

Food Allergy in Clinical Practice

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ABSTRACT

Food allergy is increasingly prevalent in the US, with as many as 3 million individuals reporting allergies to peanuts or tree nuts and more than 6.5 million to seafood. Food-induced allergic reactions are responsible for a host of symptoms involving the gastrointestinal tract, respiratory tract, and skin. Though it is possible for any food to provoke an allergic reaction, relatively few foods are responsible for the majority of food-related reactions. Ongoing research seeks to identify new therapeutic strategies for treatment of food allergy; however, the only current treatment for patients is avoidance of their food trigger.

Keywords: anaphylaxis, case study, food allergy, food allergy guidelines, serum-specific IgE testing, skin prick test
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In contrast to a generation ago, it is common to know a family member or friend with a food allergy. Though food allergies often present in childhood, they may be diagnosed at any age. Food allergy is the most common cause of anaphylaxis treated in the emergency department (ED) in all age groups.¹ In the past decade, hospital admissions related to food-induced anaphylaxis have increased more than 3-fold.¹

The National Institute of Allergy and Infectious Diseases (NIAID) publishes food allergy guidelines designed for health care providers in both specialty and primary care practice settings. The NIAID is 1 of the 27 institutes that make up the National Institutes of Health (NIH), an agency of the Department of Health and Human Services. These guidelines offer an evidence-based template for the diagnosis and management of food allergies.² This article and case

This CE learning activity is designed to augment the knowledge, skills, and attitudes of nurse practitioners and assist in their understanding of the diagnosis and management of food allergies.

At the conclusion of this activity, the participant will be able to:

- A. Explain how food allergies are diagnosed
- B. Describe the management of food allergy patients
- C. Critique the 3 causative theories for the rising prevalence of food allergies

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study focus on the clinical application of the NIAID guidelines for food allergy patients.

DEFINITION

Allergy is defined as a condition triggered by a specific, acquired immune response to a harmless, non-pathogenic antigen. An antigen is any substance that causes the immune system to produce antibodies against it. The allergic reaction results from the production of specific Immunoglobulin E (IgE) antibodies, 1 of the 5 major classes of immunoglobulins, directed against innocuous and typically widespread antigens. An antigen in this type of hypersensitivity reaction is called an *allergen*. Although any food can trigger an allergic response and more than 170 foods have been reported to cause an IgE-mediated reaction, 8 foods have been identified as causing 90% of food allergies (Table 1).³

Food allergy is an abnormal immune system response to a food protein.² The mechanism of action of a food allergy involves a local response in the mucosal immune system, which then drives a systemic response. Food allergy is typically IgE mediated and is distinct from other adverse responses to food, such as food intolerances and mixed IgE- and non-IgE-mediated reactions.⁴

PREVALENCE

In the US, 3 million people report being allergic to peanuts or tree nuts and more than 6.5 million people report being allergic to seafood.⁵ Food allergy affects 8% of children and 4% of adults, and its prevalence is increasing.⁶ Children are particularly affected by food allergies. In the past decade, the rate of food allergies among US children under 18 years has increased 18%.⁶ It is estimated that 2 children in every US classroom have food allergies.⁷

Table 1.8 Foods That Cause 90% of Food Allergies

Cow's milk
Egg
Fish
Peanut
Shellfish
Soy
Tree nut
Wheat

THEORIES OF CAUSATION

Dietary Hypothesis

It is theorized that individuals who grow up eating a common Western diet have less biodiversity of organisms in their gut compared to those growing up in Southern Europe eating a Mediterranean diet. Typically a Western diet contains more sugar, animal fat, and calorie-dense foods than a Southern European or Mediterranean diet.⁸ The decreased intestinal biodiversity, in theory, leads to increased prevalence of food allergies. Foods typically considered a part of a Mediterranean diet include plant foods; fresh fruit as dessert; high consumption of beans, nuts, cereals, and seeds; olive oil as the main source of dietary fat; cheese and yogurt as the main dairy foods; moderate amounts of fish and poultry; limited eggs (no more than 4 per week); limited meat consumption; and 25% to 35% of calorie intake from fat, with no more than 8% of calorie intake from saturated fats.

A large birth cohort study, after controlling for confounding factors (parental atopy, early onset of eczema, and wheeze), found that regular fish consumption during the first 12 months of life was associated with a decreased risk for allergic sensitization to foods by 4 years old.⁹ This was an observational birth cohort study, and therefore the association between increased oily fish consumption and reduced IgE allergic sensitization to foods is not necessarily causal.

Hygiene Hypothesis

A second posit is the *hygiene hypothesis*. Sanitation measures and vaccines in the West may have controlled infectious disease, but they decreased exposure to a variety of bacteria that may have opened the door to allergies. This hypothesis states the natural development of the immune system is suppressed by a lack of early childhood exposure to infections, symbiotic microorganisms, and parasites.¹⁰ This immune suppression leads to increased susceptibility to allergic diseases, including food allergy.

In a study of the effects of early exposure to food allergens, wheat allergies were 4 times more prevalent in infants who were exposed to cereal grains (wheat, barley, rye, oats) after 6 months compared with those who were exposed earlier than 6 months (1.8% vs. 0.41%).¹¹ Consistent with the Hygiene Hypothesis, this finding supports that early exposure

to food allergens has a protective and beneficial effect for the child. The study followed 1,612 children for an average of 4.7 years.¹¹

Air Pollution Theory

A third causative theory is that air pollution exposure during infancy may contribute to sensitization to certain food allergens. A recent study of 614 children showed that exposure to pollutants during the first year of life was directly linked to an elevated risk of pollen sensitization at 4 years old and food allergen sensitization at 8 years old.¹² The food allergy sensitization was particularly strong in children without earlier sensitization. Of note in the findings was that no association existed between air pollution exposure after the first year of life and sensitization. The researchers discussed that the infancy period presented a potentially valuable time window of susceptibility to various risk factors, including air pollution.¹²

NATURAL HISTORY

In an IgE-mediated response, foods enter the gastrointestinal (GI) tract and undergo protein digestion and then antigen (Ag) processing. Sensitization begins with the transit of food antigens across the intestinal barrier. As the antigen-presenting cell presents Ag to the T cell, specific cytokines are produced. The T cell, in turn, regulates specific IgE production by B cells. The specific IgE then attaches to mast cells in the GI tract, skin, and respiratory tract mucosa. Subsequently, on ingestion of the allergic food, the protein is digested and the Ag binds the specific IgE on the mast cell, causing activation of the mast cell with mediator release at the mucosal site.¹³

There are 3 phenotypes associated with food allergies: transient food allergy, persistent food allergy, and oral allergy (food-pollen) syndrome.¹⁴ Emerging research suggests that each of these forms of IgE-mediated food allergy results from different immune-based mechanisms. Consequently, they require different approaches in treatment.

Transient food allergy patients appear to have the most favorable response to avoidance diet therapy as their food allergies only last a few years. Though some health care providers may argue that transient food allergies do not require treatment, the benefit of implementing an avoidance diet accelerates the

development of tolerance and improved quality of life and nutrition.¹⁴

Persistent food allergy patients continue to have food sensitivities year to year and are often more challenging to treat because of the chronic condition of their diagnosis. Adhering to a strict avoidance diet as treatment for persistent food allergies can be very difficult for patients due to cost, inconvenience, or lifestyle alteration. Compared to transient food allergy patients, these patients need a more prolonged treatment course and may develop greater serious adverse reactions during therapy.¹⁴

Oral allergy syndrome (OAS) is an IgE-mediated immune response provoked by eating certain (usually fresh) fruits, and vegetables typically develop in patients with tree or weed pollen allergies. OAS represents cross-reactivity between the proteins in tree or weed pollen and certain fruits and vegetables. OAS is most often limited to uncooked fruits or vegetables.¹⁵ Patients with oral allergy syndrome often respond favorably to avoidance treatment of the allergenic food.

In the past several years, the IgE-binding epitopes (any site on an antigen at which an antibody can bind) of different food allergens have been identified.¹⁴ Foods may contain either or both *conformational* or *sequential* epitopes. A *conformational* epitope has a specific 3-dimensional shape, where as a *sequential* epitope has a linear shape.

Patients with sensitivities to *sequential* epitopes have been associated with persistent milk and egg allergy.¹⁴ Research demonstrates that extensive heating and food processing destroys *conformational* epitopes and only the *sequential* IgE epitopes survive.¹⁴ Studies show that patients with persistent milk or egg allergy (with sensitivities to *sequential* epitopes) are unlikely to tolerate baked foods containing milk and egg. This is because the heating process of milk and eggs limits its destruction to the *conformational* epitopes present in the foods, leaving the *sequential* epitopes intact.

On the other hand, patients with milk and egg allergy who have sensitivities to *conformational* epitopes tend to tolerate foods with baked milk and egg and eventually are able to tolerate the whole food. Though not currently available for clinical use, IgE-binding epitope testing may be seen in the future.

SYMPTOMS

Symptoms of food allergy vary from person to person, as does the amount of food needed to trigger a reaction (Table 2). A patient's presentation may range from 1 or 2 of these symptoms to virtually all of them. Children with food allergies classically present with symptoms of vomiting and urticaria. Adults with food allergy frequently have reactions that involve urticaria, angioedema of the lips and face, and abdominal symptoms. Extragastrintestinal symptoms are common and may be the only symptoms present (eg, urticaria).³

DIAGNOSING FOOD ALLERGIES

The NIAID guidelines recommend a skin prick test (SPT) to identify foods that may be provoking IgE-mediated, food-induced allergic reactions.³ SPTs are the cornerstone of allergy practice and have been used for over 100 years. However, SPTs alone cannot be considered diagnostic of food allergy.⁷ Both a detailed medical history (Table 3) and a physical examination are needed to diagnose IgE-mediated food allergy.² The medical history should include possible provoking foods, the form or forms in which they were ingested (raw, semi-cooked, cooked, or baked), the quantity ingested, and the timing and nature of reactions.² Increasingly higher concentrations of a serum-specific IgE (ssIgE) result and larger SPT response size correlate with a mounting risk of a clinical reaction.²

The SPT wheal size is correlated with the likelihood of clinical allergy and 95% positive predictive thresholds (wheal size above which there is a > 95% chance of clinical allergy). Sporik, Hill, and Hosking¹⁶ demonstrated among children that it is possible to define

Table 2. Food Allergy Symptoms

Abdominal pain, diarrhea, nausea or vomiting
Anaphylaxis
Angioedema of lips, tongue, eyelids, or the whole face
Dizziness or fainting
Dysphagia
Flushing of skin
Pruritus of mouth, lips, tongue, throat, eyes, skin, or other areas
Rhinnorhea
Urticaria
Wheezing, coughing, or shortness of breath

Table 3. Specific Key Questions to Ask Patients

What particular food was suspected to have provoked the reaction?
How much of the suspected food was ingested?
What other foods were ingested at the time?
Are all of the ingested ingredients known?
How was the food prepared and served?
Did similar symptoms develop on other occasions when the food was eaten?
How much time elapsed between food ingestion and development of symptoms?
Does the patient have a history of avoiding or refusing to eat the suspected food?

SPT wheal size greater to control to egg, milk, and peanut above which an open oral food challenge (OFC) would be positive with 100% specificity: milk, 8 mm; egg, 7 mm; peanut, 8 mm.¹⁶

Intradermal testing (often used as adjunct testing to SPT for aeroallergens) is *not* recommended in the diagnosis of food allergy.²

When SPTs are not available or further evaluation is needed, ssIgE tests for foods are recommended.⁷ The NIAID recommends fluorescence enzyme-labeled assays for ssIgE allergy testing, such as ImmunoCAP, instead of previously recommended radioallergosorbent test (RAST) measurements.² ssIgE tests may not distinguish between similar proteins in different foods and may result in false positive test results, limiting its clinical correlation. While ssIgE tests have the advantage that patients are able to stay on their antihistamine medications (patients are required to abstain from antihistamines before SPT), serum tests are more expensive than skin tests.

When evaluating patients with suspected fruit and vegetable allergies whose SPT and ssIgE results are negative, the preferred method is the prick-prick technique:³ pricking the fruit or vegetable directly with a lancet and then using the same lancet immediately for the skin prick on the patient. Studies show the prick-prick method to be much more sensitive than SPTs with commercial extracts for most foods implicated in food allergy, particularly OAS.¹⁷

In the case where it is not possible to identify the specific food allergy by history and appropriate testing, patients with chronic symptoms may benefit from a trial elimination diet. Such diets are diagnostic

and may be used to determine allergenic foods that may be causing symptoms. Elimination diets are particularly beneficial when patients have multiple food allergies identified on positive sIgE testing.

The double-blind placebo-controlled OFC is the *gold standard* for food allergy diagnosis.² OFCs provide the most definitive means of diagnosis and generally consist of a gradual feeding of the test food under close observation. Although oral food challenges offer a more precise method for diagnosing food allergy, the complexities involved may not be suitable for all clinical situations. Food allergy can be ruled out if the OFC fails to elicit symptoms. In contrast, OFC is considered diagnostic if it evokes objective symptoms *and* those objective symptoms correlate with medical history *and* are supported by laboratory tests.⁷

It is uncommon for an individual to have an IgE-mediated food allergy to a food eaten on multiple previous occasions after having a reaction when eating that food originally. IgE-mediated reactions to foods are usually stereotypical, and therefore atypical reactions, such as a previously tolerated food causing a reaction or a delayed-onset reaction, suggests an alternative diagnosis.²

TREATMENT AND MANAGEMENT OF FOOD ALLERGIES

The treatment and management of food allergies is limited to strict dietary avoidance, nutritional counseling, and emergency treatment of adverse reactions.¹⁸ There is no standardized desensitization therapy currently available. Strict avoidance can be complicated when the patient has multiple food allergies. In the case that a patient is having difficulty avoiding multiple foods because of diagnosed sensitivities, it may be beneficial to prioritize those foods (to avoid) that have a higher sIgE and/or larger SPT response size so as to decrease the likelihood of clinical reaction.

The NIAID guidelines recommend that patients and families receive education on how to interpret ingredient lists on food labels.² The Food Allergen Labeling and Consumer Protection Act, which was most recently updated in 2006, mandates that the labels of foods containing the 8 major food allergens declare that allergen in plain language, either in the ingredient list or by using the word “contains”

followed by the name of the major food allergen.¹⁹ Food ingredients must be listed if they are present in any amount, regardless of how small that may be. Additionally, manufacturers must list the specific nut (eg, almond, walnut, cashew) or seafood (eg, tuna, salmon, shrimp, lobster) that is used in the product. Because food manufacturers can change their products’ ingredients at any time, patients must be taught to check the ingredient list *every time* before buying a particular food item. Products with precautionary labeling, such as “this product may contain trace amounts of allergen” also should be avoided.

SPECIAL MANAGEMENT CONSIDERATIONS

Pregnancy

Given the lack of evidence supporting the benefit of avoiding peanuts during intrauterine and early-life, the NIAID states that expectant mothers should restrict their diets only from those foods to which they are allergic. Any other diet restrictions during pregnancy, implemented as a strategic means of preventing the development of food allergy in their child, should be avoided.² This recommendation is the same for all pregnant women, including those who are considered high risk because they have a other conditions, such as asthma, eczema, or allergic rhinitis. Patients should continue to avoid foods they are known to be allergic to throughout pregnancy. The same recommendations apply to breastfeeding.⁵

Infancy

Three large cohort studies demonstrated that early introduction of peanut, milk, and egg into an infant’s diet decreases the future risk of IgE-mediated allergy to those foods.²⁰⁻²² The NIAID recommends all infants be exclusively breast-fed until 4 to 6 months old, unless breastfeeding is contraindicated for medical reasons. Breast milk is less likely to cause allergic reactions than any type of formula. For *high-risk* patients who have a parental or sibling food allergy history who are not exclusively breastfed, the use of hydrolyzed infant formulas, as opposed to cow’s milk formula, is recommended.

The introduction of solid foods should *not* be delayed beyond 4 to 6 months of age, unless risk factors are present.²³ After 4–6 months, single-ingredient (stage I) infant foods may be introduced 1 at a time. The single-ingredient foods may include fruits, vegetables, and cereal grains. The slow introduction process allows for the identification and elimination of any food that causes an allergic reaction. Delaying the introduction of potentially allergenic foods, even in infants at risk for food allergy, has not been clearly shown to be beneficial.²³

Childhood

When prescribing allergen-free diets for children, it is important to ensure that those diets have sufficient nutrients and calories to maintain a healthy and active life. Fortunately, the majority (85%) of children who are allergic to milk, egg, wheat, or soy will eventually develop a tolerance to those allergies. Conversely, only 15% to 20% of children who are allergic to peanut, tree nuts, fish, and shellfish will develop a tolerance to those allergies.³ Smaller SPT and lower allergen-specific serum IgE levels equate to a higher probability of the allergic patient outgrowing the food allergy.² Additionally, the severity of a patient's allergic reaction may be a factor when identifying the likelihood of developing tolerance. For example, if the child's allergic reaction is limited to 1 organ system, such as the skin (urticaria or eczema), the food allergy is more likely to be eventually outgrown compared to if they experienced anaphylaxis.²

Oral Immunotherapy

Among the most recent and promising food allergy treatments are findings from Burke et al²⁴ that oral immunotherapy can desensitize a high proportion of children with egg allergy.²⁴ Historically, patients with egg allergies adhere to strict avoidance, as that is the only currently approved treatment. In this double-blind, randomized, placebo-controlled study, 55 children, 5 to 11 years old, with egg allergy received oral immunotherapy or placebo. After 10 months of therapy, 55% of children in the oral-immunotherapy passed the OFC and were desensitized, and after 22 months, 75% of children in the oral-immunotherapy group were desensitized.²⁴ No children in the placebo group were desensitized.

FOOD ALLERGY ANAPHYLAXIS

Anaphylaxis is a severe allergic reaction that, within minutes of exposure to an allergen, produces signs and symptoms that may result in death if not promptly treated. Epinephrine injection is the first-line treatment for anaphylaxis in all patients. Research has demonstrated that a delay in the use of epinephrine for anaphylaxis treatment increases risk for death.² The use of antihistamines is the most common reason reported for not using epinephrine and may place a patient at significantly increased risk for progression toward a life-threatening reaction.²

The average onset of anaphylaxis is 5 to 30 minutes, and the signs and symptoms may vary (Table 4).²⁵ The most common areas affected in an anaphylactic reaction are skin (80%–90%), respiratory (70%), gastrointestinal (30%–45%), heart and vasculature (10%–45%), and central nervous system (10%–15%), with usually 2 or more systems being involved.²⁶

In some cases, a patient may experience a second anaphylactic reaction (known as a biphasic reaction) up to 72 hours after their initial recovery, despite no further exposure to the trigger. Studies show that a biphasic reaction can occur in up to 20% of all anaphylactic reactions.²⁷ Because biphasic reactions are unpredictable and always possible, it is recommended that patients at risk for anaphylaxis carry 2 doses of epinephrine.²⁷ When there is suboptimal response to the initial dose of epinephrine, or if symptoms progress, repeat epinephrine dosing remains first-line therapy over adjunctive treatments, such as antihistamines or steroids.²⁷

Food-dependent exercise-induced anaphylaxis (FDEIA) is a distinct condition in which anaphylaxis

Table 4. Anaphylaxis Signs and Symptoms

Angioedema of conjunctiva, face, lips, mouth, tongue, or throat
Cardiac—tachycardia, hypotension
Central nervous system—loss of consciousness, confusion, headache, anxiety
Epidermis—urticarial, pruritus, flushing
Gastrointestinal—abdominal pain, diarrhea, or vomiting
Nasal rhinorrhea
Respiratory—shortness of breath, wheezing, dysphagia, cough
Urinary incontinence

develops only if physical activity occurs within a few hours after eating a specific food. This disorder has been reported with a wide variety of foods.²⁸

The foods most commonly implicated in FDEIA include wheat, shellfish, tomatoes, peanuts, and corn.²⁸ Someone who has eaten an allergenic food and then exercises is at higher risk for exercise-induced anaphylaxis. Neither food intake nor physical activity by itself produces this type of anaphylaxis in these patients.² While the cause for FDEIA is unclear, there is speculation that exercise causes greater intestinal absorption of the food protein. As a result, higher concentrations of the protein circulate through the body and eventually come in contact with the mast cells, which then respond.

FDEIA is a particular concern for allergic school-children participating in physical education class or recess shortly after lunch. Keen observation by trained school personnel to the signs and symptoms of anaphylaxis is warranted in such cases.

CASE STUDY

SOAP Notes

Subjective. Ethan is a 19-year-old white male with a history of 2 severe allergic reactions in the past 4 weeks and daily pruritus who was seen in the clinic for a new patient evaluation. He reported that he developed hives and swelling of his lips after eating honey nut oat cereal with milk and a banana 4 weeks ago. His primary care provider treated his reaction with oral antihistamines and oral corticosteroid taper for 1 week, after which his urticaria and angioedema resolved.

The day after completing his prescribed corticosteroid medication, Ethan had a second allergic reaction while eating at a Mexican restaurant. He said that those symptoms (urticaria; angioedema of his lips, facial cheeks, and tongue; nausea; and pruritus of his throat and skin) were more intense than in the first reaction and they developed within 20 minutes of starting his meal. He had eaten tortilla chips, salsa, rice, black beans, and steak fajitas. While at the restaurant, his friend called 911 for emergency treatment. Ethan was given an injection of epinephrine 0.3mg IM and responded well with symptom improvement. He was transported to the local ED, and, after being evaluated, was prescribed oral antihistamines and a corticosteroid taper for 4 days.

Ethan said his current skin pruritus started after his second food reaction and persisted since that time. He reported trying to avoid Mexican food and honey nut oat cereal but admitted he has not totally eliminated these from his diet. He said he may be able to do better with avoidance if he knew exactly what he needed to avoid.

He also reported a history of perennial allergic rhinitis and drug allergy to clindamycin. He did not have any previously diagnosed food allergies, latex sensitivities, or venom reactions. He lived in a suburban home that is 36 years old and worked full time at a sub sandwich shop.

CASE STUDY QUESTION 1

Q: What questions would you ask to learn more about Ethan's allergy reactions?

A: Exploring a patient's *history of present illness* is the most valuable tool used for diagnosis food allergy. Pertinent questions to ask include: "Was there anything else you ate or drank those days when you developed the allergic reactions?" "Do you have prior history of this type of food reaction or anaphylaxis occurring?" "How much of the food had you eaten before having the reaction?" "Do you have a family history of food allergies or anaphylaxis?"

It is important to know everything Ethan ate at or around the time of his allergic reaction. A complete list of ingredients from the foods he ate at the Mexican restaurant, as well as the ingredient list from the cereal would be valuable for the clinical correlation of test results.

Ethan's prior history of any related food reactions or anaphylaxis will offer clues to possible contributors to current reactions. There is evidence that family food allergy history increases a patient's risk of developing food allergy.⁷

Asking Ethan about the quantity of food may lend information about the amount of food required to provoke a reaction or anaphylaxis. In general, the less food required inciting a reaction, the greater the patient's sensitivity to it.

After further questioning, Ethan said he had not eaten or drank anything other than what he previously reported, before the reactions occurred. He would get a list of ingredients from the both the restaurant and cereal box and bring those with him on his return visit. Ethan said that he has no prior history of any food reactions or anaphylaxis and that he developed symptoms 1 hour after finishing his cereal with milk, banana, and raspberries. He said that he had only eaten a portion of his Mexican food when his second reaction started.

Objective. His physical exam was normal. An SPT was conducted with foods, and he found to be positive (≥ 3 mm than control) to multiple foods. His largest SPT reaction was to peanut (6 mm > control). No additional labs were ordered.

Assessment.

1. SPT+ to almond, hazelnut, peanut, asparagus, string bean, blueberry, white seedless grape, hops, lettuce, mustard seed, green pea, green pepper, white potato, rice, and sunflower seed.
2. Pruritus: related to diagnosed food sensitivities
3. Anaphylaxis: food related

While there is not adequate evidence to determine which food allergy phenotype Ethan was at time of initial diagnosis, he is not likely to be *transient* because his onset of food allergies was postpubertal.³

Plan.

1. Ethan was provided educational handouts about his new diagnosis.
2. He was started on cetirizine 10 mg once daily and ranitidine 150 mg BID. The H2 antihistamine, such as ranitidine, may be useful in combination with H1 antihistamines, such as cetirizine. Research demonstrates an improved outcome in patients with acute allergic syndromes, including urticaria, who were treated with both H1 and H2 antihistamines.²⁹ An intramuscular 0.3 mg epinephrine auto-injector (2 pack) was prescribed, and its proper administration/indication were reviewed. The importance of prompt treatment with epinephrine and the

life-threatening nature of anaphylaxis were discussed.

3. He was recommended to start an avoidance diet of all foods to which he tested positive. Details of label reading were also discussed, and he was provided several educational resources.
4. Ethan was scheduled to return to the clinic for follow up in 2 weeks. At that time, the ingredient list, which he will provide, and his response to the prescribed avoidance diet and medications would be evaluated.

CASE STUDY QUESTION 2

Q: What information would you include when teaching Ethan how to approach his avoidance diet?

A: Strict avoidance of known food sensitivities is the only approved treatment for food allergies. Accurate food label reading is a must to avoid allergic reactions. Ethan needs to understand that product ingredients can legally change without notice. Since manufacturers may change their products' ingredients at any time, Ethan should be taught to check the ingredient list *every time* before he purchases a food product—even if that food product didn't cause a previous reaction. Some manufacturers include cautionary labels when there is a possibility that a food allergen could be present. Ethan should know that it is safest to avoid food items that have these labels, such as "may contain..." and "this product was processed in a facility with..." Ethan must know that contact with food even with minute amounts of allergen is enough to cause a serious allergic reaction. Washing and drying cooking utensils does not necessarily eliminate allergens. Cross contamination of packaged foods or foods eaten in restaurants are potential hazards.² Common sources of cross contamination include hands, utensils, cups, dishware, sponges, dishrags, kissing, and anything that has food or someone's saliva on it.

Follow Up Visits

Ethan returned to the office 2 weeks later symptom free and said his pruritus resolved within 1 week. He was continuing the prescribed cetirizine 10 mg daily and ranitidine 150 mg every 12 hours. He provided a list of ingredients from the Mexican restaurant and the box of cereal as requested. When cross-referencing the ingredient lists with his SPT, commonalities on both the ingredient list and SPT results included almond, blueberry, lettuce, green pepper, and rice.

Ethan was directed to challenge himself at home with those SPT+ foods that were less reactive (≤ 3 mm than control on testing) and were NOT on either the list from the Mexican restaurant or the cereal label. (He was SPT- to milk and banana.) The foods in that category to which he was to challenge included asparagus, string bean, white seedless grape, hops, mustard seed, green pea, and white potato. He was given instructions to restore those foods back into his diet 1 at a time in the space of every 3–5 days. He was asked to keep a food diary of symptoms occurrence. If his SPT had been larger to the foods prescribed for challenge, the challenge process would have taken place in the clinic for safety purposes.

Ethan returned to the clinic 8 weeks later and had successfully tolerated asparagus, blueberry, string bean, white seedless grape, hops, mustard seed, green pea, and white potato. He remained symptom free and did not have any urticarial, angioedema, pruritus, or allergic reactions to report in the interim. Ethan said he was feeling much better and had adjusted very well to his new avoidance diet. He is cautious by nature and said he still has to limit eating out at restaurants because he wants to maximize his diet control and minimize any possibility of anaphylaxis.

Because of Ethan's history of anaphylaxis and that the remaining SPT+ foods had either a moderate to large SPT response (defined as ≥ 3 mm than control—hazelnut, peanut, sunflower seed) or were on the Mexican restaurant list (lettuce, rice, green pepper) or on the cereal label (almond), he was directed to continue avoiding these food and re-check in 4 to 6 months. At that follow-up visit, the possibility of any future food challenges would be discussed.

CONCLUSION

Research demonstrates that multiple food allergies are common and have many moving parts to their diagnosis and management.^{29,30} Some patients with multiple food allergies may be provoked only when the allergenic foods are consumed in combination, yet those same patients may not experience any symptoms when eating those foods individually. Consequently, each food allergy patient's response to treatment and management is highly individualistic and their treatment plan should be reflective of such.

Food allergies account for 1 of the largest and increasingly prevalent antigen-type challenges confronted today. The NIAID Food Allergy Guidelines provide evidence-based recommendations that serve to optimize patient outcomes. While the current treatment and management of food allergy patients focuses on strict avoidance of the allergenic food, doing so often presents potential challenges for the patients. Food allergy patients need education on how to read product labels and the associated risks of anaphylaxis. More research is needed to define additional treatment strategies for this increasingly prevalent condition. **JNP**

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