OPINION

Food allergy testing in atopic dermatitis

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ABSTRACT

Food allergy and food-related worsening of dermatitis can occur in patients with Atopic Dermatitis (AD). We reviewed the relationship of AD with food allergen hypersensitivity and the risks and benefits of food allergen testing and avoidance in patients with AD. Skin prick testing and specific immunoglobulin E to aeroallergens may identify patients with immediate hypersensitivity. Atopy patch tests may detect non–immunoglobulin E–mediated reactions but are not standardized or routinely used. Younger children with more severe AD in whom the optimal management failed may have food-triggered AD. Egg, milk, and peanut account for most food allergens. Elimination of relevant food allergens should improve AD but must be guided by appropriate allergy testing and establishing clinical relevance. Serum immunoglobulin E panels for food allergens are discouraged in the primary care setting because of their difficulty of interpretation. Empiric avoidance of foods is entirely discouraged in AD because of their risk of causing nutritional issues, food allergy, and other problems.

Key Words: *Food allergy; Atopic dermatitis; Immediate hypersensitivity; Immunoglobulin E; Food allergens`*

INTRODUCTION

The role of food sensitization in Atopic Dermatitis (AD) has been debated throughout the years. Food allergy (FA) has been reported in up to one-third of patients with moderate-to-severe AD2 and is associated with morbidity and even mortality. It is important to identify and eliminate exposure to clinically relevant food allergens. However, unnecessary allergen avoidance can have harmful consequences, including the loss of oral tolerance to foods, nutritional deficiencies, increased costs, and inconvenience. Clinicians who treat patients with AD should be aware of the evidence-based FA testing guidelines and best practices. AD is a risk factor for immediate, Immunoglobulin E (IgE)–mediated FA. The Canadian Healthy Infant Longitudinal Development birth cohort study found that sensitization and AD at 1 year of age were strong risk factors for FA at the age of 3 years. Neonatal skin barrier dysfunction, even when transient, was associated with FA at the age of 2 years. A recent systematic review supports that early-onset AD is particularly associated with the development of FA. A large, population-based study (HealthNuts) highlighted these findings, as 1 in 5 Australian infants with AD had FA compared with 1 in 20 without AD. Patients with AD were 6 times and 11 times more likely to have egg and peanut allergies, respectively. Among children with moderate-to-severe AD, FA confirmed by double-blind, placebo-controlled, food challenges or open food challenges was identified in 33% to 81%. Numerous population-based studies have found associations between AD and food allergen sensitization (ie, the presence of food-specific IgE). Food sensitization in infants with AD is up to 6 times higher than that in healthy controls at the age of 3 months. Moreover, up to 53% of children with AD have positive food-specific immunoglobulin E (sIgE) and/or Skin Prick Tests (SPTs) with up to 15% demonstrating signs of FA on an oral food challenge, compared with 0.1% to 6% FA prevalence in the general population. The effect of AD on acquiring a natural tolerance (or outgrowing FA) is not clearly understood. In some studies, AD was associated with a persistent egg allergy. AD severity was associated with prolonged timing until the resolution of a diagnosed milk allergy. In contrast, another study found that AD did not affect the natural history of cow's milk allergy. .A study of 400 children with AD from the Mechanisms of Progression from Atopic Dermatitis to Asthma in Children cohort found that the most common SPT^+ allergens were egg white, followed by peanut, egg yolk, dog, trees, cat, ragweed, cockroach, mold, dust mite, grass, milk, weeds, soy, and wheat. Children with AD and sensitization to peanut, egg, cat, and dog had a greater baseline skin barrier dysfunction (increased transepidermal water loss and decreased skin FLG expression) in nonlesional skin and AD severity than those with allergy to other

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allergens or no allergen sensitization. Peanut and egg sensitization may occur through skin exposure in children. Individuals with AD are more likely to develop peanut sensitizations through skin exposure. Moreover, children with severe AD show a dose-dependent relationship between household peanut allergen levels and peanut sensitization, even without enteral peanut exposure. Less is known about cutaneous sensitization to egg in childhood AD. Cutaneous exposure to food via a defective skin barrier is an important route of sensitization to food allergens. The epidermis provides an essential barrier to the external environment, preventing water loss and intrusion of infectious agents and allergens. A leaky skin barrier may promote allergic sensitization by facilitating allergen uptake. When allergens are captured and processed by epidermal Langerhans cells, they migrate to draining lymph nodes and can interact with naïve T cells to promote helper T cell type 2 (Th2) immunity leading to allergies. Absorption of allergen through the disrupted skin barrier of AD is believed to lead to a Th2 response, IgE class switching, and clinical Food allergens penetrating the stratum corneum are taken up by local dendritic (Langerhans) cells, which migrate to local lymph nodes and induce Th2 immune response. Murine studies showed that innate immune cells (eosinophils and basophils) accumulate in the skin in response to exaggerated thymic stromal lymphopoietin production after cutaneous application of food allergens. Interleukin 4 produced by basophils and eosinophils promotes dendritic cell activation and presentation of food antigens to naïve T cells, leading to Th2 polarization and intestinal IgE-mediated FA in mice. Further, mice sensitized epicutaneously to food allergens display intestinal mast cell expansion and subsequent anaphylaxis upon exposure. In clinical studies, epicutaneous sensitization occurred after the application of peanut oil on eczematous skin and peanut sensitization is associated with environmental exposure to peanuts. In addition, exposure to hydrolyzed wheat protein in facial soaps was suggested as a risk factor for a special phenotype of wheat allergy. In contrast, oral ingestion of allergens generally promotes tolerance. Genetic factors underlying skin barrier disruption are associated with FA. Filaggrin loss-of-function mutation is associated with peanut sensitization in children with AD via environmental exposure to house dust peanut protein. Skin microbiome dysbiosis may also contribute to food sensitization in patients with AD through skin barrier disruption. *Staphylococcal* superantigens (eg, enterotoxin B) enhance epicutaneous sensitization to peanut in mice. In addition, FA occurs more commonly in children with AD and colonization with *Staphylococcus aureus*. Most foods reported to trigger AD are common food allergens, in general. In infants, eggs were most commonly reported, followed by cow's milk, peanuts, and soy. In children, eggs, cow's milk, and peanuts were again most common, followed by soy, wheat, tree nuts, fish, and shellfish. In older children and adults, peanuts were most common, followed by tree nuts, fish, and shellfish. It is important to distinguish between immediate hypersensitivity reactions that may lead to anaphylaxis and be life threatening from delayed reactions that may exacerbate eczema. Additionally, reactions can be mixed, with some immediate symptoms and then eczema exacerbation. Several studies linked FA and AD, especially in children with refractory, moderate-to-severe AD that has failed standard management. One study found that one-third of children with refractory, moderate-to severe AD (judged by the Scoring of Atopic Dermatitis index) had sIgE antibodies and IgE-mediated clinical reactivity to milk, egg, wheat, soy, peanut, and/or fishproteins.Immediate reactions were not distinguished from isolated eczematous reactions in this study. This study and others led a task force on FA in children with AD commissioned by the European Academy of Allergy and Clinical Immunology to recommend that children aged ≤ 6 years with mild AD (respond easily to treatment, Scoring of Atopic Dermatitis index ≤ 25, Eczema Area and Severity Index ≤ 7, and Patient-Oriented Eczema Measure ≤ 8) with a history of immediate reactions to food or moderate-to-severe AD (more difficult to treat, Scoring of Atopic Dermatitis index>25, Eczema Area and Severity Index>7, and Patient-Oriented Eczema Measure>8) with persistent AD or poor response to treatment be evaluated for FA.40 Food allergens to be tested must be carefully selected according to the medical and dietary history of the child, including possible allergenic triggers and foods introduced into the diet. Evaluation for sensitization to egg, wheat, milk, peanut, and soy in children aged<5 years with moderate-to-severe AD should be considered in the following situations: after ingestion of a specific food, there is a reliable history of an immediate reaction; and there is persistent AD despite topical therapy and optimized management. Only relevant allergens should be tested because IgE testing has low specificity, especially for food. Clinicians should be aware of the iatrogenic harm of elimination diets based exclusively on blood or skin IgE testing due to the high false-positive rate for these tests. Testing should aim to confirm the clinical relevance of suspected allergens, especially with an equivocal history, and to facilitate safe dietary expansion wherever possible. Tests include both SPT and sIgE, with or without component-resolved diagnostics. Although oral food challenge is the gold standard for diagnosis, it is likely unattainable to most patients who have FA. If patients consume specific foods in their diet without adverse reactions, then allergy to that food is unlikely.