FOOD ALLERGIES GUIDELINES:

REFRENCES: National Institute of Allergy and Infectious Diseases
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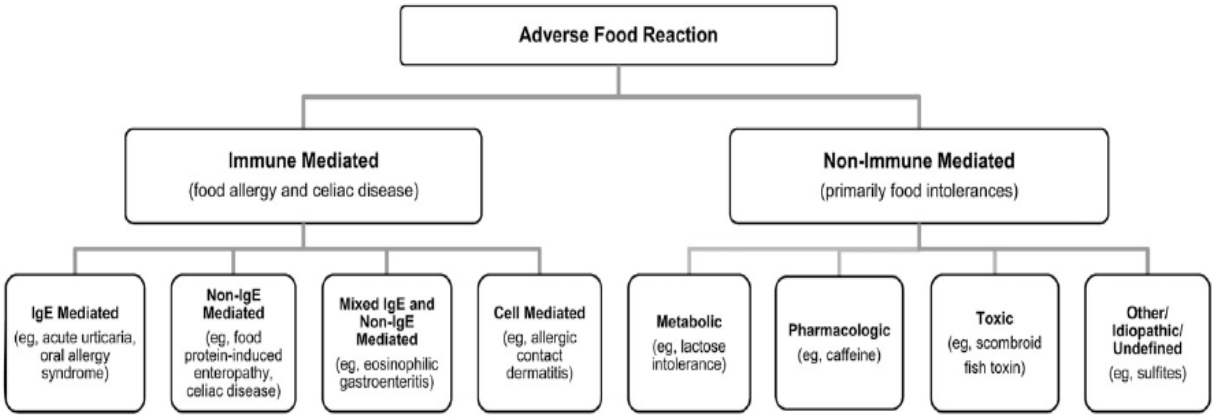

FIG 1. Types of adverse reactions to food

170 foods have been reported to cause IgE-mediated reactions.

FA for 5 foods: milk, egg, peanut, fish, and crustacean shellfish.

Prevalence of peanut allergy in the United States is about 0.6% of the population.

Prevalence of tree nut allergy in France, Germany, Israel, Sweden, and the United Kingdom varies between
0.03% and 8.5%.

Milk, egg, and peanut account for the vast majority of allergic reactions in young children.

Rates were significantly lower for children than for adults: fish allergy, 0.2% for children vs 0.5% for adults (p 50.02);
crustacean shellfish allergy, 0.5% vs 2.5% ($p < 0.001$); any seafood allergy, 0.6% vs 2.8% ($p = 0.001$).

Allergy to milk was suspected in 6.7% BUT confirmed in 2.2% IN A STUDY. 54% had IgE-mediated allergy, and the remaining 46% were classified as non-IgE mediated.

At the age of 2.5 years, the combination of prevalence of allergy and intolerance to milk IS estimated to be 1.1%.Most reactions to milk were not IgE mediated.

The prevalence of egg allergy Is estimated to be 1.6%, and most egg reactions were IgE mediated.

ONCE FOOD ALLERGY :
d 35% to 71% with evidence of AD
d 33% to 40% with evidence of allergic rhinitis
d 34% to 49% with evidence of asthma

The prevalence of FA in individuals with moderate to severe AD is 30% to 40%, and these patients have clinically significant IgE-mediated FA.

Egg allergy resolution or tolerance, defined as passing an
egg challenge or having an egg sIgE level <2 kUa/L and
no symptoms in 12 months, occurred in:
– 11% of patients by age 4
– 26% of patients by age 6
– 53% of patients by age 10
– 82% of patients by age 16
d Risk factors for persistence of egg allergy were a high initial level of egg sIgE, the presence of other atopic disease,
and the presence of an allergy to another food.

Specific examples of non-IgE-mediated adverse reactions to
foods include:
d Eosinophilic GI diseases (EGIDs)
d Food protein-induced enterocolitis syndrome (FPIES)
d Food protein-induced allergic proctocolitis (AP)
d Food protein-induced enteropathy syndrome
d Allergic contact dermatitis (ACD)
d Systemic contact dermatitis
d Heiner syndrome

SYMPTOMS:
FOOD ALLERGY : GI .

IMPORTANT POINTS:

1. Most children with FA eventually will tolerate milk, egg, soy, and wheat; far fewer will eventually tolerate
tree nuts and peanut. The time course of FA resolution in children varies by food and may occur as late as the teenage years. A high initial level of sIgE against a food is associated with a lower rate of resolution of clinical allergy over time.

2. For many patients, sIgE antibodies to foods appear within the first 2 years of life. Levels may increase
or decrease; a decrease is often associated with the ability to tolerate the foods.

3. AD and FA are highly associated. When tolerance develops to a food, the reintroduction of the food in the
diet will not result in recurrence or worsening of the AD.

4. FA may coexist with asthma, AD, EoE, and exercise-induced anaphylaxis.

5. The severity of a reaction cannot be accurately predicted by the degree of severity of past reactions nor by the level of sIgE or the size of the SPT wheal. The factor most commonly identified with the most severe reactions is the coexistence of asthma.

6. IgE-mediated reactions to foods are more common in young children, affecting up to
6% of children under 5 years old, and are more frequently seen inchildren with certain atopic disorders, such as AD.

7. IgE- and non-IgE-mediated mechanisms should be suspected when symptoms, which generally involve the GI tract,
are of a more chronic nature, do not resolve quickly, and are not closely associated with ingestion of an offending food.

8. medical history and physical examination to aid in the diagnosis of FA.

9. parent and patient reportsof FA must be confirmed, because multiple studies demonstrate
that 50% to 90% of presumed FAs are not allergies

10. SPT (SKIN PRICK TEST) alone cannot be considered diagnostic of FA. A positive SPT is generally considered a wheal with a mean diameter 3 mm or greater than the negative control.

11. SPTs are the most commonly performed procedure in the evaluation of IgE-mediated FA.

12. intradermal testing should not be used to make a diagnosis of FA.

13. the routine use of measuringtotal serum IgE should not be used to make a diagnosis of FA

14. Specific IgE tests for identifying foods that potentially provoke IgE-mediated food-induced allergicreactions, but alone these tests are not diagnostic of FA.

15. Atopy patch test should not be used in the routine evaluation of non-contact FA.

16. not to use the combination of SPTs, sIgE tests, and APTs for the routine diagnosis of FA.

17. elimination of 1 or a few specific foods from the diet may be useful in the diagnosis of FA, especially
in identifying foods responsible for some non-IgE-mediated food-induced allergic disorders, such as FPIES, AP, and Heiner syndrome, and some mixed IgE- and non-IgE-mediated food-induced allergic disorders, such as EoE.

18. oral food challengesfor diagnosing FA. The DBPCFC is the gold standard. However, due to the expense and inconvenience of DBPCFCs, single-blind and open-food challenges may be used in the clinical setting.

19. SPTs, sIgE tests, and APTsmay be considered to help identify foods that are associated with EoE, but these tests alone are not sufficient to make the diagnosisof FA. The role of these tests in the diagnosis of other EGIDs has not been established.

20. FPIES is diagnosed based on a supportive medical history, resolution of symptoms with the elimination of the causative food, and, in many cases, provocation of symptoms following an open or single-blind oral food challenge.

21. the medical history, resolution of symptoms when the causative food is eliminated from the diet, and recurrence of symptoms following an oral food challenge to diagnose Allergic proctocolitis.

22. the medical history, including the absence of symptoms while the causative food is avoided, and positive patch tests to diagnose Allergic contact dermatitis.

23. the medical history, including the absence of symptoms while the causative food is avoided, positive sIgE tests or SPTs, and positive immediate epicutaneous skin tests (for example, positive immediate responses

to APTs), to establish the diagnosis of food-induced IgE-mediated contact urticaria.

24. children less than 5 years old with moderate to severe AD be considered for FA evaluation for milk, egg, peanut, wheat, and soy, if at least 1 of the following conditions is met:
d The child has persistent AD in spite of optimized management and topical therapy.
d The child has a reliable history of an immediate reaction after ingestion of a specific food.

25. Overall, the risk-tobenefit ratio of FA evaluation should be considered on an individual basis, especially for the highly allergenic foods in high-risk young children.

26 it is not recommended using soy infant formula instead of cow's milk infant formula as a strategy for preventing
the development of FA or modifying its clinical course in at-risk infants

27. the use of hydrolyzed infant formulas, as opposed to cow's milk formula, may be considered as a strategy for preventing the development of FA in at-risk infants who are not exclusively breast-fed.

28. It is suggested that the introduction of solid foods should not be delayed beyond 4 to 6 months of age. Potentially
allergenic foods may be introduced at this time as well.

29. Patients at risk for developing FA are defined as those with a biological parent or sibling with existing, or history of, allergic rhinitis, asthma,AD, or FA.

30. the presence of any 1 of these criteria indicates that anaphylaxis is highly likely:
Acute onset of an illness (over minutes to several hours) involving
skin, mucosal tissue, or both (for example, generalized
hives, pruritus or flushing, swollen lips-tongue-uvula),
and at least 1 of the following:
– Respiratory compromise (for example, dyspnea,
wheeze-bronchospasm, stridor, reduced peak expiratory
flow rate, hypoxemia)
– Reduced blood pressure (BP) or associated symptoms of
end-organ dysfunction (for example, hypotonia (circulatory
collapse), syncope, incontinence) OR

d Two or more of the following that occur rapidly after exposure
to a likely allergen for that patient (minutes to several
hours):
– Involvement of the skin-mucosal tissue (for example,
generalized hives, itch-flush, swollen lips-tongue-uvula)
– Respiratory compromise (for example, dyspnea, wheezebronchospasm,
stridor, reduced peak expiratory flow rate,
hypoxemia)

– Reduced BP or associated symptoms of end-organ dysfunction (for example, hypotonia, syncope, incontinence)
– Persistent GI symptoms (for example, crampy abdominal pain, vomiting) OR

d Reduced BP after exposure to a known allergen for that patient (minutes to several hours). Reduced BP is defined:
– In adults, as a systolic BP of less than 90 mm Hg or greater than 30% decrease from that person's baseline
– In infants and children, as a low systolic BP (age-specific) or greater than 30% decrease in systolic BP.
Low systolic BP is defined as:
B Less than 70 mm Hg for ages 1 month to 1 year
B Less than (70 mm Hg plus twice the age) for ages 1 to 10 years
B Less than 90 mm Hg for ages 11 to 17 years
Note: In infants and young children, hypotension may be a late manifestation of hypovolemic shock. Tachycardia, in the absence of hypotension, also may indicate shock.

31. The highest risk groups for fatal anaphylaxis associated with food ingestion are:
d Adolescents and young adults

d Individuals with known FA and with a prior history of anaphylaxis

d Individuals with asthma, especially those with poor control (although fatal reactions may occur even in individuals with mild asthma)

TABLE VI. Summary of the pharmacologic management of anaphylaxis (modified[279])

Note: These treatments often occur concomitantly, and are not meant to be sequential, with the exception of epinephrine as first-line treatment.

In the outpatient setting

- First-line treatment:
  - Epinephrine, IM; auto-injector *or* 1:1,000 solution
    - Weight 10 to 25 kg: 0.15 mg epinephrine autoinjector, IM (anterior-lateral thigh)
    - Weight >25 kg: 0.3 mg epinephrine autoinjector, IM (anterior-lateral thigh)
    - Epinephrine (1:1,000 solution) (IM), 0.01 mg/kg per dose; maximum dose, 0.5 mg per dose (anterior-lateral thigh)
  - Epinephrine doses may need to be repeated every 5-15 minutes
- Adjunctive treatment:
  - Bronchodilator (β_2-agonist): albuterol
    - MDI (child: 4-8 puffs; adult: 8 puffs) *or*
    - Nebulized solution (child: 1.5 ml; adult: 3 ml) every 20 minutes or continuously as needed
  - H_1 antihistamine: diphenhydramine
    - 1 to 2 mg/kg per dose
    - Maximum dose, 50 mg IV or oral (oral liquid is more readily absorbed than tablets)
    - Alternative dosing may be with a less-sedating second generation antihistamine
  - Supplemental oxygen therapy
  - IV fluids in large volumes if patient presents with orthostasis, hypotension, or incomplete response to IM epinephrine
  - Place the patient in recumbent position if tolerated, with the lower extremities elevated

In the hospital-based setting

- First-line treatment:
  - Epinephrine IM as above, consider continuous epinephrine infusion for persistent hypotension (ideally with continuous non-invasive monitoring of blood pressure and heart rate); alternatives are endotracheal or intra-osseous epinephrine
- Adjunctive treatment:
  - Bronchodilator (β_2-agonist): albuterol
    - MDI (child: 4-8 puffs; adult: 8 puffs) *or*
    - Nebulized solution (child: 1.5 ml; adult: 3 ml) every 20 minutes or continuously as needed
  - H_1 antihistamine: diphenhydramine
    - 1 to 2 mg/kg per dose
    - Maximum dose, 50 mg IV or oral (oral liquid is more readily absorbed than tablets)
    - Alternative dosing may be with a less-sedating second generation antihistamine
  - H_2 antihistamine: ranitidine
    - 1 to 2 mg/kg per dose
    - Maximum dose, 75 to 150 mg oral and IV
  - Corticosteroids
    - Prednisone at 1 mg/kg with a maximum dose of 60 to 80 mg oral *or*
    - Methylprednisolone at 1 mg/kg with a maximum dose of 60 to 80 mg IV
  - Vasopressors (other than epinephrine) for refractory hypotension, titrate to effect
  - Glucagon for refractory hypotension, titrate to effect
    - Child: 20-30 μg/kg
    - Adult: 1-5 mg
    - Dose may be repeated or followed by infusion of 5-15 μg/min
  - Atropine for bradycardia, titrate to effect
  - Supplemental oxygen therapy
  - IV fluids in large volumes if patients present with orthostasis, hypotension, or incomplete response to IM epinephrine
  - Place the patient in recumbent position if tolerated, with the lower extremities elevated

Therapy for the patient at discharge

- First-line treatment:
  - Epinephrine auto-injector prescription (2 doses) and instructions
  - Education on avoidance of allergen
  - Follow-up with primary care physician
  - Consider referral to an allergist
- Adjunctive treatment:
  - H_1 antihistamine: diphenhydramine every 6 hours for 2-3 days; alternative dosing with a non-sedating second generation antihistamine
  - H_2 antihistamine: ranitidine twice daily for 2-3 days
  - Corticosteroid: prednisone daily for 2-3 days

IM, Intramuscular; *IV*, intravenous; *MDI*, metered-dose inhaler.

TREATMENT:

1. individuals with documented IgE-mediated FA should avoid ingesting their specific allergen or allergens.
2. individuals with documentednon-IgE-mediated FA should avoid ingesting their specific allergen or allergens.
3. In individuals with documented or proven FA who also have 1 or more of the following—AD, asthma, or EoE its recommended to avoide of specific allergen.
4. In individuals without documented or proven FA, it is not recommend avoiding potentially allergenic foods as a means of managing AD, asthma, or EoE.
5. nutritional counseling and regular growth monitoring for all children with FA.
6. Caregiver education is important
7. no medications currently recommended to prevent IgE-mediated food-induced allergic reactions
   from occurring in an individual with existing FA.
8. no medications currently recommended to prevent non-IgE-mediated food-induced allergicreactions from occurring in an individual with existing FA.
9. It is not recommended to use allergenspecific immunotherapy to treat IgE-mediated FA.
10. It is not recommended to use immunotherapy with cross-reactive allergens for treating IgE-mediated FA.
11. Milder forms of allergic reactions, such as flushing, urticaria, isolated mild angioedema, or symptoms of OAS, can be treated with H1 and H2 antihistamine medications.

Corticosteroids benefit allergic and inflammatory disease and also because they may help prevent biphasic or protracted reactions which occur in up to 20% of individuals

TABLE V. 2010 ACIP and AAP Red Book recommendations and PI information for administering vaccines to patients with egg allergy

Vaccine	ACIP	AAP Red Book	PI
MMR/MMRV	May be used[197]	May be used[198]	May be used with cautions, citing the 1997 AAP recommendations[199]
Influenza	Consult a physician[200]	Contraindicated[201]	Contraindicated[199]
Rabies	Use caution[202]	No specific recommendation	May be used with caution[203]
Yellow fever	Contraindicated, but desensitization protocols may be followed to administer vaccine if necessary (citing PI)[204]	Contraindicated, but desensitization protocols may be followed to administer vaccine if necessary (citing PI)[205]	Skin testing and desensitization protocols (citing 2000 AAP recommendations) provided in the PI[206]

AAP, American Academy of Pediatrics; *ACIP,* Advisory Committee on Immunization Practices; *PI,* package insert.