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Understanding IgE-Mediated Food Allergies

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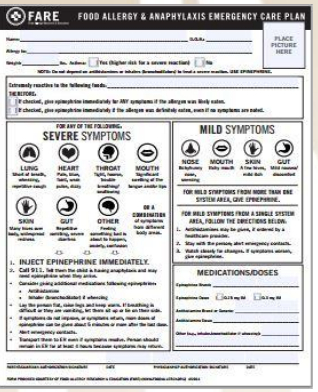
Introduction

Working as a nurse in a pediatric Allergy/Immunology clinic one would observe that the prevalence and resulting awareness of IgE-mediated food allergies in children has increased over the past decade. Many schools and camps are now going "nut free" and requiring Food Allergy Action Plans from a diagnosing physician. A study by Gupta et al. (2011) estimated 8% of children in the United States have a food allergy. Primary care providers (PCPs) are at the forefront of recognition, diagnosis, treatment and patient education of IgE-mediated food allergies. PCPs can benefit greatly from further education of the current evidence based diagnostic tools available. A review of the pathophysiological concepts of a true IgE-mediated food allergy reaction, and how it differs from non-IgE mediated food allergies can help guide diagnosis, treatment, and indications for referral to an allergist for more specific testing.

Figure 1 FARE Food Allergy Action Plan, 2016 www.foodallergy.org



Signs and Symptoms

IgE-mediated

IgE-mediated food allergy reactions are acute, immediate onset (minutes up to 3 hours), reproducible with every exposure regardless of source, and can progress to life threatening anaphylaxis. Symptoms include:

- Cutaneous: urticaria, angioedema, pruritus, flushing
- Ocular: periorbital edema, conjunctival erythema, lacrimation
- Upper respiratory: nasal congestion, sneezing, laryngeal edema, cough
- Lower respiratory: cough, dyspnea, wheezing, chest tightness, accessory muscle use/retractions
- GI: angioedema-lips, tongue, palate, pruritus of mouth, vomiting
- Cardiac: tachycardia, hypotension, dizziness
- General: sense of impending doom

Non-IgE Mediated

Non-IgE or food intolerance symptoms can occur hours to days later, can depend upon quantity consumed or source, and typically include GI symptoms- abdominal pain/cramping/bloating, diarrhea or constipation.
(Simmons, 2014)

Underlying Pathophysiology

- Due to failure of achieving oral tolerance through the robust T-cell mediated suppression that typically occurs with initial antigen exposure through GI tract (Vickery, et al., 2011)

First exposure: "Loading the gun" Sensitization- the first exposure to an allergen/antigen -such as Ara h 1 and 2, major offender allergens in peanut allergic patients

- Stimulated by antigen-presenting dendritic cells within the mucosal-associated lymphoid tissues of the GI tract, type 2 helper cell (Th2) are activated to produce cytokines such as IL-3, IL-4, IL-5. Th cells also produce IL-9, a growth factor of T cells also known for driving mast cell activation (Berin, 2015).
- DC-SIGN, a c-type lectin expressed on the surface of dendritic cells recognizes Ara h 1 (Vickery, et al., 2011).
- IL-4 leads to class switching of B cells to IgE-producing cells. IgE antibody binds to Fc centers of mast cell's plasma membrane. The mast cell is now sensitized and ready to degranulate when it meets the antigen again (Berin, 2015). Activated basophils play a secondary role in releasing inflammatory mediators.

Subsequent exposures: "Pulling the trigger" Antigen binds and causes degranulation of the mast cells and basophils and discharge of multiple primary chemical mediators

- Histamine, eosinophilic-chemotactic factor of anaphylaxis, proteases

•Histamine targets H1 receptors, responsible for a majority of identifiable symptoms:

- Constriction of bronchial smooth muscle (wheezing, dyspnea)
- Increased vascular permeability (edema)
- Vasodilation (flushing)

• Production of secondary mediators-

- Arachidonic acid: leukotrienes, prostaglandins
- Platelet activating factor

(Simmons, 2014)

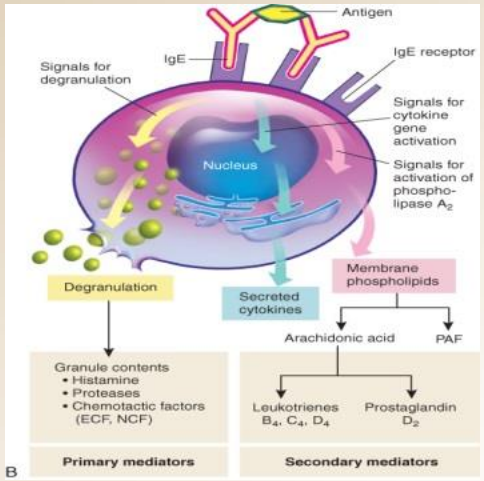


Figure 2 Type 1 Hypersensitivity McCance & Huether, 2014

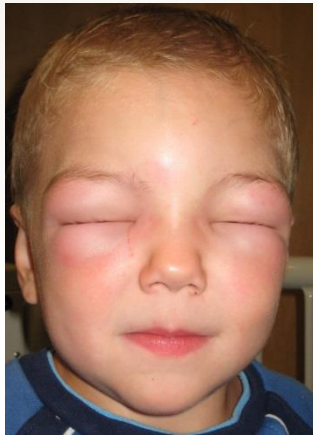


Figure 3 Image of anaphylaxis www.wikipedia.com

Figure 4 EpiPen twinpacks www.EpiPen.com

Significance of Pathophysiology

Diagnosis:

•First step: Accurate clinical history (food consumed up to 3 hours prior to any suspected anaphylactic event and details of presenting symptoms)- MOST important piece of evidence, testing most effective at predicting future anaphylaxis when it correlates with history.

•Physical examination: high co-occurrence of food allergy with atopic dermatitis, allergic rhinitis, and asthma, so a focus on skin, nasal mucosa, and lungs can provide clues that may point to an increased likelihood of food allergy.

•A note on efficacy of testing: many more patients will be sensitized to foods than will experience reactions with exposure (Stukus & Mikhail, 2016, p.34).

•If indicated based on history/exam:

oFood specific IgE lab testing- important ADJUNCT tool, should be based on history of potential offending foods, serum IgE panels (formerly RAST) can lead to misinterpretation, improper diagnosis and unnecessary dietary elimination. The normal "ranges" provided by the laboratory are inaccurate and should be ignored.

oSkin prick testing (SPT)- reliable at any age, causes local mediator release that results in pruritus and a wheal/flare. Wheals measuring greater than 3mm considered clinically relevant, but not diagnostic alone.

oOral food challenge (OFC) - if testing/history are inconclusive, an oral food challenge conducted in a physician's office with proper resuscitation equipment and medications to manage reactions can be a safe and effective food allergy diagnosis tool. OFC consists of increasing amounts of food over a short period, followed by an open feeding and 2-3 hours of observation for a reaction.
(Stukus & Mikhail, 2016)

Treatment

Counseling patients and their family on avoidance measures for their specific allergen, proper EpiPen (epinephrine autoinjector) use and storage, and what to do in the event of anaphylaxis (provided within an easy to follow Food Allergy Action Plan) are current treatment guidelines for food allergies (Stukus & Mikhail, 2016).

Implications for nursing care

Education is the first line of defense that patients and their families have against exposure and potential anaphylactic reaction to their food allergens. The education should include:

•Day to day management teaching- constant vigilance required

- Reading food labels (including the potential different names/versions of their allergen)
- Notifying restaurants/others preparing food of their food allergy and ask about potential cross contamination
- Maintaining good nutrition while avoiding allergen- nutritional counseling may be required (Simmons, 2014)

•Anaphylaxis teaching: Review patient's individualized Food Allergy Action Plan (provide copies for all caregivers if patient is a minor) and sign and symptoms that would require EpiPen vs. when antihistamines would be sufficient

•EpiPen education:

- Demonstration by RN and then return demonstration by patient or family with EpiPen tester
- Ensure patient/family can verbalize: symptoms of anaphylaxis, follow up care required (calling 911 after any EpiPen use), and importance of keeping both EpiPens in provided twinpack together in the event of user error or the need for a second dose

Conclusion

Understanding the pathophysiology behind IgE-mediated food allergies and how and why the symptoms of anaphylaxis present themselves is essential to consider during the medical history and exam portion of a patient with a possible food allergy. Ordering testing and referral to an allergist for food allergies should be based on recognizing the difference between IgE-mediated food allergy and non-IgE food allergy/intolerance. Proper diagnosis of a food allergy can avoid unnecessary dietary elimination and significant lifestyle changes that can affect quality of life for the patient and family. Once the diagnosis is made, providers should ensure patients comprehend appropriate avoidance measures and EpiPen use by allowing them to verbalize and physically demonstrate what they have learned.

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