2016

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Recommended Citation
Malinky, Melissa M., "Understanding IgE-Mediated Food Allergies" (2016). Master of Science in Nursing (MSN) Student Scholarship. 163.
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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 the 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 and how it differs from non-IgE mediated food allergies can help guide diagnostic tool, treatment, and education for referral to an allergist for more specific testing.

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 (Simmons, 2014)”

• Production of secondary chemical mediators by activated mast cells and basophils and discharge of multiple primary chemical mediators

Subsequent exposure: “Firing the gun”

Activated basophils play a significant role in the pathogenesis of anaphylaxis, and this results in the release of mediators such as histamine, leukotrienes, and proteases, which are responsible for the symptoms of anaphylaxis. Histamine causes vasodilation and increased vascular permeability, which leads to edema and hives. The leukotrienes can cause bronchoconstriction and cause the patient to wheeze. Proteases can cause swelling and itching. Activated mast cells and basophils can also release other mediators that contribute to the symptoms of anaphylaxis, such as tryptase, chymase, and mast cell proteases.

Signs and Symptoms

IgE-mediated food allergy reaction process: 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/effort
- GI: angioedema, nausea, vomiting, diarrhea, constipation
- Cardiac: tachycardia, hypotension, arrhythmia

General: sense of impending doom

Non-IgE Mediated

Non-IgE mediated 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)

Significance of Pathophysiology

Diagnosis:

• Skin test: 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 when considering an IgE-mediated reaction at predicting future anaphylaxis when it correlates with history.

• Oral challenge: high co-occurrence of food allergy with atopic dermatitis, allergic rhinitis, and asthma, or a focus on skin, nasal, mucous, and lungs can provide clues that may point to an IgE-mediated food allergy.

• In vitro screening: many more patients will be sensitized to foods than will experience reactions with exposure (Stukus & Mikhail, 2016, p.34).

• Abnormal: if indicated based on history/exam of specific IgE test, important. ADVANTAGE 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.

• Skin prick testing (SPT): reliable at any age, most common food-mediated reaction that results in pruritis and a wheal/flare. Wheels measuring greater than 3mm considered clinically relevant, but not diagnostic alone.

• Oral food challenge (OFC): if testing/history are inconclusive, an oral food challenge conducted in a physician’s office with appropriate equipment and medications to manage reactions can be a safe and effective method to diagnose food allergy. 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 allergens 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-mediated food allergy/intolerance. Proper diagnosis of a food allergy can avoid potential serious and fatal anaphylactic reactions. Counseling patients on avoidance measures should be provided as early as possible in 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.

Implications for nursing care

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

1. Step-by-day meal management:
   a) Reviewing the importance of the individual Food Allergy Action Plan (FAAP) and ask about potential cross contamination.
   b) Monitoring food nutrition while avoiding allergen.

2. Education:
   a) Demonstration by RN and then return demonstration by patient or family with Epipen tester.
   b) Ensure patient/family can verbalize symptoms of anaphylaxis, follow up care (calling 911 after an Epipen use), and importance of keeping both Epipens in provided backpack together in the event of user error or need for a second dose.

Conclusion

Understanding the pathophysiology of the IgE-mediated food allergy 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-mediated food allergy/intolerance. Proper diagnosis of a food allergy can avoid potential serious and fatal anaphylactic reactions. Counseling patients on avoidance measures should be provided as early as possible in 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.

References Cited


Additional Sources