

Food Allergy in Atopic Dermatitis

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Abstract

Food allergy in atopic dermatitis (AD) is debatable from decades. Role of diet in the cause and treatment of AD is controversial and is not well-defined. Allergists and pediatricians are convinced about the food allergy in AD whereas many dermatologists are contrary for this. However, there are studies in the Indian and western literature supporting the evidence that elimination diet may improve the severe type of AD. There is increasing awareness and lot of misconception among caregivers about food allergy and hence careful understanding about this concept is necessary to counsel parents. Recent evidence-based literature suggests avoidance of proven food allergens in AD could be beneficial in moderate to severe type of AD.

Keywords: *Atopic dermatitis, dietary elimination, food allergy, hypersensitivity reaction*

Introduction

What was known?

Food allergy can exacerbate atopic dermatitis and hence routine diet elimination would decrease the severity of AD.

Atopic dermatitis (AD) is a chronic, relapsing, inflammatory skin disease characterized by pruritus and eczematous skin lesions. It is often associated with increased serum IgE levels and personal and family history of “atopic diathesis” which includes type 1 allergies, allergic rhinitis, and asthma.[1,2,3] The pathogenesis of AD is complex with multifactorial etiology involving genetic, immunological, and environmental factors leading to disrupted skin barrier and immune system.[4] Environmental factors include microbes, irritants, and extremes of temperature, psychological stress, and food allergens.[5,6] There are many studies demonstrating the role of food allergens in triggering or exacerbating in small set of AD patients. Around 60% of children develop AD by 1st year of life with food allergies developing early in this group.

Food Allergy and Atopic Dermatitis

Clinical studies have documented the prevalence of food allergy in AD from 20% to 80%.[7] Common food allergens triggering AD are milk and milk products, peanuts, eggs, soy, wheat, seafood, and shellfish.[8] Many studies have documented that food allergy plays an important role in exacerbating severe form of AD and diet elimination will decrease the severity. An open pilot study conducted by Dhar

and Banerjee on the effects of dietary elimination in AD in 100 Indian children showed statistically significant decrease in the severity score after dietary elimination alone. A group of 100 children without systemic disease and not on systemic steroids were assessed for severity of AD by SCORAD index. They were advised to strictly avoid diet containing milk and milk products, nuts, nut containing foods, egg, sea fish and prawns, brinjal, and soybean, for 3 weeks. To maintain proper nutrition, they were asked to take lots of dal, rohu fish, chicken, and fruits. After 3 weeks, the severity of AD was measured and showed significant improvement.[9]

Werfel *et al.*, compiled the results of eight studies and found a prevalence of food allergy proven by double-blind placebo-controlled food challenge (DBPCFC) to be 33%–63%.[10] In a study of Japanese nursery school children, increased serum IgE levels, maternal history of atopy, and food allergy was linked to developing AD in children <6 months of age.[11] On the contrary, there are few studies with no temporal association of food allergy and AD. Guillet, *et al.* in a study of cohort of 250 children found a direct correlation between food allergy and increased severity of AD.[12] Few studies have shown that patients suffering from food allergy have been significantly associated with allergic rhinitis, bronchial asthma, and persistent eczematous reactions.[13]

Pathophysiology of Aggravation of Atopic Dermatitis with Food

Raw food ingestion and effects of foodborne microbes on the intestinal flora may affect the development of food allergy in AD. Skin barrier plays an important role as it prevents entry of pathogens and allergens. Filaggrin gene defect in AD increases the risk of AD. In AD, there is an epidermal barrier dysfunction leading to entry of food allergens and subsequent percutaneous and mucosal sensitization.[14] The pathogenesis of AD associated food allergy is complex. Due to the break in the skin, Langerhans cell gets exposed to allergens, which trigger an immediate or delayed type of reaction causing inflammation.[15]

Exacerbation of AD by food occurs by increased binding of antigen to immature gut microvillus, increased intestinal permeability that initiates immune responses, with primarily altered antigen transfer. The gut bacterial pathogen acts as infectious agent and superantigen thus exacerbating AD by food.[16,17] Through sensitization, atopic march can result in food allergies, environmental allergies, asthma, and eosinophilic esophagitis. Immediate types of allergic reactions are IgE mediated type III, activating the complement system whereas delayed reactions are mediated by T-cells and activated eosinophils.[17]

Clinical Features

Food allergy in AD may result in IgE-mediated immediate and non-IgE mediated late eczematous reactions. 40%–60% constitute IgE-mediated food allergy. Food allergy can manifest as flares, hives, pruritus, and other cutaneous symptoms in the absence of AD flare. Reactions can occur in the skin, oropharynx, gastrointestinal, respiratory, and cardiovascular systems.[8] Immediate IgE-mediated reactions occur within few minutes to hours after ingestion of food. Cutaneous reactions include urticaria, angioedema, pruritus, erythema, morbilliform eruptions, contact urticaria, and allergic contact dermatitis. The most common reactions are acute urticaria with or without angioedema. These reactions give rise to pruritus and in return exacerbate or worsen preexisting AD. Nondermatological manifestations include vomiting, diarrhea, abdominal pain, rhinitis, asthma, and anaphylaxis.[18] In a study of 113 children with severe AD, 63 children had developed symptoms after food challenge within 2 h with a recurrence of pruritus in few after 6–8 h.[19] In a recent study of soy allergy in patients suffering from AD, early reactions was seen in 2.8% cases. One-third of patients (27.2%) were sensitized to soy without clinical symptoms.[20]

Late non-IgE mediated eczematous reactions rarely occur, and their pathogenesis is not very clear. Delayed reactions develop after 2–6 days following ingestion of allergen food. This reaction has also been described as “food responsive eczema.” Late reactions can occur as isolated phenomenon or along with immediate type reaction.[21] Only few studies have documented the late phase reactions. Rowlands *et al.* in their study, described that out of the 58 DBPCFCs only 1 had eczematous food reactions.[22] Breuer *et al.* in his study of food challenge in 106 children for cow’s milk, hen’s egg, wheat, soy; isolated eczematous reaction was seen in 6% and combined reactions in 21%.[21]

Diagnosis

Diagnosis of food allergy is not based on history or clinical examination. Parents giving a history of exacerbation of AD due to food should not be taken as a reliable indicator. There are many tests available but must be interpreted properly as there is a high rate of false-positive reaction with low predictive value. An expert panel set up for publishing guidelines for AD reviewed the literature and found that multiple studies demonstrated 50%–90% of presumed food allergy are not allergies.[23] Many patients may have sensitization but may not develop symptoms. Diagnoses of IgE-mediated reactions require sensitization and development of symptoms. In recent guidelines put forward by American Academy of Dermatology, they have recommended testing of children younger than 5 years for food allergy with intractable pruritus.[24]

Investigations

Several investigations done to confirm food allergy are increased serum IgE, skin prick test (SPT), skin application food test, radio allergen sorbent test, atopic patch test, and oral challenge test (DBPCFC). IgE-mediated immediate reactions can be evaluated by SPT and allergen-specific serum IgE tests. These tests done alone are not diagnostic as they give false positive reaction and have low predictive value. The gold standard test for confirmation of food allergy is DBPCFC.[8,25] Non-IgE mediated eczematous reactions are not assessed and documented in many studies. In a study by Breuer *et al.*, 106 children were administered with DBPCFC; 43% were immediate reactions, 45% were immediate and late eczematous reactions, and 12% were late eczematous reactions.[21] In another study of DBPCFC, 10% of positive food challenges were not associated with food-specific IgE. In the same trial, children were given exclusion diet of egg and milk and AD improved significantly.[26] In an Indian study by Dhar and Saxena, SPT positivity to common food allergens were egg white, fish, milk, brinjal, dal, groundnut, and banana.[27] Different studies have shown variation in positive predictive value for atopic patch testing, hence not routinely performed. Routine testing for food allergies is not recommended. It should be considered in young children with intractable AD or with a history of a reaction following a specific food. A diagnostic elimination diet may be helpful to improve AD.

Dietary Elimination and Risk

Dietary elimination is not recommended routinely as there is not much evidence that has demonstrated improvement in AD. Few studies have shown good improvement in AD with dietary elimination. A Cochrane systematic review based on randomized controlled trials (RCTs) have shown that eliminating egg or cow's milk did not show any benefit and do not support the concept of dietary elimination.[28] Avoidance of food with proven food allergy and confirmed by oral challenge elimination diet may be useful. In an RCT, 55 children with AD with egg allergy proven by oral challenge showed good improvement with the exclusion of egg in diet. In an open pilot Indian study of dietary elimination in 100 children, they showed significant improvement in AD.[9]

Although food elimination is beneficial in one subset of AD, it has its own risk. Effects of elimination diet include nutritional deficiency, affects growth and development of the child, causes social isolation, may cause anaphylaxis following reintroduction of restricted food, and low-quality health.[29] Food allergies decrease with age except with nuts. The patient develops immunological tolerance to restricted food when reintroduced to the diet after restriction for 6–12 months.[30] Parents should be advised to give complementary foods rich in nutrients if elimination diet is followed.

Conclusion

Food allergies are common in AD. A careful history and clinical approach are necessary to detect food allergy as food elimination may give rise to a severe nutritional deficiency in children. There is sufficient evidence in literature that food allergies exacerbate AD; however, it should be diagnosed only after proven specific allergen tests. Although dietary elimination improves AD, DBPCFC is the gold standard test to diagnose food allergies. Parents should be counseled about the risks of food elimination and educated about myths associated with food allergy.

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Conflicts of interest There are no conflicts of interest.

What is new?

There is not much evidence that food elimination routinely improves atopic dermatitis. Avoidance of food with proven food allergy can be beneficial in improving moderate to severe AD and hence food elimination is not recommended for management of AD.

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