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Food allergies: Updates for nurses

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Abstract: Food allergies are on the rise; the incidence and types of foods implicated have increased worldwide. While peanut allergies are the most well-known, allergies exist to almost all types of foods. This article discusses various types of food allergies along with the most recent prevention and treatment strategies.

Keywords: allergy types, anaphylaxis, epinephrine, food allergy, food introduction, food allergy sensitization

Allergic reactions can affect the respiratory, gastrointestinal, cardiovascular, and integumentary systems.[1,2] The prevalence of food allergies is higher in early childhood and decreases with age.[1]

The nine most common foods that trigger allergic reactions are shellfish, cow's milk, peanuts, eggs, fish, wheat, soy, sesame, and tree nuts.[2] Reactions to these allergens are classified into three categories: acute-onset IgE-mediated, delayed-onset mixed IgE and non-IgE-mediated, and cell-mediated.[1] Immediate responses are IgE-mediated and driven by mast cells, histamine release, and a cascade of additional inflammatory mediators. Allergic reactions leading to anaphylaxis are triggered through the IgE-mediated pathway.[3] This article discusses the different types of food allergies, treatment, and

prevention strategies. Incorporating this knowledge into clinical practice enables nurses to recognize at-risk patients, identify symptoms and food triggers, and provide appropriate patient and family guidance.

Epidemiology

Rates of food allergies are increasing across the world, likely affected by greater awareness and symptom identification.[1] Annually, there are 30,000 food allergy-related emergencies worldwide, which result in 100-200 deaths.[1] This increasing trend is particularly true for Westernized countries but is seen globally.[1] India has reported an increase in pediatric food allergies, although this may be due to increased awareness.[1]

Approximately 25% of the population has adverse reactions to at least one food item, which is more common than food allergies.[2] Adverse reactions can occur from any food or food additive and are immune- and nonimmune-mediated.[1] For example, lactose intolerance or rhinorrhea from spicy foods are adverse reactions, not allergies.[1,3,4]

Allergen sensitization can occur in response to pollen, fungi, dust mites, animals, insects, foods, food additives, and some materials such as latex (see *Types of allergic reactions*).[3,5-7] A hypersensitivity response requires previous exposure and sensitization to an allergen. Children with allergic comorbidities such as atopic dermatitis or eczema are more likely to have food allergies.[1]

Pathophysiology

A food allergy is an immune response to allergenic proteins found in certain plants or animal foods.[8] Nuts, for example, are particularly allergenic; even trace amounts can trigger an immune response and allergic reaction.[9]

The underlying etiology of food allergies is poorly understood. current evidence indicates major risk factors most likely include genetics, allergic comorbidities, exposure during pregnancy or breastfeeding, and environmental exposures.[10-11] Food sensitization may occur in infants or children through an impaired skin barrier caused by atopic rashes.[4,8-10] It is notable that infantile atopic dermatitis is suspected to be a risk factor for the development of food allergies.[1] This may be related to the loss of skin integrity, moisturizer choice, and opportunity for allergen exposure.[10,11]

Food allergies are also thought to be related to altered gut mucosa and microbiome variations.[8] The microbiome is affected by birth delivery methods, living conditions, feeding practices, antibiotics, and probiotics.[8] Additionally, current evidence demonstrates a low vitamin D level in infancy is associated with the development of food allergies.[1] While

Types of allergic reactions[5-7,22]

Pathophysiology

Acute onset IgE-mediated food allergy • True food allergy • Type I hypersensitivity • Mast cell and histamine driven	**Delayed onset** Non-IgE-mediated • Hypersensitivity to food • A group of disorders primarily affecting the GI system	**Delayed hypersensitivity**[a] Cell-mediated and mixed IgE • Type IV hypersensitivity • T cell driven
Symptoms or associated diagnoses		
Histamine-related food-induced rhinitis	Allergic proctocolitis	Atopic dermatitis
Urticaria	Food protein-induced enterocolitis	Contact dermatitis
Angioedema	Food protein-induced enteropathy	Eosinophilic gastroenteritis
Immediate GI hypersensitivity	Cow's milk protein induced iron deficiency anemia	Eosinophilic esophagitis
Delayed mammalian product GI symptoms	Dermatitis herpetiformis	Eosinophilic esophagitis
Food-dependent exercise-induced anaphylaxis	Celiac disease	Eosinophilic colitis
Asthma		Stevens-Johnson syndrome/toxic epidermal necrolysis
Pollen food/oral allergy syndrome		Drug-induced hypersensitivity syndrome
Anaphylaxis		

[a]Delayed hypersensitivity reactions are beyond the scope of this article

the association is unclear, vitamin D plays important roles in the immunologic response and has been implicated in allergic diseases.[12]

It has been suggested that exposure to food proteins through an altered skin barrier may promote sensitization, whereas oral exposure may be more protective.[8] Early exposure to nuts through cutaneous and oral routes has been studied.[8] Household surfaces and dust can carry trace amounts of peanut protein; atopic dermatitis increases the likelihood of allergen absorption.[8,13]

Oral allergy syndrome

Oral allergy syndrome (OAS), also called pollen-food allergy syndrome, is one form of allergic response related to food.[8] This syndrome is common in persons with allergic rhinitis, with up to 76% having an oral allergy reaction to at least one food and 70% reacting to more than one.[8] OAS occurs due to cross-reactivity between a plant pollen or environmental allergen to which a person has been sensitized and a particular food.[8]

Cross-reactivity examples that can trigger OAS include cantaloupe with Bermuda grass, bananas with ragweed, and pears with birch tree pollen.[8] Cross sensitivity may occur between shellfish and cockroaches or dust mites; this reaction to shellfish is more significant as it may progress to anaphylaxis.[8]

Although rare, OAS has been known to progress to anaphylaxis. OAS signs and symptoms include pruritis of the oral mucosa and can include edema of the mouth, face, throat, lips, or tongue.[8] These signs and symptoms occur after eating specific raw vegetables or fruits and can appear immediately or up to 1 hour afterward.[8] Offending foods can be cooked or peeled to decrease OAS symptoms. Severe or multisystem reactions are not common because stomach acids typically destroy the offending proteins [8,14] Systemic reactions are possible and occur more commonly with peaches, peanuts, tree nuts and mustard than with other foods.[14] Recommendations for eating offending foods may include peeling the skin or potentially pretreating with antihistamines, and vary based on the severity of signs and symptoms.[14]

Alpha-gal syndrome

Alpha-gal syndrome is an emerging food allergy to red meats and other mammalian products.[8] Alpha-gal (galactose-alpha-1,3-galactose) is a carbohydrate molecule found in most mammals.[8] This allergy is triggered after a tick bite, primarily by the Lone Star tick.[8] The Lone Star tick is found in Texas and Oklahoma to as far north as Maine.[15] The tick bite sensitizes the person to oligosaccharides present in mammalian products.

This allergic reaction causes primarily gastrointestinal symptoms with delayed onset. Signs and symptoms including abdominal pain, nausea, vomiting, and diarrhea often occur 2 to 6 hours after consuming food products from mammalian sources. Urticaria, angioedema, or anaphylaxis are less common in IgE-mediated food allergy, though initial allergic reactions can progress to the latter.[8] Alpha-gal syndrome can be challenging to recognize because of the delayed response and predominance of gastrointestinal signs and symptoms.[16]

Identifying food allergies

Allergy potential starts in infancy, so pediatric clinicians play an essential role in early identification. For example, difficult-to-treat atopic dermatitis in infants is somewhat predictive of the development of food allergies.[17] Additionally, males are more at risk for developing food allergies than females, although the etiology of this pattern is unclear.[1]

Experts contend that food allergies are both misdiagnosed and overdiagnosed.[17] Food allergies are IgE-mediated and will have objective, reproducible findings.[17] Food sensitivities are not IgE-mediated and do not result in anaphylaxis.[17] Blood and skin testing are screening tools and do not differentiate between sensitization and an allergy, which may result in false positives and overdiagnosis.[17] Unfortunately, no validated, standalone tests are available for food sensitivities. These tests were designed to be part of a comprehensive clinical assessment, not as routine screening tools for food sensitivity in the primary care setting. [17]

Anaphylaxis

Anaphylaxis is the most severe form of an allergy due to its rapid onset (minutes to several hours), life-threatening potential, and multisystem involvement.[18] A less severe allergic reaction typically only involves one organ system and does not progress to anaphylaxis. The United Kingdom and Australia report almost twice the rate of mortality from overall anaphylaxis than the US.[19] Anaphylactic reactions can be mild and localized or involve several different organ systems, complicating the diagnosis of anaphylaxis.[18] The risk of fatal anaphylaxis is not predictable and may present with mild signs and symptoms (see *When to seek emergency care*).[19,20] Children less than 4 years of age are at the highest risk for hospitalization due to anaphylaxis; however, the risk of death from anaphylaxis increases with age.[18,20]

Diagnosing anaphylaxis can be challenging due to the wide range of potential clinical manifestations, and the differentiation from a milder allergic reaction is not always easy to distinguish.[23] Many allergic reactions can appear similarly to the early phase of anaphylaxis with symptoms such as urticaria, swelling, and abdominal pain. Anaphylaxis involves

multiple body systems simultaneously and then progresses in severity to include hemodynamic or respiratory compromise.[18]

The most common cause of anaphylaxis is food, followed by medications.[18] Many foods can illicit an anaphylactic response, but the most common are cow's milk (for infants), peanuts, tree nuts, and shellfish.[18] Fatal food anaphylaxis is primarily preceded by respiratory signs and symptoms, which then trigger cardiovascular collapse.

During anaphylaxis, the immune system produces immunoglobulin E (IgE), which induces the release of mediators from mast cells and basophils, causing anaphylactic signs and symptoms.[18] The immune system produces IgE in response to allergens, including ingested foods, pollen, animal dander, dust mites, and medications.[24] Typically, patients with IgE-mediated reactions are also susceptible to some respiratory allergens, including seasonal allergies and asthma.[21]

The most common body systems affected by anaphylaxis are dermatologic, respiratory, and gastrointestinal (GI).[18] The most common clinical findings are skin and mucosal signs and symptoms; pruritis with or without urticaria; flushing; edema of the lips, tongue, and uvula; conjunctival edema, and periorbital edema. Respiratory signs and symptoms may include nasal congestion, rhinitis, dyspnea, stridor, a sensation of throat closure or choking, wheezing, or hypoxia.[18] GI signs and symptoms manifest most commonly in the oral mucosa with pruritus; tingling; and tongue, pharynx, and uvula edema. Patients may present with acute vomiting and be misdiagnosed with gastroenteritis. Other milder signs and symptoms may include nausea, abdominal pain, cramps, and diarrhea, particularly in alpha-gal syndrome.[18]

The cardiovascular and central nervous systems may also be involved in anaphylaxis. Cardiovascular signs and symptoms may present as diaphoresis, chest pain, syncope, tachycardia, bradycardia, hypotension, and end-organ dysfunction.[18] Central nervous system signs and symptoms include confusion, unconsciousness, hypotonia, headache, incontinence, and seizures.[18] Behavioral changes in infants and small children may also occur.[23] In severe cases of anaphylaxis, there is multisystem involvement, including intense bronchospasm, laryngeal edema, cyanosis, hypotension, and shock.[24]

Anaphylaxis can be life-threatening if not treated immediately. Clinicians should suspect anaphylaxis and initiate treatment if the individual experiences acute onset of signs and symptoms from any two body systems.[18] One significant sign of potential anaphylaxis is if the patient reports a sense of impending doom, which may be the beginning of a rapid decline and impending shock.[4] Progression and severity of a significant reaction is unpredictable; therefore, treatment should be initiated immediately for multisystem signs and symptoms.[25]

A biphasic reaction, a secondary or late-phase anaphylactic reaction, may occur several hours after the complete resolution of the initial reaction. Biphasic reactions can develop 4 to 12 hours after the initial reaction and last longer than 70 hours.[22] Biphasic anaphylaxis is not well understood and not common, but awareness of the potential is important when providing care for individuals with a history of anaphylaxis. If any signs or symptoms in the initial anaphylactic reaction return, they are usually first respiratory and GI, then cardiovascular.[25] Individuals with a history of anaphylaxis must be educated regarding this potential secondary reaction and be advised

When to seek emergency care[20]

Sudden onset of:

- Itching of lips, mouth, tongue, throat
- Facial swelling
- Rash, hives
- Wheezing
- Difficulty breathing
- Sensation of throat closure
- Noisy breathing
- Sensation of choking
- Vomiting
- Abdominal pain
- Confusion
- Behavioral changes
- Diaphoresis
- Racing heart beat
- Dizziness
- Chest pain
- Cyanosis

How to use an epinephrine autoinjector

1. Remove the pen from the package.
2. Remove the cap.
3. Hold the injector in your fist with the needle end pointing down. Never put your thumb, fingers, or hand over the needle end.
4. Firmly push the needle end against the outer midthigh—on skin or through clothes at a right angle (perpendicular to the thigh).
5. Hold in place for 3-10 seconds per the package instructions.
6. Remove the needle by pulling straight out.
7. Massage the area for 10 seconds after injection.
8. Get emergency help immediately. Give the autoinjector to emergency medical services (EMS).

to seek immediate medical attention whenever an initial dose of epinephrine is administered.

Treatment

First-line therapy is I.M. epinephrine administration with an autoinjector or syringe/needle.[20] Epinephrine is dosed by weight up to a maximum single dose of 0.5 mg/dose. A second dose may be needed if signs and symptoms do not appear to be stabilizing or improving, and may be given 5 to 15 minutes after the first dose.[22] The dose for infants weighing less than 10 kg is 0.01 mg/kg.[22] Children weighing 10 to 30 kg should receive 0.15 mg; children weighing more than 30 kg should receive 0.3 mg.[22] Utilizing the vial of epinephrine solution, the maximum dose is 0.5 mg.[22] Autoinjectors are prefilled with the epinephrine doses of 0.1 mg, 0.15 mg and 0.3 mg. While epinephrine is an essential treatment for anaphylaxis, fatalities have occurred despite timely administration. Therefore, it is imperative to seek immediate emergency care if an epinephrine autoinjector is utilized[19], additional doses of epinephrine may be necessary due to the unpredictable nature of anaphylaxis and the risk of a biphasic reaction.[22,25]

Medications that may be given as adjunctive therapies to epinephrine to treat anaphylaxis include H1 antihistamines (such as diphenhydramine), H2 antihistamines (such as famotdine), bronchodilators (such as albuterol), and glucocorticoids (such as methylprednisolone). None of these medications should be used as initial treatment or as monotherapy for anaphylaxis, as they do not relieve upper or lower respiratory tract obstruction, hypotension, or shock and are not lifesaving.[22]

Prevention

Environmental control

Avoiding foods that cause an allergic reaction is the primary way to manage a food allergy. Checking all prepackaged food labels and knowing which product constituents to avoid is critical. The Food Allergy Labeling and Consumer Protection Act of 2004 (FALCPA)[25] mandated that all manufacturers of packaged foods produced in the US label the most common food allergens.[26,27] Precautionary labels are printed on products made in the same facility as high-allergen foods, alerting consumers to contamination risks.

Those with allergies should avoid foods with precautionary labels to avoid a possible reaction. The FALCPA labeling requirements do not apply to the US Department of Agriculture and the Alcohol and Tobacco Tax and Trade Bureau.[27] There is always a concern for unintentional exposure to an allergen, which may occur despite the regulation of labels and avoidance of foods that cause an allergic reaction.

Food introduction

Early introduction of known allergenic foods may decrease the development of food allergies; however, it is not clear if breastfeeding in infants is protective against or a risk factor for the development of food allergies.[28,29]A definitive link between breastfeeding and the development of food allergies in infants has not been identified; therefore, elimination diets are not recommended for breastfeeding mothers.[8] Partially hydrolyzed, extensively hydrolyzed, and free amino acid-based formulas may provide benefits in allergy prevention when compared with cow milk formula.[28]

At the time of this writing, no published trials regarding breastfeeding and formula feeding compare the time of introducing allergenic foods. The Enquiring About Tolerance (EAT) trial[30] examined introducing allergenic foods in exclusively breastfed infants at 3 months of age. Still, the trial's results did not show a statistically significant increase in food allergies.[30]

Guidelines

The American Academy of Pediatrics (AAP) recommends introducing allergenic foods at 6 months of age (see *Key recommendations*).[11]

The American Academy of Allergy, Asthma, and Immunology (AAAAI) and the Canadian Paediatric Society (CPS) recommend that allergenic solid foods should be introduced after 4 months of life, around 6 months, and given regularly thereafter; there is insufficient evidence to support an exact dose and frequency of intake.[31]

The National Institute of Allergy and Infectious Diseases (NIAID) provides practitioners and parents

Key recommendations[11-13]

- Majority of infants should be given nonchoking forms of peanut protein no later than 6 months of age.
- Introduction of other allergenic foods, besides peanuts, should likely be introduced no later than 6 months of age, such as dairy, egg, nonchoking forms of tree nuts.
- A small percent (0.9%) of infants require in office testing, indications for high-risk infants include severe eczema or egg allergy.
- Once food is tolerated, continue food exposure three times per week.
- Prevent early sensitization and decrease atopic dermatitis through maintaining healthy skin barriers in infants leading to lower food allergies.
- Bathing and moisturizing no more than two to three times per week.
- Moisturize with trilipid skin barrier creams.
- Accurate diagnosis through appropriate testing and referrals reduce unnecessary food avoidance and food allergies, improve patient safety, and decrease patient/caregiver anxiety.

with specific strategies for food introduction.[31] Before peanut introduction, the guidelines recommend introducing non-allergic foods to determine infants' readiness for solid foods.[11] Recording a food diary of new foods may be helpful for parents.[32]

Practical interventions

Using hypoallergenic and sensitive skin moisturizers in infants beginning at less than 1 month of age, who are born into a family with a history of atopic dermatitis may prevent allergen sensitization.[11] However, Perkin et al.[10] have found that infants, with or without atopic dermatitis, who received frequent moisturizer application at an early age developed more food allergies.[10] The authors hypothesized that the moisturizer may have acted as a conduit for allergen exposure as the moisturizer could have picked up particles on the parent's hands. The authors suggest caregivers should diligently wash their hands and apply a trilipid skin barrier cream before moisturizing the infant.[10]

Introducing cow's milk, eggs, and non-choking forms of peanuts (see *Peanut feeding tips*) between 4 and 6 months of age may reduce the risk of food sensitivities.[11] Peanut allergies are rare in countries where peanuts are introduced in the first year of life and higher in countries where delayed introduction is common.[11] At-risk infants who are tolerant to allergenic foods should maintain consistent exposure to maintain tolerance (see *Introducing peanuts to infants*).[11]

Testing

Outdated guidelines, misinformation, and unnecessary testing or screening can lead to inappropriate dietary avoidance, patient or caregiver anxiety, and delayed introduction of foods that subsequently increase the risk of food sensitivities.[4,11] Those with severe atopic dermatitis, egg allergy, or both need to be medically assessed for peanut allergy.[11]

No single test completely identifies all food allergies.[13,33] Testing can be performed through an oral food challenge (OFC), atopy patch testing (APT), serum IgE tests, or skin prick tests (SPTs). OFC is considered the gold standard; however, its utility is limited due to the risk of triggering a severe allergic reaction.[13] Conducting OFCs is resource-intensive and often inappropriate in a primary care setting; the provider and clinical setting must be able to manage all possible reactions, including anaphylaxis.[34] Therefore, when food allergy testing is necessary, it should be initiated by allergy, asthma, or immunology specialists.[13]

APT is a less invasive option that is more diagnostic for late-phase allergic reactions and may be most sensitive in children less than 2 years of age.[33]

IgE testing interpretation is complex due to low specificity, variable sensitivity, and false positives. The positive predictive value of IgE tests is limited and must be interpreted in the context of individual patients.[13]

SPT specificity is impacted by multiple variables, including patient age and testing technique. For example, non-IgE-mediated reactions to cow's milk occur in children less than 2 years of age. Likewise, different foods may have varying sensitivi-

Peanut feeding tips[13]

	Peanut butter	Peanuts	Peanut flour or peanut butter powder	Bamba[a]
Amount containing about 2 g of peanut protein	9-10 g or 2 tsp	8 g or 10 whole peanuts (2.5 tsp of grounded peanuts)	4 g or 2 tsp	17 g or 2/3 of a 28 g (1 oz) bag or 21 sticks
Serving size	Spread on slice of bread (16 g)	2.5 tsp of ground peanuts (8 g)	N/A	1 bag (28 g)
Peanut protein per serving	3.4 g	2.1 g	N/A	3.2 g
Tips on feeding	- Mix with warm water, breast milk, or infant formula for smooth texture (let cool). - Mix with pureed, mashed fruits and vegetables, or yogurt and mashed potatoes.	- Blend to create a powder or paste. - Add 2-2.5 tsp of ground peanuts to yogurt or pureed fruit.	- Mix into yogurt or applesauce.	- Mix with warm water, breast milk, or infant formula for smooth texture, mash well (let cool). - Mix with pureed, mashed fruits/vegetables. - Sticks of Bamba for older children.

[a]Bamba—used in the LEAP trial and has known peanut protein content, has proven efficacy and safety. Other peanut puff products with similar peanut protein content can be used as a substitute.
Teaspoons (tsp) and Tablespoons are US measures (5 mL and 15 mL)

Introducing peanuts to infants[11]

No eczema or any food allergy (87% of infants)	Mild to moderate eczema (12% of infants)	Severe eczema and/or egg allergy (0.9% of all infants)				
Introduce safely at home around 6 months of age (developmentally appropriate and in agreement with family preference and cultural practices).	Introduce at home at 6 months of age.	Test in office if able and no access to a specialist or recommended to see an allergist.				
		Peanut serum IgE levels		**Peanut Skin Prick Test diameter**		
		≥0.35 IU/mL	<0.35 IU/mL	0-2 mm	3-7 mm	>8 mm
		Refer to specialist	Low risk of reaction; options based on preference		Moderate to high risk of reaction; options based on expertise	Probably allergic: avoid peanut and refer to a specialist
			Introduce at home at 4-6 months of age (developmentally appropriate)	Supervised feeding in the office at 4-6 months of age (difficult to complete with PCP, recommend involving a specialist).	Refer to specialist	

ties and specificities with SPT. A positive IgE test does not equal a clinically significant food allergy. IgE testing has a place in determining patient risks; however, considerable expertise is required to utilize and interpret these testing methods appropriately.[34]

Alpha-gal serum IgE testing is available commercially and is useful in assessing mammalian product allergies. Diagnosing meat allergy is important because it can mimic other GI disorders and there is cross-reactivity with gelatin.[34] Alpha-gal testing does not predict severity but is reliable for diagnosis with a positive blood test and delayed manifestation of clinical signs and symptoms (longer than 3 hours).[35]

Lastly, an OFC is beneficial for identifying foods causing reactions in a few different situations, such as when SPT and serum IgE testing are inconsistent with the patient's history or when a patient's diet is limited due to possible food allergies.[36] OFCs can be dangerous due to the risk of anaphylaxis. They must be limited to offices where I.V. access, emergency medications and equipment, and constant physician supervision are available. Before starting an OFC, appropriate doses of emergency medication should be readily available. Emergency response plans should also be in place.[36]

If considering an OFC, a few different protocols exist.[36] In one protocol, the food protein is introduced in 0.06 g/kg to 0.6g/kg amounts in three equal doses in 15-minute increments. The initial dose should not exceed 10 grams of food or 3 grams of pure protein. Patients are then monitored for 2 to 3 hours before an actual serving of the food is given, followed by an additional 2-to-4-hour observation. If the patient has had a previous severe reaction, monitoring increases to 2 to 3 hours between each food dose. Specialist input is critical for guidance after an OFC.[36]

Treatment

Other than avoidance of food allergens, treatment for a subset of patients with oral allergen immunotherapy or desensitization provides a temporary threshold for reacting to food allergens.[29] Oral immunotherapy is only recommended in children and only to eggs, cow's milk, and peanuts.[37] This type of treatment requires continuous exposure to the allergen but may cause anaphylaxis or a local reaction; the optimum treatment

frequency or duration for sustained desensitization is unknown.[28,37] Tolerance to allergens is generally improved with oral ingestion.[13]

Studies suggest omalizumab may be effective for treating patients who unintentionally consume a severe food allergen, possibly as monotherapy or in combination with oral immunotherapy.[37] Studies are ongoing and these drugs are currently not approved for treating food allergies.[37] Treatment for alpha-gal syndrome is an avoidance diet, which can be challenging due to inadequate food labeling and extensive use of gelatin in food and healthcare products.[16,35]

Nursing implications

Nurses are well-positioned to affect health outcomes related to food allergies positively. A key opportunity exists in identifying infants and children at risk for food allergies and providing appropriate patient and family guidance.

Research is ongoing and information regarding food allergies is changing regularly. Nurses should be aware of and implement recommendations on prevention, testing and treatment from recognized organizations such as the AAP and the AAAAI. Changes can be challenging for providers and families at risk to implement. The previous recommendations for avoidance versus current guidance for early food introduction epitomize this.

Nurses must understand multiple aspects of food allergies to intervene, such as the mechanisms of sensitization, strategies to desensitize or improve tolerance, and referral for specialized testing or treatment. Accurate information on details regarding OAS signs, symptoms, and pathophysiology, as well as when to suspect a severe allergic response, can significantly impact patients' lives and potentially prevent life-threatening situations. As noted in the case study, food allergy symptoms may be vague or not obviously related to the exposure, which makes diagnosis difficult.

The relationship between tick bites and the development of allergies to mammalian products is a recently recognized phenomenon that may be unfamiliar to patients, families, and even healthcare providers. Nurses can incorporate food allergy knowledge into practice to identify food allergy exposures, symptoms, and potential triggers.

Conclusion

Recommendations for introducing specific foods have evolved in recent years. Emerging evidence suggests the former recommendations to delay the introduction of allergenic foods contributed to the rise in food allergies. Re-education of providers, patients, and caregivers is needed.

Identifying high-risk infants is key in protecting against sensitization and improving tolerance to prevent progression to a true allergy. Maintaining a healthy skin barrier in infants will prevent early sensitization. Likewise, following food introduction recommendations with continued exposure to allergenic foods is also expected to improve tolerance and reduce the risk of developing food allergies.

Appropriate testing and referral are necessary to accurately diagnose and manage food sensitivities and allergies. Nurses should know about allergy signs and symptoms and allergenic foods and quickly identify and treat anaphylaxis.

Nurses are also well-positioned to recognize at-risk patients and educate them on food allergy prevention and care. Healthcare providers must apply the most recent evidence to reduce the incidence of food allergies, improve patient safety, and decrease patient and caregiver anxiety related to unnecessary food avoidance and food allergies.[11] ■

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Unless otherwise specified, the information in the preceding summaries applies to adults, not children. Consult the package insert for information about each drug's safety during pregnancy and breastfeeding. Also consult a pharmacist, the package insert, or a comprehensive drug reference for more details on precautions, drug interactions, and adverse reactions.

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