceptible patients, extra care should be taken by reading the labels carefully, and avoiding potential harmful agents.

### Table 3 Food additives

- Foods containing histamine or releasing histamine Fresh or Canned tuna, mackerel,
- Preservatives

   Sulphites and derivatives E220 227
   Nitrites E249 252
   Benzoic acid and derivatives E210 219
   Sorbic acid
- Antioxidants
   Butyl hydroxyanisole E 321
   Butyl hydroxytoluene E 321

- Colourings Tartrazine E102
  - Yellow orange E110 Azorubine E122 Amaranth E123 Cochineal red E124 Eythrosine E127 Brilliant black BN E151
- *Flavourings* Glutamates B 550 – 553
- Whitening agents Potassium bromide E 924 Chlorine E 925

### References

- Spergel J M, Pawlowski N A. Food allergy: Mechanisms, diagnosis and management in children. *Paediatr Clin North Am* 2002; 49: 73-96.
- Host A, Husby S, Ostorballe O. A prospective study of cow's milk allergy in exclusively breast-fed infants. *Acta Paediatr Scand* 1988; 77: 663-70.
- Sampson H. Food Allergy. In Kay AB (ed): Allergy and allergic diseases. London; Blackwell Science, 1997, pp 1517-1549.
- 4. Kelly K. Eosinophilic gastroenteritis. *J Pediatr Gastroenterol Nutr* 2000; **30(suppl):** 28-35.
- 5. Dupont C, Heyman M. Food protein-induced enterocolitis syndrome: Laboratory perspectives. J Pediatr Gastroenterol Nutr 2000; **30(suppl):** 50-57.
- Collin P, Kaukinen K, Maki M. Clinical features of coeliac disease today. *Dig Dis Sci* 1999; 17: 100-6.
- Majamaa H, Moisio P, Holm K. Cow's milk allergy: Diagnostic accuracy of skin prick and patch tests and specific IgE. *Allergy* 1999; 54: 851-6.
- Bock A S, Sampson H A, Atkins F M. Double blind placebo-controlled food challenge (DBPCFC) as an office procedure: a manual. J Allergy Clin Immunol 1984; 74: 26-33.
- Sampson H: Food allergy II. Diagnosis and management. J Allergy Clin Immunol 1999; 103: 981-9.