

Diagnosis and management of food allergy

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Abstract

Food allergy is a common medical problem in infants, children and teenagers. With a good history, appropriate allergy tests and some training, food allergy is relatively easy to diagnose. A correct diagnosis can allow the child to avoid the relevant allergens and reduce their risk of experiencing further allergic reactions. They and their families can also be taught how to recognize and manage any future reactions. This is important as allergenic foods can precipitate anaphylaxis, particularly in children with co-existing asthma. This article will provide a description of how to diagnose and manage infants, children and teenagers with possible food allergy.

Keywords adrenaline autoinjector; anaphylaxis; food allergy; food challenge; serum specific IgE; skin prick testing

Introduction

Food allergy is a term used to describe an immunological adverse reaction to food. It is an increasingly common problem affecting children and adults at a global level. The prevalence of food allergy in Europe has been reported to be between 1 and 2% in adults and 5% in children diagnosed using a double-blind, placebo-controlled food challenge, which is the gold standard test. However, 35% self-reported food allergy. A correct diagnosis of food allergy, followed by counselling and advice based on valid test results, is important. It will help to reduce further adverse reactions and prevent unnecessary dietary exclusion of foods that are safe and should be eaten as part of a normal,

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healthy diet. There is often confusion between allergy and sensitization to a food (see [Box 1](#)).

The differential diagnoses of food allergy need to be considered. Food hypersensitivity describes any adverse reactions to food, encompassing both immune and non-immune mechanisms. Non-immune mechanisms include metabolic (e.g. lactose intolerance, alcohol dehydrogenase deficiency), toxic reactions (e.g. tyramine in aged cheese, histamine release from spoiled dark-meat fish also known as scomboid), psychological (e.g. food aversion, anorexia nervosa) and neurological (e.g. facial flush from tart foods, gustatory rhinitis from spicy foods) (see [Figure 1](#)). Immune mechanisms include IgE and non-IgE mediated reactions, food allergy. There is better understanding of the classical IgE mediated food allergy, which will be discussed in detail within this manuscript. Non-IgE mediated food allergy includes gastrointestinal inflammatory dysmotility disorders such as eosinophilic gastroenteropathies, dietary protein enterocolitis, and recently food additive allergy has been described. Non-IgE mediated food allergy is typically delayed and manifests as exacerbations of eczema, urticaria or gastrointestinal symptoms with persistent wheeze (see [Figure 1](#)).

Risk factors and pathogenesis of food allergy

The pathogenesis of IgE-mediated food allergy is multifactorial and depends on an interplay between genetic predisposition and environmental exposure. A family history of allergic disease is a non-modifiable risk factor for development of allergy in the offspring. The risk is particularly significant with a history of maternal allergic disease. Environmental exposure is a modifiable risk factor, and much information has been gained from well-designed, long-term birth cohorts. The hygiene hypothesis suggests that higher order siblings are less likely to develop allergic diseases due to Th1 induction from exposure to infections from their siblings. Caesarean section delivery has been associated with allergy development and this may be due to lack of exposure to maternal gut microbiota. Exposure to environmental tobacco smoke in utero and postnatally can alter early immune development, causing impairment of Th1 function and predisposing to allergic sensitization. Early and persistent sensitization to food allergens is associated with physician-diagnosed food allergy later in childhood. Development of eczema in early infancy increases the likelihood of food allergy later in childhood. Dietary supplementation may also influence the development of allergic disease, an imbalance between omega-3 and omega-6 polyunsaturated

Definitions

Allergy is a term used to describe the development of reproducible symptoms or signs initiated by exposure to allergens at a dose tolerated by normal persons by a specific immune mechanism, which can be IgE mediated or non-IgE mediated.

Sensitization is the detection of specific IgE (sIgE) to an allergen on skin prick test or in serum. Sensitization is not synonymous with clinical allergy; a child who has sIgE to milk but who is regularly ingesting milk is described as sensitized (or atopic), but not allergic.

Box 1

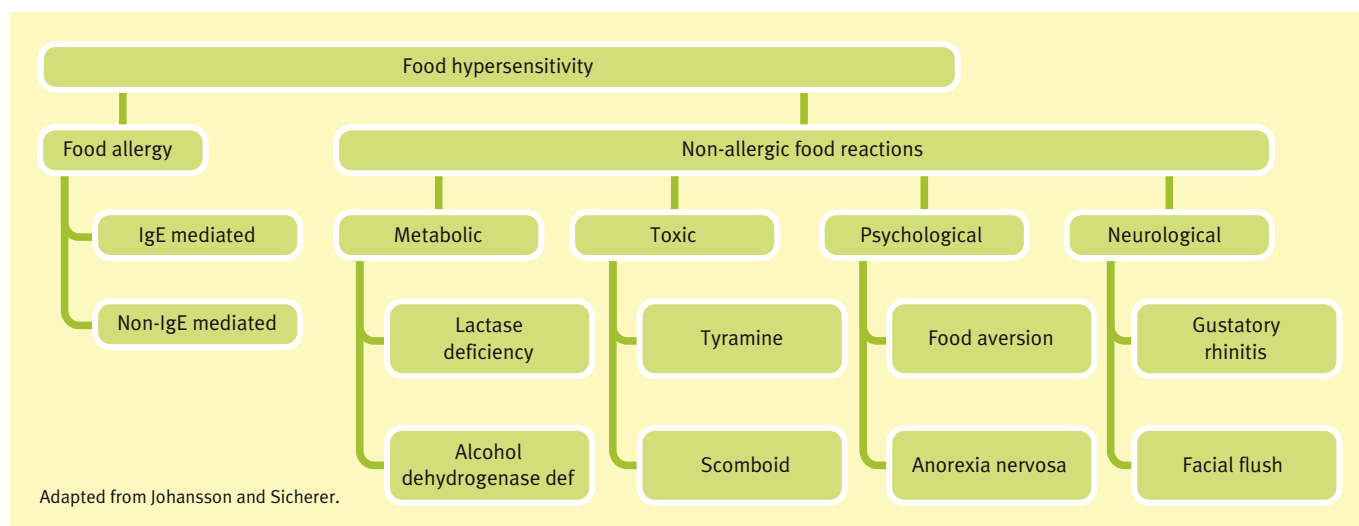


Figure 1 Classification of adverse reactions to food.

fatty acids, as well as excess or deficiency in vitamin D have been associated with allergy.

The timing of food allergen exposure may play a role in development of allergy. Breastfeeding guidelines and allergen avoidance advice has changed in the last few years to reflect an increased understanding in this field. Pregnant women are no longer advised to avoid common food allergens such as peanuts during pregnancy, and exclusive breastfeeding beyond 4–6 months is not recommended particularly in atopic families. For the infant, there is now evidence to suggest that solid foods should be introduced early, including traditionally allergenic foods such as peanut. A recent cohort among Jewish schoolchildren in Israel and in North London found a discrepancy in peanut allergy, 0.17% compared to 1.85%. This was a statistically significant difference, which may be explained by the common practice of feeding children peanut-flavoured snacks before the age of 1 year in Israel. This has led to the hypothesis that the development of food allergy is due to a failure of oral tolerance induction associated with seeing food first via the gastrointestinal tract; where the first exposure to food allergens is via the skin, particularly if inflamed with eczema, sensitization is likely.

Sensitization occurs after the initial exposure of the immune system to the allergen, with formation of specific immunoglobulin E (sIgE) antibodies under the control of T helper 2 (Th2) cytokines. With further exposure, some children who are sensitized to a food develop a chain of reactions leading to release of inflammatory mediators and a clinical allergic reaction. Typical symptoms of IgE-mediated food allergy are shown in Table 1 below.

The pathogenesis of non-IgE mediated food allergy is much less well understood, is poorly defined both clinically and scientifically, and is believed to be T-cell-mediated. Symptoms are summarized in Table 2 below.

Common food allergens

Food allergy is a dynamic process, and different proteins can cause allergy at different time points in a person's lifetime. During childhood, the most prevalent IgE-mediated food allergens are cow's milk, egg and peanut. The natural history is for children to outgrow cow's milk and egg allergy although a small proportion

will persist. Only around 20% of children are thought to outgrow peanut allergy. During adolescence and adulthood, fish and shellfish allergy plus reactions to fruit and vegetable predominate. Non-IgE-mediated food allergy can be triggered by a similar range of food but cow's milk, wheat and soya predominate.

Diagnosing food allergy

History

A detailed history is fundamental to the correct diagnosis of food allergy (Box 2). A comprehensive history can help predict the likelihood of food allergy. Patients with IgE-mediated food allergy typically have reproducible type I hypersensitivity reactions within 30 min, or at most 2 h after exposure. Routes of exposure include contact, ingestion, and inhalation of aerosolized food proteins (cooking fumes). There may be co-existing allergic diseases such as eczema or asthma.

More investigative work is required when assessing a child suspected of having non-IgE mediated food allergy. Symptoms may arise in the skin, respiratory tract or gastrointestinal tract (Table 3). Onset is typically delayed, and can be up to 24 h after ingestion. It should be considered in children whose symptoms do not respond adequately to treatment for chronic atopic eczema, chronic gastrointestinal symptoms, gastro-oesophageal reflux, chronic constipation and diarrhoea.

Physical examination

It is important to look for any cutaneous rash, abnormalities of nose or ear, or abdominal discomfort. Signs may be absent if a patient is being reviewed in clinic; however parents may have taken photographs during the acute reaction. When conducting an assessment of a child in the acute setting, it is important to recognize anaphylaxis — signs of upper or lower airway obstructions or circulatory collapse in association with cutaneous signs of an allergic reaction.

Investigations

The most widely used diagnostic tests for food allergy are skin prick test (SPT) and serum specific IgE (sIgE). These tests are extremely good at detecting the presence of specific IgE

Symptoms and signs of IgE-mediated allergy

Key features	Mechanism	Age affected	Likely allergens
Urticaria/angioedema Triggered by ingestion or direct skin contact	Local release of mediators such as histamine	Children > adults	Major allergens, e.g. peanut, tree nut, fish, shellfish, egg, milk
Oral allergy syndrome (pollen-food related) Pruritus, mild oedema confined to mouth	IgE to pollens that also binds certain homologous food proteins e.g. apple Mal d 1 and birch Bet v 1	Onset after pollen allergy established; adult > young child	Raw fruit or vegetables; cooked forms are tolerated
Rhinitis, asthma May accompany food-induced allergic reaction or with inhalation of aerosolized food protein (e.g. cooking fumes)	Mechanism unclear	Infant or child > adult	Major allergens, e.g. peanut, tree nut, fish, shellfish, egg, milk
Anaphylaxis Rapidly progressive, multiple organ system reaction, can include cardiovascular collapse	Massive release of mediators such as histamine	Any age	Any allergens but commonly peanut, tree nuts, shellfish, fish, milk, egg
Food-associated, exercise induced anaphylaxis Food triggers anaphylaxis only if ingestion followed temporally by exercise	Exercise is presumed to alter gut absorption, allergen digestion or both	Onset more commonly later childhood or adulthood	Wheat, shellfish, celery

Adapted from Sicherer.

Table 1

Symptoms and signs of mixed IgE/non-IgE-mediated allergy

Key features	Mechanism	Age affected	Likely allergens
Atopic dermatitis Associated with food in 35% children if moderate-to-severe	Might relate to skin-homing of food-responsive T cells to the skin	Infant > child > adult	Major allergens particularly egg, soya and milk
Eosinophilic gastroenteropathies Symptoms depend on site and degree of eosinophilic inflammation. Oesophageal: dysphagia & pain Generalized: ascites, oedema, weight loss, obstruction	Mediators which are gut-homing and activate eosinophils which play a role with eotaxin and IL-5	Any	Multiple
Food protein induced enterocolitis syndrome (FPIES) Chronic exposure: vomiting, diarrhoea, poor growth, lethargy Re-exposure after restriction may give hypotension	Increased TNF- α response, decreased response to TGF- β	Infancy	
Food protein proctocolitis (prev cow's milk protein intolerance) Mucus laden, bloody stool in well infant	Eosinophilic inflammation	Infancy	Cow's milk (via breast milk)
Non-IgE-mediated dysmotility disorder Eczema, GORD, abdominal pain, diarrhoea, flatulence, constipation, tenesmus, failure to thrive, blood in stools	May relate to gut plus/minus skin homing T-cell activity. May be associated with eosinophilic inflammation	<1 year: upper GI and lower GI dysmotility (GORD and diarrhoea) 2–5 years: mid- and hind gut dysmotility (constipation) >5 years: Upper to lower GI dysmotility	Cow's milk, soy, egg, fish, wheat

Adapted from Sicherer.

Table 2

Salient points in history taking

- The age of the child or young person when symptoms first started
- Who has raised the concern and suspects food allergy
- What the suspected allergen is
- The speed of onset of symptoms following exposure
- Route of exposure (contact, ingestion, inhalation)
- The duration of symptoms
- The severity of reaction
- The frequency and reproducibility of reaction
- The setting of the reaction (for example, at school or home)
- The smallest quantity of food to produce reaction
- Whether or not exercise was associated
- Any cultural and religious factors that affect the foods they eat
- Feeding history: breast or formula-fed, age at which they were weaned onto solid food, if child is currently breastfed, consider mother's diet
- The details of any previous treatment for the presenting symptoms and the response to this
- Any response to the elimination and re-introduction of foods
- Any history of malnutrition or growth failure

Box 2

antibodies, i.e. sensitization. They can support positive findings in a detailed history (Box 2), and may be sufficient to reach a definitive diagnosis. When results are equivocal, an oral food challenge is indicated to obtain a diagnosis. The gold standard for diagnosis is a double-blind, placebo-controlled oral food challenge as it removes observer and patient biases.

Patients with non-IgE food allergy may benefit from sIgE testing in order to exclude IgE-mediated food allergy to guide patient management. This may also diagnose co-existing food induced allergic reactions. Investigations used may include SPT, sIgE and atopy patch tests. Excluding IgE food allergy can provide assurance that severe reactions (e.g. anaphylaxis) are unlikely to occur. Often non-IgE food allergy can only be definitively diagnosed by removing and re-introducing the suspected allergen and observing the consequences. Mixed IgE and non-IgE disease is the most challenging to manage and treat as results may overlap.

Skin prick tests: skin prick tests (SPT) identify specific IgE attached to mast cells in the skin, i.e. sensitization. It can be used to help predict the likelihood of clinical allergy. The test involves applying droplets of allergen onto the forearm, and scratching the surface of the skin using a single headed lancet (Figure 2). Negative (saline) and positive (histamine) controls must be used. Results are read within 15 min. Development of a wheal represents a positive reaction, and the larger the reaction the more likely they are to indicate food allergy. In clinical practice

Differentiating symptoms of IgE and non-IgE food allergy

IgE-mediated food allergy

Skin

Pruritus
Erythema
Acute urticaria — localized or generalized
Acute angioedema — most commonly of the lips, face and around the eyes

Respiratory tract

Upper respiratory tract symptoms: nasal itching, sneezing, rhinorrhoea or congestion, hoarse voice, stridor
Lower respiratory tract symptoms: cough, chest tightness, wheezing or shortness of breath

Gastrointestinal tract

Angioedema of the lips, tongue and palate
Oral pruritus
Nausea
Colicky abdominal pain
Vomiting
Diarrhoea
Faltering growth in conjunction with at least one or more gastrointestinal symptoms above (with or without significant atopic eczema)

Non-IgE-mediated food allergy

Pruritus
Erythema
Atopic eczema

Lower respiratory tract symptoms: Nocturnal cough and wheezing associated with postnasal drip, gastro-oesophageal reflux disease and chronic upper airway aspiration of foods due to dysmotility disease

Gastro-oesophageal reflux disease
Loose or frequent stools
Blood and/or mucus in stools
Abdominal pain and flatulence
Nocturnal sweating and enuresis
Infantile colic
Food refusal or aversion
Constipation
Perianal redness/excoriation
Pallor and tiredness
Faltering growth in conjunction with at least one or more gastrointestinal symptoms above (with or without significant atopic eczema)

Table 3



Figure 2 Skin prick test on an infant.

a positive result is a wheal diameter measuring at least 3 mm in diameter. At this level, a negative result is useful to exclude diagnosis but a positive result is not conclusive. Wheal sizes at which there is a 95% probability of clinical allergy have been established for the common food allergens (see [Box 3](#)). These are helpful but do need to be interpreted in the light of the history. Clinical allergy is still likely in the presence of a very good history of an IgE-mediated food allergy and a small SPT wheal.

SPTs are ideal to perform in an outpatient clinic. The test is easy particularly with good, trained staff. It is cheap, safe, cost-effective, and results are available during the outpatient appointment. There are many commercially available SPT reagents for a wide range of allergens. There are limitations to this useful test: false negative results can occur with antihistamine use, not recommended on severe eczematous skin, and results may vary with different nurses. Although SPT reagents are available, the list is not exhaustive and prick-to-prick test using fresh foods may be needed.

The 95% positive predictive values for skin prick tests and serum specific IgEs

	SPT wheal (mm)	Specific IgE levels (IU/ml)
Egg	7	≥ 7
Egg (≤ 2 years)	5	≥ 2
Milk	8	≥ 15
Milk (≤ 2 years)	6	≥ 5
Peanuts	8	≥ 15
Peanuts (≤ 2 years)	4	–
Tree nuts	8	≥ 15
Fish	–	≥ 20

Box 3

Serum specific IgE (sIgE): measurement of serum sIgE used to be referred to as the RAST test. This term is now outdated, as newer assays currently in use provide a more sensitive assay (detecting sIgE down to 0.1 kU/litre) without using radioisotope labels. The most widely used test is the ImmunoCAP fluorescence enzyme immunoassay (FEIA) system. Serum is added to a solid matrix (allergen), which binds antibody; following a series of incubation and washes, bound IgE antibody is detected using enzyme-labelled anti-IgE reagent. This assay is calibrated against World Health Organization standard for IgE.

The traditional positive cut off value of 0.35 kU/litre is excellent for excluding a food allergy but not useful in diagnosing clinical food allergy. Around 30–40% of people with positive sIgE were subsequently found to pass oral food challenge as they had developed tolerance. Levels of serum sIgE with a 95% positive predictive value (for clinical food allergy) have been correlated with DBPCFC and listed (see [Box 3](#)). As with SPT, the result needs to be interpreted in light of the presenting history.

Serum sIgE is useful where there is lack of trained staff to perform SPT as there is negligible inter-operator variability, and may be used for patients on antihistamine therapy. Results will not be affected when performed on patients with eczema flare up. Limitations of this test are that results can take up to a few weeks and patients may not be keen to undergo venepuncture. Similar to SPTs, increased levels of sIgE increases the likelihood



Figure 3 Eczema affecting the face, flexor and extensor surfaces.

Illustration of diagnostic process in IgE-mediated food allergy

History, examination and investigations: An 8-year-old girl presents with generalized urticaria and oral symptoms (itching, swelling of lips and tongue) following a bite of peanut butter sandwich. The reaction occurred within 5 min of exposure and resolved with two doses of cetirizine (antihistamine). This is the first known episode of peanut ingestion. As a toddler, Mum recalls that she developed generalized urticaria having been kissed by granddad who had just eaten peanuts. She has asthma which is generally well controlled on minimal inhaled corticosteroids. Claire has asthma which is generally well controlled on minimal inhaled corticosteroids. She also has mild hayfever symptoms in early spring, which Mum says is controlled with daily cetirizine (antihistamine). Physical examination revealed mild eczema. SPT showed a 5 mm wheal to peanut and sIgE was 7 kU/litre.

Interpretation: This 8-year-old girl has a good history for IgE-mediated allergy and she has a personal history of allergic disease (asthma and eczema), which makes peanut allergy likely. Her sIgE tests are not entirely conclusive, they fall below the 95% positive predictive value mentioned earlier. The next obvious step would be to perform an oral challenge in hospital in order to firmly diagnose peanut allergy. However, she could be offered component testing. This is a more accurate method of differentiating between primary peanut allergy and oral allergy syndrome, particular as her SPT to birch pollen is 10 mm and sIgE is 19 kU/litre. Components testing reveal negligible results on Ara h 1, 2, 3 (primary peanut allergens) with a positive Ara h 8 (9 kU/litre). Ara h 8 is a homologous protein to birch pollen confirming a diagnosis of oral allergy syndrome to peanut with a primary birch pollen allergy. Oral challenge may not be necessary. She should still avoid peanuts but is not at risk of anaphylaxis.

Box 4(a)

of clinical food allergy; however negative results do not completely exclude allergy. In addition, the level of sIgE does not reflect the severity of reaction.

Components resolved diagnostics: oral allergy syndrome describes reproducible symptoms following exposure to raw fruit (e.g. apples, peaches, pear) and nuts (e.g. hazelnut). Specific IgE to pollen, associated with hayfever, can also reaction with homologous fruit or nut proteins. This gives rise to local symptoms of oral allergy syndrome with tingling, itch, swelling and numbness. Component resolved diagnosis can be used to distinguished between, for example, a primary hazelnut allergy and oral allergy syndrome due to cross reactivity to birch pollen. Oral food challenge may be avoided in some patients if components testing leads to a definitive diagnosis (Box 4).

Food challenge: a food challenge is indicated when physicians need to reach a definitive diagnosis of food allergy having performed sIgE tests. Indications are set out in Box 5. An oral food challenge can differentiate between sensitization and clinical food allergy.

Illustration of diagnostic process in non-IgE-mediated food allergy

History, examination and investigations: Baby L presents at 4 months of age with an 8 week history of persistent wheeze, severe eczema, gastro-oesophageal reflux, diarrhoea and failure to thrive. He was commenced on Aptamil formula milk from birth. Severe pruritus and excoriation of the skin, irritability, insomnia and poor eating further developed. From 10 weeks of age he suffered from non-projectile vomiting, bloody loose stools, poor weight gain and milk refusal. On examination he has severe lichenified, excoriated eczema over the face, nape of the neck, flexure and extensor surfaces of the body. Infected areas included the wrists and ankles (Figure 3). He is reviewed by a paediatric dietician and commenced on an extensively hydrolyzed milk formula (nutramigen). Due to poor response and further loss of weight he is commenced on an amino acid formula (neocate) with good effect. He has negative skin prick tests and specific IgE to milk and soya but positive results for egg (6 mm) and peanut (8 mm) confirming allergic sensitization. Due to these results he undergoes a double-blind, placebo-controlled challenge to cow's milk protein. Symptoms develop 12 h after the food challenge with severe excoriation of eczema with skin bleeding, pruritus, insomnia, night sweats, vomiting, diarrhoea, flatulence, severe abdominal pain and profuse diarrhoea. Cow's milk, egg, soya and nuts are excluded from his diet to good effect.

Interpretation: His main disorder is a non-IgE-mediated allergy cow's milk involving the skin, respiratory and gastrointestinal systems. He may also have a non-IgE-mediated soya allergy and IgE-mediated egg and peanut allergy. Many of his cutaneous, GI and respiratory symptoms are likely to disappear with exclusion of cow's milk, soya, egg and peanuts but he made some specific therapy for eczema and gastro-oesophageal reflux.

Box 4(b)

Oral food challenges can be conducted open, single blind (to patient) or double-blind (to observer and patient). The latter is the gold standard as it removes observer and patient bias and is of utmost use when assessing a patient with a potential psychological mechanism of food reaction, e.g. food aversion. In open challenges, food is given unmasked and unblinded in its natural form where objective symptoms such as urticaria and angioedema are anticipated and concern for bias is low. In blinded challenges food is masked using a vehicle or placed in an opaque capsule to reduce bias. Challenges should be conducted to a high standard, following available international guidelines. Challenges where an acute reaction is possible, should be conducted in hospital by staff trained to recognize and manage severe allergic reactions. Food (including blinding) should be prepared by a dietician. Positive endpoints should be standardized, tailored to the patient with clearly recorded timing of symptoms. Not all patients are suitable for a food challenge; for example those on medications which can enhance, mask, delay or prevent evaluation of a reaction or interfere with treatment of a reaction should not be challenged (e.g. antihistamine, neuroleptics, oral steroids (more than 5 mg a day), aspirin/NSAIDs, ACE-inhibitors, beta-blockers).

Indications for oral food challenge

- Initial diagnosis of food allergy
- Monitoring resolution of food allergy (assessing tolerance)
- Determine threshold value or degree of sensitivity
- Determine whether food allergens associated with atopic dermatitis will cause immediate reactions
- Expand the diet in patients with multiple dietary restrictions due to non-immune mechanism of food reaction (headaches, behaviour change)
- Assess the affect of food processing on food tolerability, e.g. patients with oral allergy syndrome who can tolerate cooked forms of fruit and vegetable

Box 5

Unvalidated allergy tests: there are many alternative practitioners who offer unvalidated allergy tests to the general public. These attract many people as the tests are accessible and in some cases results are obtainable on the same day. Among available tests are kinesiology, VEGA or electrodermal tests, hair analysis and measurement of specific IgG levels (Box 6). None of these methods are reproducible, nor can they correctly diagnose IgE or Non IgE mediated food allergy. The recent NICE guidelines advise against the use of the above methods in diagnosing food allergy.

Trial elimination diet: whilst skin prick tests and serum specific IgE tests offer some guidance to clinicians and dieticians for future management, this is not the case for non-IgE disease. The results are often negative. Patch tests are also unreliable and require further evidence based medicine data to determine their efficacy and value. The NICE guidelines recently concluded that the preferred clinical pathway for children and young people with suspected non-IgE-mediated food allergy would be a full allergy focused clinical history followed by a food elimination diet. Food elimination represents both a diagnostic tool for food allergy but also a treatment. A trial elimination of the suspected allergen should be for 2–6 weeks. If the food allergy is confirmed by their symptoms improving the diet is continued as treatment. The answer may not be obvious and a series of periods of exclusions and then re-introductions may be required to confirm the diagnosis. Paediatricians must seek advice from an allergy dietician about the elimination of the suspected allergen. Children with non-IgE-mediated disease may have multiple foods excluded from their diet. Their intake is often that of low calorie and as such may fail to thrive with regards to weight but preserve their height velocity (see Figure 4).

Managing food allergy

Multidisciplinary team approach

Once a diagnosis of food allergy is made, management has to be tailored to the patient's and their families' needs. The best way to approach this is by providing multidisciplinary care, which involves an allergy clinical nurse specialist, allergy dietician and an allergy specialist consultant. A close relationship with paediatric dermatologists, gastroenterologists and respiratory

Unvalidated food allergy tests

Kinesiology: patient holds a vial of food allergen in a stoppered glass bottle in one hand; muscle weakness is tested in the contralateral arm. Presence of weakness in contralateral arm means the test is positive.

Vega (electrodermal test): observation of changes in electrical impedance in the skin at different acupuncture points in response to substances placed within an electrical circuit. Used to test for food & inhaled allergens.

Hair analysis: a forensic screen for metal intoxication. It has been modified to screen for food allergy. Food allergen avoidance advice given according to results of analysis.

Specific IgG levels: this has been used in research as a tool to reflect development of tolerance; however its usefulness does not translate into the clinical setting. IgG levels do not reflect outcome of oral food challenge.

Box 6

physicians may also be required. Educating patients to recognize and manage possible allergic reactions enables them to feel empowered and encourages compliance with treatment.

Food allergen avoidance

The best way to prevent accidental exposure and allergic reaction, IgE- or non-IgE-mediated, is to completely avoid the allergen. Patients and their families will need to learn to read food labels and ingredients extremely carefully. They will need to learn about "hidden" ingredients such as use of nuts in many Indian curries and use of milk protein in many processed foods. Cross-reactivity of allergenic food proteins occur and patients should be made aware of this. For example, there is a risk of reacting to soy in patients with milk allergy; there is also a risk of reaction to many tree nuts in peanut allergic patients. A risk assessment should be conducted with individual patients and family, and a joint decision made as to whether peanut allergic children should avoid all nuts as a precaution. Different patients have different thresholds of reactivity to allergens which need to be taken into account when determining the degree of dietary restriction that they require. Some patients may react to a trace of a food allergen while others need to consume much more before they have a reaction. This is particularly relevant with peanuts and tree nuts as many products are labelled "may contain traces of nuts" although the chance of them containing nut protein is often extremely small. Avoiding all these can be very restrictive and it may be appropriate for a patient to consume such products in a safe environment such as the home but not, for example, on holiday abroad where access to medical care is unfamiliar.

Allergen avoidance is not straightforward. Planning ahead is required when eating out in restaurants. The child's safety is paramount; families should consider ringing the restaurant in advance to ensure they are able to cater for a child with food allergy. Ideally they should also speak to the chef directly to ensure not only will the food be free of the allergenic ingredient, but also utensils used should not be contaminated. There is also high risk of food contamination at takeaways, friend's homes and parties; parents need to be aware and take necessary precautions. The whole

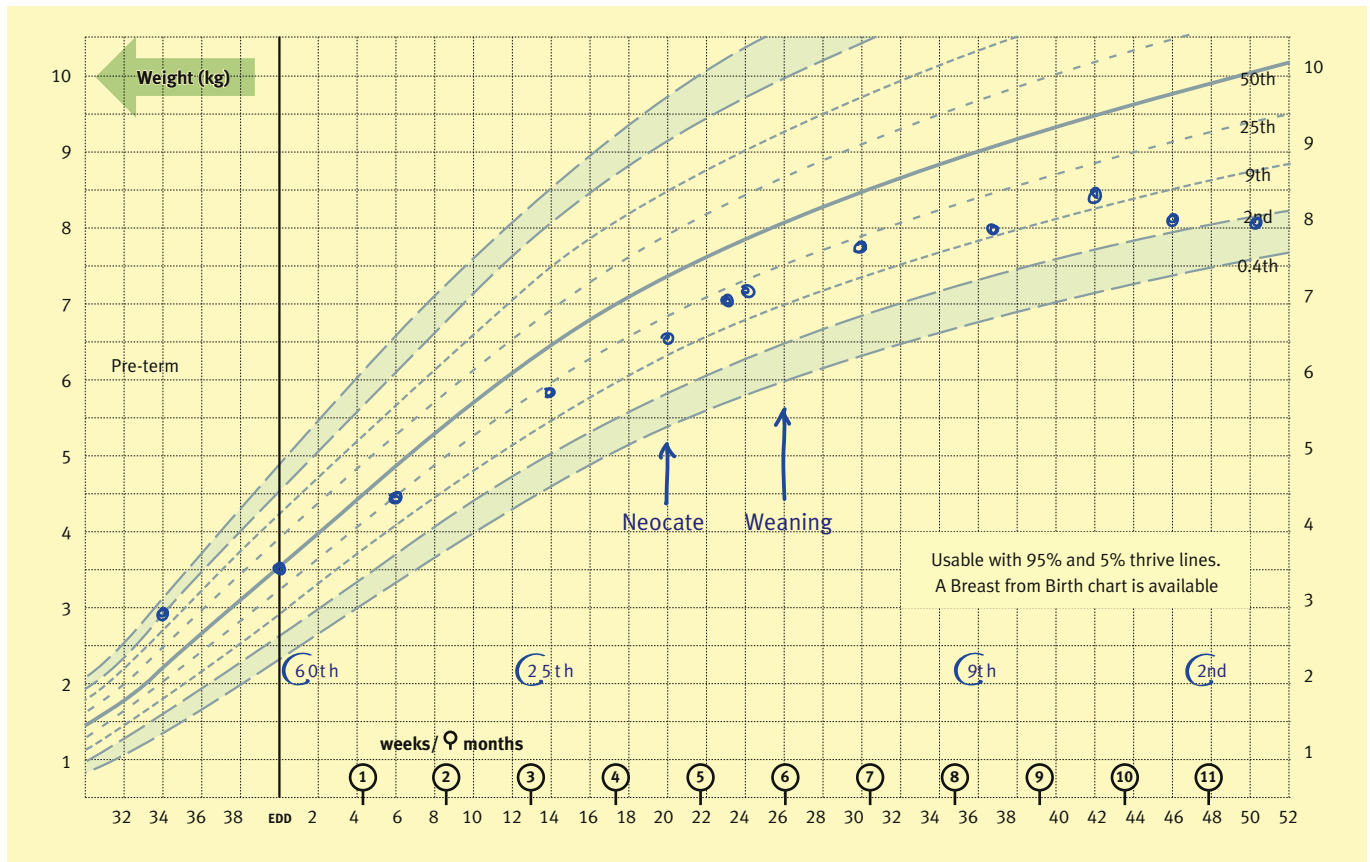


Figure 4 Growth chart demonstrating failure to thrive.

family's approach to food will have to change with significant impact on the patient and their family's quality of life. If certain foods have to be avoided, patients will need their nutrition intake reviewed closely, which is why dietitians have to be involved. The clinical nurse specialist also plays a vital role in educating and supporting the family with food allergy. Teenagers should be a particular focus for education. They are a special high-risk group as they are known to have unplanned lifestyles which may often involve risk taking with allergen exposure, easily influenced by peers and a failure to carry their rescue medication. Compliance with allergen avoidance needs to be reviewed regularly.

Rescue medication for IgE-mediated food allergy

In the event of accidental allergen exposure, patients need to correctly identify symptoms of an allergic reaction in order to institute appropriate management. Verbal education in clinic can be supplemented with a written action plan to be kept with the patient and at school. This will serve as a "crib sheet" or guideline during an acute reaction when nerves are fraught.

For minor allergic reactions, such as localized angioedema, urticaria, oral symptoms, patients should be given a second generation antihistamine (e.g. cetirizine) immediately. This may not be the end of the reaction and patients need to be aware that symptoms may recur or progress. If anaphylaxis develops, with wheeze, cough, collapse, feeling of impending doom, throat tightening with difficulty breathing, the most effective treatment is intramuscular adrenaline given using an autoinjector. It is not practical for a child or their family or carer to correctly and

rapidly draw up a small dose of adrenaline from an ampoule. The first dose of adrenaline should be administered and an ambulance called immediately. If symptoms do not improve within 5–10 min, a second dose of adrenaline should be administered. When available, bronchodilators, oxygen and corticosteroid are secondary treatments. Patients with asthma who has had a previous anaphylactic reaction should be given prednisolone in order to avoid late phase reaction.

Patients should be provided with rescue medications before leaving clinic. This should include a second generation antihistamine (e.g. cetirizine) and, where appropriate, an adrenaline auto-injector which should be weight appropriate. Indications for prescription of adrenaline autoinjector are previous anaphylaxis, co-existing asthma on preventer therapy patients, allergens such as peanut where there is a high risk of anaphylaxis and previous reactions of a trace of an allergen. Patients with asthma are at higher risk of anaphylaxis and it is imperative to ensure their asthma is well controlled in order to minimize this risk, and that they have an adrenaline autoinjector to hand.

The patient and family will require training to recognize anaphylaxis and to administer the adrenaline autoinjector whilst in clinic. A written action plan will consolidate the information received in clinic. There has been debate over the number of auto-injectors required per patient. The devices are not cheap and expire within a year or so. The decision should be individualized with patients at high risk of a future severe reaction (e.g. a previous severe reaction or severe asthma) having access to two at all times. Every person who looks after the patient should

be trained (via community nurses) on how to recognize an allergic reaction and how to manage it safely, including the use of the adrenaline autoinjector.

Follow up

Allergen avoidance or dietary elimination is the appropriate initial management for all patients. It is important to remember that many patients outgrow their food allergy, particularly with cow's milk, egg, soy and wheat. For IgE-mediated food allergy, the timing of re-introduction can be guided by repeat SPT and serum specific IgE tests. Dropping levels reflect the development of tolerance. In general if the level has halved and the patient has not had any recent reactions, an inpatient food challenge is indicated to hopefully diagnose resolution. For egg allergy, this process can be aided by following the change in ovomucoid levels. For non-IgE-mediated food allergy, tests are not helpful. Infants usually outgrow cow's milk, soy or wheat allergy in the first few years of life and, where IgE-mediated allergy has been ruled out, caution, graded re-introduction at home can be attempted.

Vaccination and children with egg allergy

When the MMR vaccine contains traces of egg, levels are too low to represent a risk of allergy allergic individuals. All egg allergic infants can be immunized safely in the community. Children with egg allergy should receive an influenza vaccine with a very low concentration of egg ovalbumin (less than 0.12 mcg/mL). Higher risk group (e.g. previous anaphylaxis or co-existing moderate to severe asthma) should receive the influenza vaccine in hospital.

Summary

Food allergy is a common problem in infants, children and teenagers. Although any food can be an allergen, peanut, tree nuts, cow's milk, egg, fish, soy and wheat are the commonest. Allergy can present as immediate IgE-mediated symptoms or delayed type non-IgE ones. Diagnosis is based on a characteristic history with the support of allergy tests such as SPT and serum specific IgE. Sometimes an oral food challenge is required to give a firm diagnosis. To avoid repetitive reactions, children must avoid the offending allergen. They and their carers also need to be able to recognize future reactions and know how to manage them. Many children outgrow their food allergy and so need to be reassessed at regular intervals. While we do not currently have an effective preventative strategy, several studies are in progress to investigate potential ways of preventing the development of food allergy. Other studies are also investigating potential curative approaches. ♦

FURTHER READING

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Practice points

- The presenting history frequently provides the diagnosis with characteristic differences between IgE- and non-IgE-mediated food allergy
- Allergy tests, such as SPT or specific IgE, can confirm or exclude a diagnosis of IgE-mediated food allergy but must be interpreted within the context of the presenting history
- Food challenges may be required to confirm a diagnosis or to later confirm resolution of a food allergy
- Children with food allergy should be managed by a multidisciplinary team of a paediatric allergist, paediatric dietician and clinical nurse specialists
- Avoidance of the triggering food allergen is essential and should be guided by a paediatric dietician
- The child and carers should be able to recognize future reactions and know how to manage them, for high-risk cases this should include an adrenaline autoinjector