

Educational Series

Anaphylaxis to foods

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Three crucial areas of the management of anaphylaxis due to food allergy are discussed: making a diagnosis, deciding who needs a self-injectable adrenaline device and spotting the novel allergen. Managing children and teenagers with anaphylaxis is challenging due to the lack of available evidence that specifically addresses these issues. The available evidence is presented and discussed.

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Anaphylaxis is a not uncommon paediatric emergency. There is no current consensus about the specific criteria for diagnosis of anaphylaxis. A widely accepted definition would be a 'severe, life-threatening generalized or systemic hypersensitivity reaction' (1). In allergic anaphylaxis, there is an IgE mediated systemic release of mediators from mast cell and basophils. This leads to the development of the cutaneous, respiratory and/or cardiovascular symptoms and/or signs that are seen in anaphylaxis (see Box 1). The incidence of anaphylaxis is unclear but probably ranges from 1 to 20 per 100,000 person years (2–4). Routine United Kingdom hospital admission data suggests that there has been a 250% increase in severe anaphylaxis between 1995 and 1999 (5). Foods are the commonest cause of anaphylaxis in children (6–8) with peanuts, tree nuts, milk and fish being the commonest precipitants (Box 2).

Case 1: is this child allergic to peanuts?

A 6-yr-old boy complains of an itchy mouth whilst at his grandfather's 60th birthday party. Fifteen minutes later, he starts to develop an itchy rash on his face. His parents describe it as being like a nettle rash. A short while later, his lips became noticeably swollen and he indicated to his parents that he was having difficulties in

breathing. His parents gave him some chlorpheniramine syrup and called an ambulance. He was treated in the emergency department with oxygen, i.m. adrenaline, nebulized salbutamol and oral prednisolone. He recovered after a couple of hours without developing any other symptoms. The dietary history reveals that he consumes all the common food sources without problems but has always avoided peanuts and tree nuts as his cousin is peanut allergic. Perhaps surprisingly, there were peanut butter sandwiches at the party. His parents had presented him to the allergy clinic wanting to know if whether or not he is also peanut allergic. His other medical history is unremarkable apart from infantile eczema. The examination is unremarkable.

Skin prick testing with peanut and a selection of tree nuts revealed only a positive reaction to peanuts, with a 5-mm weal. The saline control was negative and the histamine control gave a 4-mm weal. Serum specific IgE levels were only raised to peanut (8 KU/l).

Identifying the cause of anaphylaxis

The accurate identification of the precipitant of an anaphylactic reaction is crucial if patients are to avoid the trigger in the future. It is particularly important where the trigger is a food as an over-restricted diet may compromise nutrition, growth

Box 1. Commons symptoms and signs associated with anaphylaxis

Cutaneous
Urticaria
Angioedema
Pruritis (including oral)
Respiratory
Rhinoconjunctivitis
Throat tightness*
Change in voice*
Stridor*
Wheeze*
Difficulty in breathing/signs of respiratory distress*
Cardiovascular
Collapse*
Hypotension*

*Symptoms or signs particularly associated with life-threatening allergic reaction.

Box 2. The common food allergens

Peanut
Tree nuts
Cows' milk
Hens' egg
Soya
Wheat
Fish
Shell fish
Sesame

and health. The history may be diagnostic but often there is still a need for further tests. For example, a history of a single adverse reaction to a specific food has only a 50% positive predictive value for correctly identifying the allergen (9). Allergy testing is indicated to confirm the diagnosis as the child may have out grown the problem or have reacted to another unidentified allergen. If a patient has a history of three similar adverse reactions to a particular food, the positive value that food is the allergen is around 100% (10).

When the history is not diagnostic, testing with either skin prick testing or serum specific IgE levels is required. The former are preferred as it is less expensive and provides immediate results. There are advantages of serum specific IgE, for example, there are a greater range of available allergens, they can be performed where skin prick testing is not available or when a child had taken an antihistamine. It has now been recognized that the greater the weal response on skin prick testing or the higher the serum specific IgE level, the more likely a positive result will indicate clinical allergy. For example, in a group of over 100 children and teenagers with possible peanut allergy who were assessed by challenge at

St Mary's Hospital, London, a skin pricked weal size of at least 8 mm in diameter had a 95% (95% confidence interval 76–100%) positive predictive value for a positive challenge. A serum specific IgE result of at least 15 KU/l had a positive predictive value of 92% (74–99) for a positive challenge to peanut. Similar data were generated by a number of other groups (9, 11–15). Cut off values are also available now for other foods. The use of these values has considerably improved efficiency in diagnosing food allergy while minimizing the number of food challenges that are required. A skin prick test result of <3 mm or a serum specific IgE level of <0.35 KU/l are generally interpreted as disproving allergy. Unfortunately a negative result can be seen in individuals with severe food allergy. Thus, where there is a clear history of allergy despite negative skin prick and serum specific IgE results, a food challenge is required.

There are, unfortunately, a number of diagnostic pitfalls associated with the use of skin prick testing and serum specific IgE results. The first concern is whether the data in the literature are generalizable to the particular patient in question. With the growing number of groups who are publishing similar data, this is becoming less of a concern. The 95% positive predictive values for smaller children are probably less than the values of those generally utilized (see Box 3). Secondly, different skin prick test methodologies and serum specific IgE systems may not give comparable results. Thirdly, saying that the cut off values represent a 95% positive predictive value for clinical allergy is an over simplification. This figure is derived by grouping all the subjects with results at or above the cut off and looking at how many have clinical allergy. If this group of subjects is further sub-divided, one finds that those with results at or around the cut off point actually have a probability of clinical reaction of 85–90%. Lastly, the precision of these cut off points needs to be considered. For each cut off point, the 95% confidence interval for the positive predictive value for allergy is only around 75–100%. These cut off points may,

Box 3. Food specific results diagnostic of food allergy

	Peanut	Egg	Milk
Serum specific IgE (kU _A /l)	15	7 (2)	15 (5)
Skin prick test (mm)	8 (4)	7 (5)	8 (6)

Results of at least the following values are 95% predictive of clinical allergy. Numbers in brackets represent the equivalent diagnostic value for children up to 2 yr of age (15, 36, 37).

therefore, provide a false sense of security about the diagnosis of a food allergy.

Positive predictive values are also dependant on the prevalence of the disease in the population. A test may seem to perform well in a population with a high rate of food allergy (e.g. hospital allergy clinic) but poorly where the prevalence is low (e.g. family doctor's practice). A cut off value also does not allow other clinical information such as the history to be taken into account. One way to deal with both of these issues is to utilize likelihood ratios. The likelihood ratio is the likelihood of a given result in a clinically allergic subject as a ratio of the likelihood of a similar result in a normal subject. Likelihood ratios can be calculated for each skin prick test or serum specific IgE result. A nomogram (see Fig. 1) is utilized to estimate the probability that each subject is clinically allergic. This is dependant on the likelihood ratio and the patient's pre-test probability. So case 1 with a single typical history of a type 1 hypersensitivity reaction has an approximate 50% chance of being clinically allergic on the basis of history alone. The likelihood ratio for a skin prick test of 5 mm is 6. From the nomogram, it can be seen

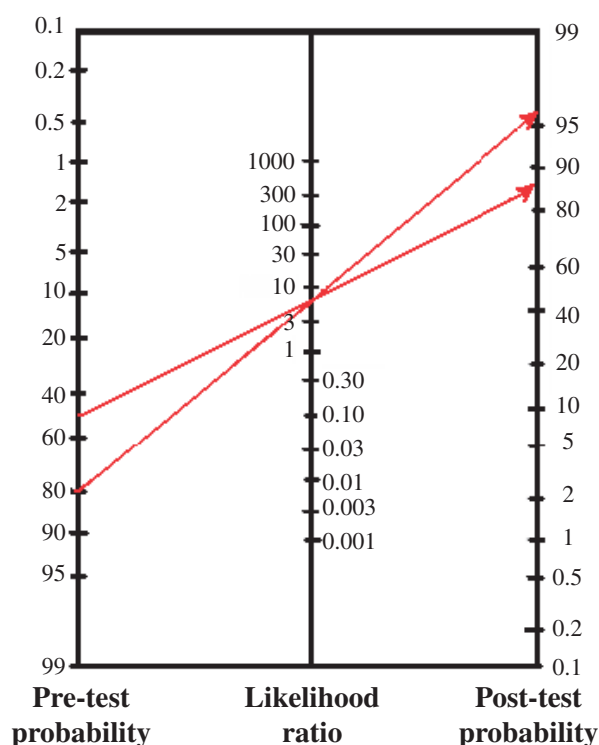


Fig. 1. Normogram for generating post-test probabilities from likelihood ratios. The use of likelihood ratios is demonstrated by two clinical examples. Adapted with permission from Fagan TJ. Nomogram for Bayes's theorem. *N Eng J Med* 1975; 293: 257.

that the probability that the case is clinically allergic to peanuts is 85%. If the child had suffered another similar allergic reaction a few weeks later on contact with peanuts, the pre-test probability might have increased to 80%. With a similar skin prick test result the post-test probability would go up to >95%. In this way we can derive maximal usage from the information from the history and test results. It must be remembered although that history and investigations can only give us an estimate on whether or not a child is clinically allergic. It is important that the family appreciates this and they are involved in the decision on whether or not a child needs a food challenge to rule in or rule out a clinical food allergy. Different families have different approaches to risk. For example, one family may be happy to avoid peanuts and tree nuts if their child just has a 50% chance of being peanut allergic whereas another family may want to know that their child is 95% likely to allergic before they introduce a lifelong peanut and tree nut ban.

Case 2: does this child need a self-injectable adrenaline device?

An 8-yr-old girl attends the allergy clinic with a history of reacting to peanuts and tree nuts. A couple of years ago, her father kissed her on the cheek after he had eaten a curry containing peanuts. Within an hour she had developed a raised itchy rash on her cheek. A few weeks prior to their appointment she was eating an ice cream cornet. She immediately developed an itchy mouth and was violently sick. She had previously tolerated ice cream and eats all the common food groups without problems. She had not had nuts before but her parents say she would have been exposed to them as traces in foods. She is otherwise well apart from asthma for which she occasionally uses a salbutamol reliever inhaler. Skin prick testing clinic reveals a 10-mm weal to walnut and serum specific IgE results of >100 KU/l for peanut, 50 KU/l for walnut, 5 KU/l for brazil nut and 2 KU/l for almond. A diagnosis of peanut and tree nut allergy was made. Does she need access to a self-injectable adrenaline device?

Who needs self-injectable adrenaline device?

There has been a huge increase in the rate of prescriptions for self-injectable adrenaline. In the United Kingdom there has been a sevenfold increase over a 10-yr period meaning that 0.2% of the paediatric population have been

prescribed one (16). In Canada although, 1.4% of children have a prescription for self-injectable adrenaline (17). Are too few devices being prescribed in the United Kingdom or are the Canadians prescribing too many? A decision of whether or not to prescribe a self-injectable adrenaline device depends on the risk of a future life-threatening anaphylactic reaction, the benefit of having a self-injectable adrenaline device, the risk associated with using one and the affordability of the intervention within the health service. There are two useful cohort studies providing information about risk of future life-threatening allergic reactions. Ewan and Clarke in the United Kingdom have prospectively followed 747 children with peanut or tree nut allergy for a median of 3 yr. In the 395 children with previous non-life-threatening reactions, 17 (4%, 95% confidence interval 2–7%) had a severe reaction during follow-up (18). Van der Leek et al. (19) followed 83 young children with peanut allergy in the United States for a median of 6 yr. Of the 61 who had an initial non-life-threatening reaction, a third subsequently had a severe cardio-respiratory one. The rate of severe reactions in the mild group is very different in these two studies. Possible explanations for this are that the American study focussed on the first reaction whereas many of the children in the United Kingdom study had had more than one reaction by the time they were seen in the allergy clinic. Additionally, only the United Kingdom children were seen annually; this would have afforded much more opportunity to ensure that families were practicing adequate avoidance. Both these studies emphasize that with peanuts there is no group that is at a low risk for a future severe reactions. Even in a mild group, the risk of a subsequent severe reaction is still 1–5% per year per subject. Most allergists are therefore prescribing a self-injectable adrenaline device to all children with peanut or tree nut allergies.

The other reason for prescribing the self-injectable adrenaline device for this girl is her co-existent asthma. Most fatal cases of anaphylaxis occur in subjects who also have asthma (2, 20, 21). In a cohort of 46 peanut allergic children followed for 2–14 yr, four died of asthma during follow-up (22). It is tempting to speculate whether these fatalities are actually due to bronchospasm precipitated by contact with peanuts. Other potential indications for prescribing self-injectable adrenaline devices are allergic reactions to a trace amount of a food, remoteness from medical care and being a teenager. All of

these have been associated with an increased risk of a future severe allergic reaction (7, 23–25). As professionals, we can objectively estimate the risk of a future episode of anaphylaxis for each patient. Families and patients although are influenced by other subjective factors which may elevate their perceived risk of anaphylaxis beyond this objective estimate. These anxieties may relate to lack of control, personal experience and information from the media and friends. No risk, however small, may be acceptable to parents.

Unsurprisingly there is little evidence based literature to assess the effectiveness of self-injectable adrenaline. There are good pharmacological reasons for using it and some supportive animal data. There are data to suggest that the early use of adrenaline is associated with better outcomes in anaphylaxis (20, 26). Furthermore, the risks associated with self-injectable adrenaline are minor compared with the risk to a life of anaphylaxis. Typical ones are palpitations and tremor. There is also the risk of accidentally injecting a digit (27). Additionally, the inconvenience of carrying and regularly replacing devices, need to educate all carers and the potential stigma associated with requiring a self-injectable device as well as the cost of devices need to be considered (28).

In summary, we currently have only sufficient evidence to guide decision making for peanut and tree nut allergy in children. There is no current consensus as to who needs a device and who does not although most clinicians would agree that a child with previous severe reaction or one with co-existent moderate and severe asthma should be prescribed a self-injectable adrenaline device (personal practice presented in Box 4). The family must be involved in this decision making and the self-injectable adrenaline device is only part of a comprehensive package of family education about avoiding food allergens, recognizing and managing an allergic (an example of a personal management plan is presented in Box 5) (29–31).

Box 4. Personal indications for self-injectable adrenaline device

Absolute indications

Previous allergic reaction with respiratory or cardiovascular symptoms or signs

Co-existent asthma

Relative indications (usually prescribed)

Peanut or tree nut allergy

Allergic reaction to trace quantities of a food

Remote from medical care

Teenager

Box 5. Example of a personal management plan

This girl is at risk of a severe allergic reaction from peanuts.

1. If she eats any of the above by mistake, she should immediately receive *Cetirizine* 7.5 mg followed by six puffs of her *Ventolin* via naerochamber plus.
2. If she develops breathing difficulties, noisy breathing, becomes blue, drowsy or unresponsive, she should be given the *Epipen junior syringe* 0.15 mg into the upper, outer thigh.
3. If *Epipen* given, dial 999.
4. If she has not improved within 5–10 min, she should be given a second *Epipen junior* and six puffs of her *Ventolin*.
If she experiences noisy breathing or difficulty breathing, she should be given 40 mg of soluble *prednisolone*, dissolved in water, within 1 h of reacting. This is to prevent a subsequent asthma attack.

Case 3: novel allergens

A 14-yr-old girl presents to the allergy clinic with peanut allergy, persistent milk allergy and asthma. She is extremely rigorous in her avoidance of nuts and cow's milk and has not had a reaction for some years. Recently, within 30 min of eating a cow's milk free chocolate bar, she had an anaphylactic reaction. She developed a raised rash on her face, felt nauseous and had chest tightness. She took 10 mg of cetirizine and six puffs of her salbutamol inhaler and was much better 10 min later. She had no late phase problems. She has brought the chocolate bar wrapper to clinic with her. The ingredients are raw sugar 35%, cocoa powder 28%, sweet lupin flower 22%, cocoa mass 14%, emulsifiers and sunflower lecithin.

Skin pick testing in clinic reveals an 8-mm weal to peanut, 6 mm weal to cow's milk and 12 mm weal to lupin. There was no reaction to the tree nuts, soy and the negative control but there was a good positive control. Her serum specific IgE to peanut and lupin were > 100 KU/l and 14.5 KU/l respectively.

Has a novel allergen caused this case of anaphylaxis?

In most cases, the allergen is obvious being one of the usual suspects (Box 2). However, sometimes, determining the cause of an anaphylactic reaction requires considerable detective skills. Successful identification of the allergen is aided by the availability of the ingredient list and a knowledge of which food allergens cross-react. Lupin is a recent novel food allergen. It is a member of the legume family like peanuts, soy, pea and broad beans. There are very isolated case reports of lupin being an allergen over the last 10 yr (32, 33). Lupin flower is increasingly being used in large-scale food manufacturing. It is used as a wheat or soy substitute in foods such as

bread, pastries, pizza, pasta, pitta bread and yogurt. Given the biological similarity between peanuts and lupin, it is not surprising that there is allergenic cross-reactivity between these foods. In a small series of peanut allergic subjects in France, the risk of being allergic to both lupin and peanut has been estimated to be 28% (34). Given this high rate of cross reactivity, it is somewhat surprising that lupin has been introduced into our food chain without allergic consumers being alerted. There is now new European Union legislation on food labelling that ensures that 12 allergenic foods are highlighted on food labels (peanuts, tree nuts, soy beans, egg, milk, fish, crustaceans, celery, mustard, sesame seeds, cereals containing gluten, sulphur dioxide and sulphites). Unfortunately there is currently no legal requirement for the presence of lupin to be highlighted in food, although this seems to be associated with severe reactions (32). The lupin story illustrates the problems associated with introducing a potentially allergenic food into our diet without first consulting the allergic consumer or healthcare professionals.

Conclusions and future perspective

In this article, three crucial areas of the management of anaphylaxis because of food allergy: making a diagnosis, deciding who needs self-injectable adrenaline device and spotting the novel allergen have been discussed. Establishing clinical management guidelines for patients with anaphylaxis is challenging due to the lack of evidence in this area. What evidence that is available needs to be utilized and practical consensus based protocols developed. A European Academy of Allergy and Clinical Immunology working party has recently developed guidelines for paediatric anaphylaxis (35). These also highlight where we are lacking clinical data, for example the pharmacokinetics of adrenaline in infants and small children.

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Answers from previous issue

1. a-F; b-F; c-T; d-T; e-F
2. a-T; b-T; c-T; d-T; e-F
3. a-T; b-T; c-T; d-F; e-T; f-T
4. f
5. e-T