Food Allergy in Childhood

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ABSTRACT

Food allergy (FA) is a common and increasing problem globally and often co-exist with other atopic diseases. Trigger foods differ according to country and ethnic origin. Some FAs are more likely to resolve but some continue lifelong. Teenagers carry the highest risk of fatal anaphylaxis. The main diagnostic tool is allergy focused clinical and diet history, and further supportive allergy tests must be guided by clinical history. Current management consists of strict avoidance of trigger food(s). Management of allergic reactions with regular clinical re-assessment is essential to educate, support, and determine if the condition is resolving by time. Research on FA treatments and prevention is ongoing.

PREVALENCE and CAUSES

The incidence of food allergy (FA) is increasing worldwide and has reached an alarming 3-6% prevalence amongst children in developed countries [1]. FA is said to be the second wave of the allergy epidemic [2], following the first wave of atopic diseases (asthma, allergic rhinitis, and eczema) which emerged in the first half of the 20th-century hand in hand with the adoption of the so-called "Western lifestyle". Though the puzzle is a complex one and beyond the scope of this review, it is important to recognize that there are both genetic and environmental factors at play in the development of FA. Many environmental influences culminate in reduced biodiversity and change in the composition of the human microbiota which has crucial effects on our immune system [3].

CLASSIFICATION and DIAGNOSIS

Food can cause a host of adverse reactions, which are not always allergic to their pathophysiology. Allergy is an immunologically mediated adverse reaction. It is essential to understand the distinction between allergic and non-allergic hypersensitivity reactions to food (e.g. cow's milk protein allergy vs lactose intolerance) as well as different types of FA, as the diagnostic and management approach differs, as does the prognosis. There are two distinct types of FA: IgE-mediated and non-IgE mediated. They present with different clinical signs and symptoms, and the main diagnostic tool for FAs is the allergy focused clinical history [4,5] (Table 1).

There are several supportive diagnostic tests including skin prick tests, specific IgE tests to whole food allergens or single food proteins (called components), and food challenge (or provocation) tests which can be utilized to confirm or rule out IgE-mediated FA. These tests must be carefully selected and interpreted in the light of the clinical history and the importance of this point cannot be overemphasized. These tests are not appropriate for the investigation of non-IgE mediated FA, where the only investigative tool beyond clinical history is an exclusion diet followed by re-introduction to prove causality between suspect food and clinical symptoms.

There are also many unvalidated alternative medicine tests such as kinesiology, Vega testing, hair testing, and IgG which patients must be strongly warned against [6].

Table 1. Characteristic features of food allergy related disorders

Disorder	Age Group	Clinic	Diagnostic Tools	Prognosis
IgE-mediated Disorders				
Acute Allergic Hypersensitivity	Any age; mostly early childhood period	Onset between minutes to 2 hours; immediate cutaneous (pruritus, erythema, angioedema, urticaria, etc.), respiratory (cough, respiratory distress, wheeze, etc.), cardiovascular (hypotension, tachycardia, shock, etc.), gastrointestinal (nausea, diarrhea, abdominal pain, etc.), and neurologic symptoms (loss of consciousness, seizure, etc.)	History Positive skin prick test (SPT) and or allergen specific immunoglobulin (sIgE), Confirmation of the diagnosis with oral food challenge test (OFC)	Depends on the type of food: milk, soy, egg, and wheat may outgrown in childhood; but peanut, tree nuts, seeds and fish persist lifelong
Oral Allergy Syndrome (Pollen-Food Allergy Syndrome)	Any age; mostly in teenagers or young adults	Immediate symptoms on contact to raw fruit with oral mucosa; pruritus, tingling, angioedema of the lips or tongue	History Positive SPT with raw fruits or vegetables and/or allergen sIgE, confirmation of the diagnosis with OFC	Depends on the type of pollen allergy; symptoms increase with the pollen season, and symptoms may improve with pollen immunotherapy
Ig-E and Non-IgE-mediated Disorders				
Eosinophilic Esophagitis	Any age; particularly in childhood	Intermittant or chronic symptoms of dysphagia, abdominal pain, heartburn, emesis, gastroesophageal reflux symptoms unresponsive to conventional treatment	History Positive SPT and/or allergen sIgE with a poor concordance with the diagnosis (50%), atopy patch test may be of valuable, elimination diet and OFC, endoscopy and biopsy for confirmation of the diagnosis and follow-up the treatment response	Varies, but not well established. Elimination diet improves the symptoms within 6-8 weeks, in case of unresponsive to elimination diet topical or systemic corticosteroids, and/or elemental diet may be required
Allergic Eosinophilic Gastroenteritis	Any age	Intermittent or chronic symptoms of abdominal pain, emesis, irritability, weight loss, failure to thrive, anemia, and protein-losing enteropathy	History Positive SPT or allergen sIgE with a poor concordance with the diagnosis (50%), elimination diet and OFC, endoscopy and biopsy for confirmation of the diagnosis and follow-up the treatment response	Varies, but not well established. Elimination diet improves the symptoms within 6-8 weeks. In case the child is unresponsive to elimination diet elemental diet may be required
Non-IgE-mediated Disorders				
Food Protein-Induced Allergic Proctocolitis (Allergic Proctocolitis)	Young infants (≤6 months), mostly breast-fed healthy appearing infants	Blood-streaked and/or mucuous stool	History Response to elimination diet with suspected food (mostly cow's milk and egg) within 48-72 hours, rarely diagnosis is confirmed by rectal biopsy	Mostly tolerance is achieved by 1-2 years of age
Food Protein-Induced Allergic Enterocolitis Syndrome (FPIES)	Young infants	Acute-subacute: Repetitive or intractable emesis, dehydration, shock in 15% of the cases Chronic: emesis, diarrhea, failure to thrive, anemia	History Response to elimination diet with suspected food (milk, soy, rice, etc.) rarely diagnosis is confirmed by OFC	Mostly resoltion is achieved by 1-3 years of age, rarely persists into late adolescence
Food Protein-Induced Enteropathy	Young infants	Protracted diarrhea (steatorrhea), emesis, anemia in 40%, failure to thrieve	History Endoscopy and biopsy Elimination diet	Mostly resoltion is achieved by 1-2 years of age

Table 2. Most common foods implicated in food allergy

*EU label requirement
1. Milk*
2. Egg*
3. Gluten containing grains (wheat, barley, rye, spelt, etc.)*
4. Fish*
5. Shellfish (mollusks and crustaceans)*
6. Peanut (a legume)*
7. Soya (a legume)*
8. Tree nuts (hazelnut, almond, cashew, pistachio, walnut, pecan, Brazil nut, Macadamia nut)*
9. Mustard (a seed)*
10. Celery*
11. Sesame (a seed)*
12. Lupin (a legume)*
13. Other legumes (peas, beans, chickpeas, lentils, fenugreek, etc.)
14. Other seeds (sunflower seed, linseed, pumpkin seed, etc.)
15. Fruits (peach, kiwi, melon, apple, banana, etc.)
16. Vegetables (potato, cucumber, etc.)
17. Other grains (rice, oat, etc.)

RISK FACTORS

Eczema and family history of atopy are the two major risk factors for the development of FA in childhood [4]. The earlier the onset (i.e. the longer the duration) and the higher the severity of eczema, the higher the risk of FA. Accordingly, the children at the highest risk are infants with severe eczema onset in the first year of life. Males are affected more than females in childhood, whereas in adults FA is more common in females.

FOODS CAUSING FOOD ALLERGY

The most common foods causing FA vary according to country. For example, milk, egg, and peanut allergies are the top 3 in the UK and Australia, whereas milk, egg, and wheat top the charts in Japan and sesame in Saudi Arabia. Besides, differences are observed amongst different ethnic groups living in the same country in terms of the type and the number of trigger foods [4]. In the European Union (EU), it is a legal requirement for some top allergens to be distinctly labeled and declared on all foods for sale (Table 2). Most FAs develop without a history of prior regular consumption, but nut, fish, and shellfish allergies can also develop after prior tolerance. Some food allergies (e.g. fruits, vegetables, and nuts) develop secondary to pollen sensitization, causing localized oral mucosa symptoms only in the majority of patients.

PROGNOSIS

Many FAs with childhood-onset will resolve in time, but the pace and rate of resolution are declining [2]. In general, non-IgE mediated FAs are more likely to resolve faster than IgE-mediated FAs. Also, the prognosis depends on the specific food. For instance, nut and fish/shellfish allergies are much more persistent, whereas egg and milk allergy are more likely to resolve by time.

MORBIDITY and MORTALITY

Fatal anaphylaxis due to FA is very rare [7], though anaphylaxis from all causes including food has increased up to 6-fold in the last two decades in some counties [8]. Anaphylaxis is only possible if one has IgE-mediated FA and there are currently no tests that can estimate the probability or even the threshold for anaphylaxis in a given individual. Patients with a previous history of anaphylaxis and those with poorly controlled asthma are at higher risk of anaphylaxis. Young people in their mid and late-teens have the highest risk of fatal anaphylaxis [8]. Foods responsible for most cases of anaphylaxis will vary according to country, but nuts and milk appear to be the most common ones in the UK [8].

The main morbidities resulting from FAs are (i) Allergic reactions due to accidental exposure, (ii) Avoidance diet which impacts on nutrition and growth unless carefully managed by the dietician and substituted with suitable alternatives to the foods avoided, (iii) Psychosocial impact due to having to follow a food avoidance diet and fear of allergic reactions, (iv) Co-existent atopic diseases/co-morbidities including asthma, eczema, and allergic rhinoconjunctivitis.

PREVENTION and MANAGEMENT

Studies have shown that it is possible to reduce the risk of peanut and egg allergy in children if these foods are introduced into the weaning diet of infants early and eaten regularly [9,10]. The studies aiming to prevent infant eczema (and later FA) by application of moisturizers have not shown benefit so far, but more are ongoing [11]. It is also not possible to prevent milk allergy by feeding high-risk non-breastfed infants hypoallergenic formula [1].

There is no cure for FA, through induction of a degree of tolerance is possible with desensitization (immunotherapy) and such interventions have been the subject of intensive research for the past decade. So far, there is no licensed food immunotherapy product for routine clinical use but several are in the development and approval application stage, particularly for peanut.

The current standard treatment for FA consists of (i) Avoidance with suitable diet substitution, guided by a dietician, (ii) Treating the symptoms of allergic reactions due to accidental exposure (e.g. oral antihistamine +/- adrenaline for IgE-mediated reactions, fluid resuscitation for food protein-induced enterocolitis syndrome (FPIES), treatment of eczema flares, or simply time and patience for gut symptoms caused by non-IgE mediated allergy), (iii) Provision of written allergy management plans and emergency medications is essential for children with IgE-mediated FAs, though not all require Adrenaline autoinjector (AAI) provision [12]. Education of patient and family/school staff in self-management, (iv) Treatment of other atopic co-morbidities, (v) Regular review and re-assessment to determine if the FA is resolving.

It is increasingly recognized that many FA patients and their families need professional psychology support as well as extra social support, as FA is proven to adversely and significantly affect patients' health-related quality of life (HRQL).

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