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Tinjauan Pustaka

Current Updates of Connection Between Food Allergies and Atopic Dermatitis: a Literature Review

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Abstrak

Pendahuluan: Hubungan antara alergi makanan (food allergy/FA) dan dermatitis atopik (atopic dermatitis/AD) melibatkan interaksi dua arah dan berkaitkan dengan konsep *atopic march*. Tinjauan ini bertujuan untuk menganalisis mekanisme patologis yang menghubungkan kedua kondisi tersebut, mengevaluasi akurasi diagnosis, serta menentukan strategi penatalaksanaan dan pencegahan yang tepat.

Metode: Tinjauan pustaka komprehensif dilakukan menggunakan basis data elektronik PubMed, MEDLINE, dan Google Scholar. Sebanyak 52 referensi yang terdiri atas jurnal internasional dan pedoman klinis hingga tahun 2025 diseleksi berdasarkan kata kunci “dermatitis atopik”, “alergi makanan”, “sensitisasi”, dan “diet eliminasi”.

Pembahasan: Tinjauan ini menunjukkan bahwa gangguan lapisan pelindung kulit pada AD, seperti mutasi filaggrin, mempermudah terjadinya sensitisasi melalui kulit (epikutaneus), sehingga AD menjadi faktor risiko utama terjadinya FA yang dimediasi IgE. Sebaliknya, FA dapat memperberat gejala AD, terutama pada bayi dengan derajat penyakit sedang hingga berat. Diagnosis memerlukan konfirmasi riwayat klinis yang didukung oleh uji tusuk kulit (*Skin Prick Test*), pemeriksaan IgE spesifik, serta uji provokasi makanan oral sebagai baku emas. Dalam penatalaksanaan, diet eliminasi yang terarah dapat memperbaiki gejala pada kasus yang terkonfirmasi, namun pembatasan diet yang tidak perlu berisiko menyebabkan defisiensi nutrisi dan hilangnya toleransi imun. Strategi pencegahan yang berfokus pada perbaikan sawar kulit melalui penggunaan emolien harian menunjukkan potensi dalam menurunkan risiko sensitisasi.

Kesimpulan: Terdapat hubungan kausal resiprokal antara AD dan FA. Dermatitis atopik berperan sebagai jalur utama terjadinya sensitisasi alergi, sementara alergi makanan dapat memperberat inflamasi kulit. Penatalaksanaan yang efektif memerlukan konfirmasi diagnosis yang ketat sebelum penerapan diet eliminasi serta menekankan pentingnya pemeliharaan dini lapisan pelindung kulit untuk berpotensi menghambat progresivitas penyakit atopik.

Kata kunci: Dermatitis atopik, Alergi makanan, Sensitisasi makanan

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Current Updates of Connection Between Food Allergies and Atopic Dermatitis: a Literature Review

Abstract

***Introduction**: The relationship between food allergy (FA) and atopic dermatitis (AD) involves a complex, bidirectional interplay often associated with the "atopic march." This review aims to analyze the pathological mechanisms connecting these conditions, evaluate diagnostic accuracy, and determine appropriate management and prevention strategies.*

***Method**: A comprehensive literature review was conducted using electronic databases including PubMed, MEDLINE, and Google Scholar. A total of 52 references, comprising international journals and guidelines published up to 2025, were selected based on keywords such as "atopic dermatitis," "food allergy," "sensitization," and "elimination diet."*

***Discussion**: The review highlights that AD-associated skin barrier defects, such as filaggrin mutations, facilitate epicutaneous sensitization, making AD a major risk factor for developing IgE-mediated FA. Conversely, FA can exacerbate AD symptoms, particularly in infants with moderate-to-severe disease. Diagnosis requires confirming clinical history with Skin Prick Tests, specific IgE levels, and the gold-standard Oral Food Challenge. Regarding management, while targeted elimination diets improve symptoms in confirmed cases, unnecessary dietary restrictions may lead to nutritional deficiencies and a loss of immune tolerance. Preventive strategies focusing on skin barrier restoration via daily emollient therapy show promise in reducing sensitization risks.*

***Conclusion**: A reciprocal causal relationship exists between AD and FA. AD serves as a primary route for allergic sensitization, while FA can aggravate skin inflammation. Effective management necessitates rigorous diagnostic confirmation before implementing elimination diets and prioritizes early skin barrier maintenance to potentially disrupt the progression of atopic diseases.*

***Keywords:** Atopic dermatitis, Food allergy, Food sensitization*

1. Introduction

The term atopy (Greek: atopos = “out of place”) refers to a genetic predisposition to allergic hypersensitivity disorders, including asthma, atopic dermatitis (AD), food allergy (FA), and allergic rhinitis. FA is characterized by immune-mediated reactions to food proteins, which may occur through immunoglobulin E (IgE)-mediated or non-IgE-mediated mechanisms, with a limited number of foods accounting for most cases. Global epidemiological data reported by the

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World Allergy Organization indicate that FA affects approximately 2.5–10% of infants and preschool children, and up to 10–15% of older children and adults[1-4].

Atopic dermatitis is a chronic, relapsing inflammatory skin disease characterized by pruritic, eczematous lesions and substantial disease burden. The incidence of both AD and FA has increased globally in recent decades. AD ranks among the most prevalent dermatological conditions (2.79%) and contributes significantly to skin-related disability-adjusted life years (DALYs: 0.36%)[6,7]. Both conditions negatively affect physical, social, and psychological well-being, while FA may also lead to severe, potentially life-threatening reactions.

Importantly, the epidemiological overlap between AD and FA reflects shared and interacting pathophysiological mechanisms rather than simple coexistence. Both conditions involve dysregulated immune responses to environmental and dietary antigens, characterized by T-helper 2 (Th2)-skewed inflammation and increased IgE production. In AD, impaired skin barrier function—particularly related to filaggrin deficiency—facilitates transepidermal allergen penetration, while microbiome alterations further influence immune dysregulation[3,5]. These processes contribute to abnormal sensitization pathways, including epicutaneous sensitization, which has been increasingly implicated in the development of FA.

This mechanistic interplay is clinically illustrated by the concept of the atopic march, in which approximately 60% of infants with AD subsequently develop FA, asthma, or allergic rhinitis[8-11]. While elimination diets may improve AD symptoms in patients with confirmed FA, emerging evidence suggests that AD itself may contribute to the development of FA through impaired immune tolerance and barrier dysfunction.

Therefore, this literature review aims to examine the bidirectional relationship between AD and FA, focusing on their shared and distinct pathophysiological mechanisms, as well as the potential impact of FA management on AD progression and, conversely, the role of AD in predisposing individuals to food allergy.

2. METHODS

This literature review was compiled from a total of 52 references, consisting of 50 international journal articles and 1 textbook. Relevant articles were identified through a comprehensive literature search conducted using electronic databases, including PubMed, MEDLINE, Google Scholar, and Google.

The search strategy incorporated the following keywords and their combinations: “atopic dermatitis,” “food allergy,” “sensitization,” “epicutaneous,” “gut tolerance,” and “elimination diet.” Additional searches were conducted using related terms such as “food sensitization,” “food elimination,” “relationship between food allergy and atopic dermatitis,” and “prevention of food allergy in atopic dermatitis.”

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Studies eligible for inclusion were primary research articles, review articles, and clinical guidelines published in peer-reviewed journals, with a particular emphasis on recent publications up to 2025. Articles were included if they addressed at least one of the following topics: (1) definitions and mechanisms of food sensitization, (2) pathophysiological links between food allergy and atopic dermatitis, (3) risk factors contributing to the development of food allergy in patients with atopic dermatitis, or (4) preventive or management strategies, including dietary interventions.

Exclusion criteria comprised non-peer-reviewed articles, conference abstracts without full text, non-English publications, duplicate studies, and articles not directly relevant to the relationship between food allergy and atopic dermatitis. Studies focusing exclusively on unrelated dermatological or allergic conditions without clear linkage to the topic were also excluded.

The literature selection process was conducted by screening titles and abstracts for relevance, followed by full-text evaluation of potentially eligible articles. Data from the selected studies were extracted and narratively synthesized. The synthesis focused on identifying common themes, consolidating evidence on pathophysiology, and summarizing current perspectives on risk factors and preventive strategies.

As this study was designed as a narrative review rather than a systematic review, a formal risk of bias assessment was not performed. However, efforts were made to prioritize high-quality evidence, including well-designed studies, recent systematic reviews, and established clinical guidelines, to minimize potential bias and enhance the reliability of the conclusions.

3. Result and Discussion

This literature review was organized into five main subtopics. The first subtopic discussed how food allergies are initiated by food sensitization the second subtopic the relationship between food allergy and atopic dermatitis, The third subtopic explain about establish the diagnosis of AD, the fourth subtopic describe about diet elimination and the risk, and the last subtopic the strategy of prevention food allergy in patients with AD

3.1. How Food Allergies are Initiated by Food Sensitization

Food allergies are initiated through a process called sensitization, where the immune system mistakenly identifies harmless food proteins as threats, triggering IgE antibody production. This sensitization phase involves specific immunological mechanisms and routes of exposure that set the stage for allergic reactions upon subsequent exposures. Food sensitization refers to the initial immune response triggered when allergens enter the body. There are two pathways for allergic sensitization: oral allergens, which sensitize through the digestive tract, and aeroallergens, which sensitize through the respiratory tract. Initial sensitization leads to memory T cells and memory IgE production, intensified by repeated allergen exposure (secondary

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immune response). Non-allergic individuals produce specific IgG and IgA upon allergen exposure, while atopic individuals prone to IgE-related allergies process allergens through APCs and Th2 cells, inducing cytokine production (e.g., IL-4, IL-13) and IgE class switching [12].

According to the National Institute of Allergy and Infectious Diseases, food allergy, as defined as involves adverse health effects from abnormal immune responses upon repeated exposure to specific foods. It can affect the skin, digestion, respiration, cardiovascular system, and more. The World Allergy Organization notes an increasing prevalence of food allergies in developed countries, affecting 10% of preschool-age children[13].

Barriers such as skin, nasal mucosa, respiratory tract, and gastrointestinal mucosa separate the environment from internal tissues. Barrier damage, immature immune systems, and T cell dysfunction can lead to ineffective oral and immunological tolerance, resulting in IgE-mediated allergic reactions or non-IgE-mediated disorders. Oral tolerance, a physiological response to ingested antigens, develops in the digestive tract, where gut-associated lymphoid tissue helps limit inflammation caused by bacteria and food proteins[14] .

Gastrointestinal cells (microfold cells, intestinal epithelium, dendritic cells) play key roles in antigen presentation, inducing and maintaining food antigen tolerance, and processing food proteins. Dendritic cells migrate to mesenteric lymph nodes, presenting antigens via MHC class II, activating effector T cells. Antigen-specific regulatory T cells (Treg) are influenced by the local microbiome, activating immune response regulation through cytokine production. Disruption of food tolerance arises from epithelial barrier damage due to factors like pathogen-associated molecular patterns (PAMPs), leading to inflammation and the differentiation of naive T cells into Th2 cells. This process induces class switching of food antigen-specific B cells and IgE production [14].

Food allergen sensitization can occur via the digestive tract, skin, and rarely, the respiratory tract, often involving inflammation and barrier dysfunction. After sensitization, re-exposure to the food antigen can cause local or systemic manifestations. Specific IgE binds to high-affinity receptors (FcεRI) on mast cells and basophils upon re-contact, leading to degranulation and the release of chemical mediators causing local and systemic symptoms[14,15].

3.1.1. Epicutaneous Sensitization

The skin acts as a primary barrier to environmental allergens. Genetic mutations affecting skin barrier proteins, such as filaggrin (FLG gene), lead to increased permeability that lead to Skin Barrier Dysfunction. This allows food proteins to penetrate the epidermis. Food allergens that penetrate the skin are captured by Langerhans cells (a subset of dendritic cells). These cells

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migrate to regional lymph nodes and present allergen peptides to naïve T cells, skewing the immune response toward a Th2 phenotype and lead to food allergen sensitization, Sensitization via the skin often precedes the development of food allergies and other atopic diseases like atopic dermatitis, asthma and allergic rhinitis[12,16,17].

3.1.2. Oral and Gastrointestinal Sensitization

Normally, the gut immune system promotes tolerance to ingested food proteins through mechanisms involving regulatory T cells (Tregs) and anti-inflammatory cytokines (IL-10, TGF-β). This prevents unnecessary immune responses to food, but several factors such as gut inflammation, dysbiosis (microbial imbalance), infections, or genetic predisposition can disrupt oral tolerance, allowing sensitization to occur. Gut microbiota composition can also influences immune responses, certain bacteria (like higher level of Clostridium difficile and Staphylococcus aureus, or Reduced Bifidobacterium & Lactobacillus). This imbalance reduces short-chain fatty acid (SCFA) production, which normally maintains gut barrier integrity and regulates immune responses. SCFA deficiency contributes to intestinal inflammation and systemic Th2 skewing. Inflammation that occur through Th2 make a person more prone to atopic dermatitis and other atopic marh or worsen atopic dermatitis [18–20].

3.2. The Food Allergy and Atopic Dermatitis.

Atopic dermatitis (AD) represents a persistent inflammatory dermatological disorder marked by recurrent episodes of pruritic eczematous eruptions. The clinical presentation of eczema encompasses acute-phase lesions exhibiting erythematous papulovesicles frequently accompanied by exudation and crust formation, while more prolonged manifestations demonstrate xerosis, lichenification, and excoriations due to chronic scratching. Epidemiological data indicate this condition impacts approximately 2.4% of the world's population, with significant geographical variation in incidence rates. According to the 2019 Global Burden of Disease (GBD) assessment, AD affected an estimated 171.17 million people globally, accounting for roughly 7.48 million Years Lived with Disability (YLDs) due to its chronic nature and substantial impact on quality of life. [12].

Atopic dermatitis (AD) is a multifactorial disorder with a complex pathogenesis involving multiple interacting factors. Current understanding suggests the disease develops through dynamic interactions between impaired epidermal barrier function, immunological abnormalities, environmental exposures, and microbial factors. Research over recent years has identified numerous exacerbating triggers, including airborne allergens, dietary antigens, contact irritants, and microbial pathogens like Staphylococcus aureus and Malassezia furfur. The immunological pathophysiology prominently features T lymphocyte-mediated hypersensitivity responses

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that drive much of the inflammatory process in AD [21].

Skin barrier abnormalities are associated with mutations or disruptions in the expression of the filaggrin gene, which encodes a structural protein crucial for skin barrier formation. Individuals with atopic dermatitic have been shown to lack ceramides (lipid molecules) and antimicrobial peptides like cathelicidins, which act as the first line of defense against infection. These barrier defects lead to increased transepidermal water loss and enhanced penetration of allergens and microbes into the skin. The most common infectious agent involved in atopic dermatitis is Staphylococcus aureus (S. aureus), which colonizes about 90% of atopic dermatitis patients. Impaired innate immune responses also contribute to increased bacterial and viral infections in atopic dermatitis patients. These factors interact to induce a T-cell response in the skin (initially a dominant Th2 response, later shifting to a dominant Th1 response), releasing pro-inflammatory chemokines and cytokines (such as IL-4, IL-5, IL-13, and tumor necrosis factor), leading to B-cell production of immunoglobulin E (IgE) and systemic inflammation causing skin inflammation and pruritus[22].

Atopic dermatitis has been considered an early stage in the "atopic march," where there is a chronological development of allergic conditions progressing to food allergies, allergic rhinitis, and asthma. Thus, atopic dermatitis is often associated with sensitization to environmental and food allergens, as well as IgE-mediated food allergies, as demonstrated by food challenge tests in about a third of moderate to severe atopic dermatitis patients [23].

3.2.1 The Role of Food Allergy in Atopic Dermatitis Exacerbation.

Food allergies commonly occur in children with atopic dermatitis (AD), ranging from 20% to 80%. Approximately 40% to 80% of AD patients have elevated specific IgE levels to foods. The presence of specific IgE and aeroallergens is associated with early onset and more severe atopic dermatitis. Higher IgE levels and early increases are linked to the likelihood of developing more severe and persistent atopic dermatitis [24].

The first documented report of food allergy as a trigger factor for AD was by Schloss in 1915, describing eczema eruptions as a response to food, with improvement upon elimination. Burks et al (1988), in two studies involving severe AD patients referred to an allergy clinic, found that 33-38.7% of children undergoing DBPCFC (double-blind placebo-controlled food challenge) reacted to food. Sampson and Scanlon (1989), in a 4-year prospective study investigating the follow-up of 34 children using elimination diets guided by DBPCFC, discovered that 17 out of 34 children improved on the diet. As confirmed through multiple studies, cow's milk, chicken eggs, wheat, soy, and peanuts are responsible for 75% of food-related AD cases[14].

In atopic dermatitis, food factors are more likely to cause exacerbations in infants or children with moderate to severe AD compared to other

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populations. Food can trigger hypersensitivity reactions mediated by IgE or lead to advanced eczematous reactions. Food-triggered AD exacerbations occur in about one-third of infants, 5% of toddlers, and 10% of older children, but are rare in adults. When the clinical history indicates that the recurrence of AD occurs several hours to a few days after eating, reintroduction of the food can be carried out at home to confirm whether it indeed triggers a worsening of AD[25].

The atopic dermatitis (AD) due to food allergens involves multiple immunological pathways. Food antigens bind to underdeveloped intestinal microvilli, and increased intestinal permeability facilitates their uptake, triggering immune activation. Gut dysbiosis contributes to this process, as pathogenic bacteria can function as both infectious agents and superantigens, amplifying AD symptoms through dietary triggers. Circulating food antigens distribute systemically, reaching the skin where they interact with food-specific IgE antibodies bound to Fcε receptors on various immune cells, including Langerhans cells, mast cells, monocytes, basophils, and skin-infiltrating T lymphocytes. This interaction initiates a type 2 inflammatory response. Supporting evidence comes from Li et al. (2001), who demonstrated in a murine model that food-reactive T cells play a pivotal role in AD pathogenesis. In their study, C3H/HeJ mice were orally sensitized to cow's milk or peanut proteins followed by low-dose allergen exposure. An AD eruption developed in about one-third of the mice after low-level exposure to milk or peanut proteins. Histological examination of skin lesions revealed spongiosis and cellular infiltrates consisting of CD4+ lymphocytes, eosinophils, and mast cells. Expression of IL-5 and IL-13 mRNA only increased in the skin of mice with AD eruptions: [2,21].

There are three patterns of skin reactions to food allergy in AD patients. First, immediate-type reactions occur minutes to 2 hours after exposure and can involve various systems such as skin, respiration, cardiovascular, and gastrointestinal system. Skin reaction of food allergy can include urticarial, angioedema, pruritus, eritema, morbiliform eruption, and contact urticarial. Second, pruritus occurs immediately after ingesting food, leading to scratching and AD exacerbation. Third, delayed hypersensitivity reactions mediated by T cells manifest symptoms 6 to 48 hours after exposure and exacerbate AD [1,26].

Clinical studies have documented food allergy prevalence in AD ranging from 20% to 80%. Common food allergens triggering AD include cow's milk, peanuts, eggs, soy, wheat, seafood, and shellfish. Raw food consumption and the effects of inherent food microbes on gut flora can influence food allergy development in AD. Exacerbation of AD by food involves increased antigen binding to immature microvilli, increased gut permeability, and immune responses altering antigen transfer[2].

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3.2.2 The Role of Atopic Dermatitis in the Onset of Food Allergy.

Atopic dermatitis (AD) is proposed as a major risk factor for food sensitization and both IgE-mediated and non-IgE-mediated food allergies. Preventing or treating AD during infancy or childhood is hypothesized to potentially prevent the development of food allergies. This idea is reinforced by evidence suggesting that skin plays a role in sensitization pathways for both AD and food allergies. The progression from AD to other atopic diseases, including food allergies, is known as the "atopic march"[27].

The prevalence of food allergies is significantly higher in children with AD compared to healthy children. National Health Interview Survey data from the US indicates that food allergy prevalence in children with and without AD is 15.1% and 3.6% respectively. Population-based studies have shown that infants with AD have a 6-fold higher likelihood of food sensitization compared to healthy controls. Prevalence of food sensitization in AD reaches 66%, with confirmed food allergies through Oral Food Challenge (OFC) testing at 81% [24].

Several studies demonstrate a significant link between severe atopic dermatitis (AD) and increased food allergy prevalence, with estimates ranging from 33% to 39% and reaching as high as 80% in some reports far exceeding the general population's 0.1-6% prevalence. This strong association positions AD as a major risk factor for food sensitization and IgE-mediated allergies. Supporting evidence includes the Danish Allergy Research Cohort (DARC), which found that 53% of children with AD (aged 6 months to 6 years) exhibited food sensitization, with 15% progressing to clinical food allergies. Similarly, Australia's HealthNut study (n=4,453) revealed that infants with AD had a sixfold higher risk of egg allergy and an elevenfold increased risk of peanut allergy by 12 months compared to non-AD infants, further underscoring the critical relationship between AD severity and early food allergy development[28].

A recent systematic review supports the association between early-onset atopic dermatitis (AD) and the development of food allergies. In another international multisite study involving a large cohort of children, Hill et al. (2008) demonstrated that the earlier the onset of AD, the higher the frequency of elevated IgE levels associated with food allergies, particularly milk, eggs, and peanuts. In fact, high levels of specific IgE for foods were found in infants whose eczema developed in the first three months (64%), and the lowest levels were observed in infants whose eczema developed after 12 months. A similar relationship was found between the severity of eczema and the results of specific IgE tests for food allergens [29].

The dual-allergen hypothesis suggests that skin exposure to food antigens promotes allergic sensitization, while early oral exposure typically induces tolerance - particularly for peanut allergens where oral introduction fosters

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immune acceptance whereas cutaneous contact without concurrent ingestion predisposes to allergy development. This process is amplified in atopic dermatitis (AD), where skin barrier impairment correlates with peanut-specific IgE levels and facilitates allergen penetration, with epithelial cytokines TSLP and IL-33 (both elevated in AD skin) playing pivotal roles in subsequent inflammatory responses. Although direct evidence in humans is lacking, mouse models demonstrate that skin sensitization occurs when peanut protein application follows stratum corneum removal, triggering Th2 cytokine production and IgE responses, while AD-like skin stripping induces IL-33-dependent gastrointestinal mast cell activation - revealing critical gut-skin interactions. Additional factors compromising cutaneous immune tolerance include filaggrin mutations and Staphylococcus enterotoxin B exposure, which provoke innate inflammatory cascades leading to sensitization. Supporting evidence from primate studies shows transcutaneous peanut exposure elevates peanut-specific IgG (though not IgE) in African green monkeys, collectively confirming the transcutaneous sensitization pathway with IL-33 as a central mediator in this immunological process[14].

Epidermal barrier dysfunction and transepidermal water loss (TEWL) serve as the foundation in the pathophysiology of atopic dermatitis preceding food allergy development. In a study using the Isle of Wight cohort (2014), loss-of-function mutations in filaggrin were linked to food sensitization in early years and later food allergies during childhood, underscoring the crucial role of skin barrier function in the development and persistence of food allergies. Skin barrier defects contribute to dehydration, persistent inflammation, and other clinical symptoms of atopic dermatitis. Environmental allergens, including food allergens, can breach the compromised skin barrier, potentially increasing food sensitization. Following penetration of food allergens, antigen-presenting cells (dendritic cells and Langerhans cells) expressing high-affinity IgE receptors bind invasive antigens that migrate to lymph nodes. Specific peptide epitopes (sometimes carbohydrate epitopes from allergen molecules) are further processed and presented to naïve CD4+ T cells. Within the lymph nodes, the presence of IL-4 and type 2 cytokines derived from endothelial cells or "alarmins" (mainly IL-33 and TSLP) leads to Th2 polarization and the development of specific CD4+ T cells producing substantial amounts of IL-4 and IL-13. Subsequently, class switching of IgE occurs, driving the production of specific IgE allergen (sIgEs) by mature plasma cells. Specific IgE binds high-affinity FcεRI receptors on the surfaces of mast cells and basophils, leading to food allergy upon subsequent allergen contact[28].

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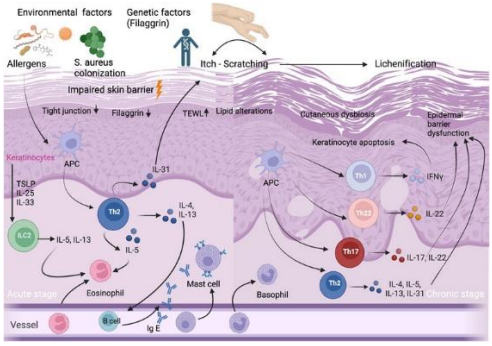

Figure 1. Penetration of food allergens through compromised skin barrier[30]. A damaged skin barrier can cause food allergens to easily penetrate into the skin, resulting in sensitization and triggering food allergies.

The skin microbiome dysbiosis in atopic dermatitis (AD) and food allergy (FA) involves significant microbial community alterations that impair skin barrier function and promote allergic sensitization. Research reveals children with IgE-mediated FA exhibit greater skin bacterial diversity than healthy controls, with Collinsella abundance correlating with elevated IgE levels and polyallergy. The skin's microbial ecosystem (comprising bacteria, fungi, viruses, and archaea) shows distinct fluctuations in AD/FA patients, particularly during early development, driven by genetic, immunological, and environmental factors interacting with barrier defects. Notably, up to 90% of AD patients demonstrate S. aureus colonization - substantially higher than healthy individuals - with specific microbial and stratum corneum abnormalities distinguishing AD+FA from AD-only cases. Leung et al. (2020) identified AD+FA as a unique endotype, showing these patients exhibit: (1) increased transepidermal water loss correlating with S. aureus on non-lesional skin, (2) significantly reduced filaggrin metabolites versus AD-only cases, and (3) heightened susceptibility to S. aureus superantigens that trigger polyclonal T-cell activation and IgE-mediated inflammation. These findings underscore how skin dysbiosis and barrier defects synergistically promote Th2-skewed inflammation and epicutaneous sensitization in allergic disease development[21,28,31–33].

Several studies support the association between transepidermal water loss (TEWL) and early onset of atopic manifestations (FA) and dermatitis atopic. Research indicates that an increase in TEWL two days after birth serves as a predictor for allergy development by the age of two. Dysfunctional neonatal skin barrier predicts FA at age 2, supporting the concept of transcutaneous allergen sensitization, even in infants without dermatitis. A study investigated the relationship between skin barrier function and FA in children. TEWL was measured during the neonatal period, at 2 months, and 6 months of life. Two-year-old children with food sensitivities (SPT+) underwent Oral Food Challenge (OFC). A total of 1,355 children were evaluated at the age of 2, with 1,260 undergoing skin prick tests and OFC screening. The results showed a prevalence of 6.27% for food sensitization and 4.45% for FA[34].

Early oral exposure to food allergens leads to antigen recognition by antigen-presenting cells (APC) in the

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intestinal mucosa, inducing differentiation of naïve CD4+ T cells within the mesenteric lymph nodes into gut-associated 4β7+ T cells. These cells stimulate the production of Foxp3+ regulatory T cells (Treg) and cytokines IL10 and TGF-b, which exert immunoregulatory effects by promoting IgG4 antibody production and inhibiting Th2-dependent allergic responses. Consequently, early epicutaneous sensitization due to compromised skin integrity and concurrent loss of oral tolerance can lead to clinical food sensitization and allergies[28].

3.3. Diagnosis of Food Alergy in Atopic Dermatitis

Establishing diagnosis of atopic dermatitis can be made from history from patien then confirmed by clinical manifestation and diagnostic test. At history taken, examiner should ask about history of food and drug that patient consume and any symptoms after consume it, or history of patient family [26].

Recent paper on food allergy by the International Collaboration in Asthma, Allergy and Immunology, allergy testing should be considered in children: (1) AD with a history of an immediate allergic reaction to one or several foods; (2) moderate to severe AD under five years of age unresponsive to topical and other skin care treatments; (3) There are foods indicated by the patient or parents as persistent AD triggers (even without a history of direct allergic reactions)[35].

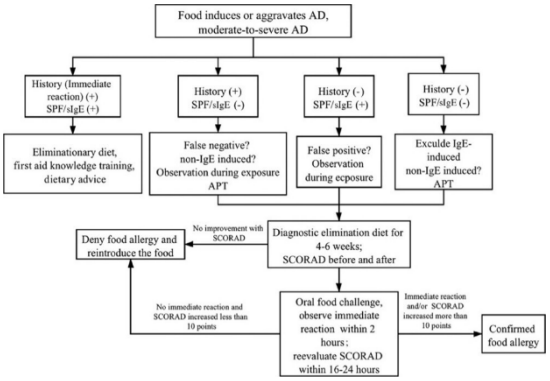

Figure 2. Flowchart of the Food Allergy Diagnosis Process in Atopic Dermatitis [26].

3.3.1 Skin Prick Test (SPT)

Allergy test that should be done is Skin Prick Test (in vivo) and measurement of specific IgE levels (in vitro). Skin Prick Test generally use for first line diagnostic instrument because it's more cheaper, minimaly invasive, results are available within 15 minutes, and allow detection of the presence of specific IgE in various foods. This test uses a drop of the allergen extract to be placed on the skin, then the skin is pricked with a standard lancet. When an allergen injected to the epidermis of the skin, sIgE bound to mast cells will cross-link resulting in mast cell degranulation and histamine release. This histamine produces urticarial responses and flares [36].

3.3.2 Specific IgE Level

Measurement of total IgE level cannot be considered as a reliable marker of allergic status because elevated IgE levels can be found in atopic conditions such as eczema as well as in non-allergic conditions. This led to the introduction of specific IgE (sIgE) antibody testing. The patient's serum is incubated with the

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relevant allergen and the sIgE from the patient will bind to the allergen. Then it will be identified with fluorescently labeled anti-IgE antibodies, so that sIgE antibodies to allergens can be measured [27].

3.3.3 Atopic Patch Test (APT)

The Atopy Patch Test (APT) serves as an important diagnostic method for identifying delayed-type (type IV) hypersensitivity reactions to food allergens in atopic dermatitis (AD) patients, particularly for detecting food allergies that may trigger or perpetuate AD symptoms. This test involves applying food allergens epicutaneously to elicit delayed eczematous reactions, providing complementary information to immediate-type tests like skin prick tests (SPT) and serum-specific IgE measurements. Research indicates APT demonstrates high specificity (>90%) and moderate sensitivity for detecting food allergies in AD patients, with optimal results achieved through prolonged allergen exposure (48-72 hours). The test shows particular diagnostic value for common allergens including cow's milk, egg, wheat, and soy, and is especially useful for identifying non-IgE-mediated food allergies (e.g., wheat allergy) that may be missed by SPT or IgE testing. While APT's high specificity makes it a reliable confirmatory tool, its variable sensitivity limits its use as a standalone test. In clinical practice, APT serves as a valuable adjunct in cases where standard IgE-mediated allergy tests yield inconclusive results despite strong clinical suspicion of food-related AD exacerbations [37–40].

3.3.4 Elimination Diet

Diagnostics by elimination diet is performed over 4 to 6 weeks and can be a practical approach to evaluate clinical relevance when a suspected food is tested positive for allergy testing.

AD diagnosis relies on observing significant improvement (e.g., >50% reduction in symptoms) during the elimination phase and relapse upon reintroduction of the suspected allergen. However, successful elimination diets alone are not completely reliable because increased AD may be due to other factors or may reflect a placebo effect, especially in older children and adults [1,41].

3.3.5 Oral Food Challenge (OFC)

The Oral Food Challenge (OFC) represents the diagnostic gold standard for both IgE-mediated and non-IgE-mediated food allergies. This procedure serves multiple critical clinical purposes: confirming or excluding food allergy diagnoses, evaluating the development of tolerance in previously allergic children, and determining individual reaction thresholds. Additional indications include assessing potential allergies to novel foods in sensitized patients who have never consumed them, as well as investigating cross-reactive foods that patients have avoided despite never being formally tested. The OFC's unique ability to objectively evaluate immediate and delayed hypersensitivity reactions makes it indispensable for accurate food

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allergy diagnosis and management. [42].

The Oral Food Challenge (OFC) utilizes three methodological approaches: open, single-blind, and double-blind placebo-controlled (DBPCFC). The open OFC is the predominant clinical method due to its efficiency, low resource burden, and procedural simplicity, though it risks false-positive results from psychological factors and occasionally requires blinded confirmation. In the single-blind OFC, clinicians administer masked allergens or placebos (concealed in neutral vehicles) while maintaining awareness of the test substance; this reduces patient psychogenic bias but retains clinician interpretation bias. The DBPCFC considered the diagnostic reference standard blinds both administrators and participants, eliminating observer and expectancy biases. Its complex implementation, however, confines primary use to research settings, with clinical applications reserved for ambiguous prior challenge results or definitive exclusion of psychosomatic influences[14].

The interpretation of the OFC test: (1) Positive, when clear objective signs of an allergic reaction occur or recur (at least three times) or multiple subjective symptoms in multiple organ systems occur; (2) Negative, if no symptoms occur; or (3) Inconclusive (or conclusive only to partial tolerance) if the test is discontinued before the total food dose is ingested[14].

3.4 Diet Elimination and The Risk

Diet elimination on atopic dermatitis patients have advantages in reducing the severity of AD and disadvantages in increasing the risk of food allergy in patients with AD

3.4.1 Beneficial Effects of Diet Elimination on AD Severity

For patients demonstrating positive Oral Food Challenge (OFC) results, dietary elimination of the identified allergen is advised as a therapeutic intervention for severe atopic dermatitis (AD) refractory to conventional treatments. This approach proves particularly beneficial when: (1) specific foods serve as persistent triggers for AD exacerbations, or (2) IgE-mediated food allergies have been definitively confirmed through diagnostic testing. The elimination diet aims to mitigate inflammatory responses and reduce clinical symptom severity in these selected cases[1].

A study using 55 children diagnosed with AD with egg allergy (as evidenced by the OFC test) experienced improvement after eliminating eggs from the diet. This is also supported by an open pilot study in India on elimination diets in 100 children which showed a significant improvement. in AD. Subsequent studies using a sample of 113 children with severe AD, children who were diagnosed with food allergies from a positive DBPCFC test were carried out by diet elimination, most of them showed significant improvement in AD complaints within 1 to 2 months [43,44].

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3.4.2 Diet Elimination Increase The Risk of Food Allergy in Patients with AD

While food elimination can provide clinical improvement in a specific AD subgroup, this intervention carries significant potential adverse effects that require careful consideration. The documented risks of elimination diets include: (1) development of nutritional deficiencies impacting pediatric growth trajectories, (2) psychosocial consequences including social isolation, (3) increased risk of anaphylaxis upon food reintroduction, and (4) overall reduction in health-related quality of life. Emerging evidence suggests a particularly concerning immunological consequence: the elimination of tolerated foods in AD patients may promote loss of immune tolerance, potentially converting previous sensitization into clinical IgE-mediated food allergy upon re-exposure. This phenomenon underscores the importance of reserving elimination diets for carefully selected cases with clear diagnostic confirmation of food-triggered AD exacerbations [1,43].

In pediatric populations, these risks are amplified by the increased nutritional requirements needed to support growth and development. Eliminating commonly allergenic foods such as cow's milk, eggs, wheat, and soy can substantially reduce the intake of essential macro- and micronutrients. Among the most critical concerns are deficiencies in calcium and vitamin D, especially in milk-restricted diets, as both are vital for bone mineralization and achieving optimal peak bone mass. Furthermore, exclusion of multiple protein sources may result in inadequate protein intake, which can impair growth and delay tissue development [45 - 46].

Micronutrient deficiencies are also commonly observed. Limited dietary variety can lead to iron deficiency, increasing the risk of anemia and negatively affecting neurodevelopment. Likewise, inadequate zinc intake may impair immune function and delay wound healing, which is particularly significant in individuals with atopic dermatitis who already exhibit compromised skin barrier integrity. Deficiencies in B-complex vitamins, such as vitamin B12, riboflavin, and folate, can further disrupt hematologic and neurologic function. In addition, insufficient intake of essential fatty acids may aggravate skin barrier dysfunction and inflammatory responses, potentially worsening the symptoms of atopic dermatitis [45,47].

3.5. Strategy of Prevention Food Allergy in Patients with AD

Strategies to prevent FA in patients with AD focus primarily on restoring and maintaining skin barrier integrity and managing early allergic sensitization. Some strategy example is include: daily emollient care, and supplementation of prebiotics, probiotics, and vitamin D.

3.5.1 Daily Emollient Care

Daily emollient therapy has been shown to prevent atopic dermatitis (AD) in high-risk newborns, as evidenced by two randomized trials involving 124 healthy infants, while also playing a crucial role in maintaining and restoring skin barrier

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function in established AD cases. Emollients, including advanced formulations like zinc lactobionate creams, work through multiple mechanisms: preserving the skin's acidic pH, reducing transepidermal water loss (TEWL), enhancing hydration, and strengthening barrier integrity, thereby decreasing skin sensitivity to irritants and allergens. These effects may help prevent food allergy development by limiting allergen penetration through the skin and subsequent sensitization. In patients with mild-to-moderate AD, regular emollient use as baseline therapy reduces corticosteroid requirements by improving barrier function while providing anti-pruritic and anti-inflammatory benefits, leading to better disease control. By maintaining skin barrier integrity and reducing inflammation, emollient therapy may indirectly lower food allergy risk by preventing the cycle of barrier disruption and cutaneous sensitization that can lead to systemic allergic responses [48–53].

3.5.2. Prebiotics, Probiotics, Vitamin D

Current evidence does not support recommending prebiotic or probiotic supplementation for preventing food allergies in children, regardless of atopic dermatitis status, due to inconsistent research findings. A recent study investigating the combination of emollients and synbiotics (probiotic-prebiotic mixtures) administered from birth to 6 months showed no preventive effect against food allergies by one year of age. Consequently, the EAACI Food Allergy and Anaphylaxis Guidelines conclude there is insufficient evidence to advocate probiotic use for food allergy prevention. Vitamin D deficiency during critical developmental periods may increase food allergy risk by promoting abnormal gut microbiota colonization, increased intestinal permeability, and subsequent excessive immune exposure to food allergens. Additionally, cutaneous sensitization to food allergens may be enhanced in vitamin D-deficient children. Therefore, vitamin D supplementation may be advisable for atopic dermatitis patients with confirmed deficiency, as it could potentially help modulate these risk factors for food sensitization[29,54].

Discussion

The reviewed literature highlights a complex, bidirectional relationship between atopic dermatitis (AD) and food allergy (FA). In clinical practice, this suggests that infants or children with early-onset, severe eczema should be evaluated for potential food allergies, while those diagnosed with FA warrant monitoring for the development of AD. Management remains controversial: although elimination diets may improve AD symptoms in selected patients with confirmed allergies, excessive or unverified dietary restriction can lead to nutritional deficiencies and may even promote the development of new allergies.

Beyond immunological effects, elimination diets pose significant nutritional and developmental risks, particularly in pediatric populations. Unnecessary or prolonged dietary restrictions increase the likelihood of deficiencies in essential nutrients such as calcium, vitamin D, protein, iron, and zinc. These nutrients are

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critical for growth, immune function, and maintaining skin integrity. Their deficiency may not only hinder physical development but also indirectly exacerbate AD by impairing the skin barrier and altering immune responses.

Consequently, current evidence supports a targeted strategy confirming food allergy through oral food challenge (OFC) prior to implementing strict elimination diets to minimize these risks. There is strong biological plausibility underlying the association: defects in the skin barrier and a Th2-skewed immune response in AD promote epicutaneous sensitization, while IgE-mediated food allergies can aggravate skin inflammation through both immediate and delayed pathways. Interventions aimed at restoring skin barrier function, such as regular emollient use, appear beneficial for AD management and may also help prevent FA, emphasizing the central role of skin health. In contrast, adjunctive approaches such as probiotics or routine vitamin D supplementation have shown inconsistent results and remain subjects of ongoing debate.

Future research should focus on well-designed longitudinal and interventional studies. These include optimizing emollient strategies (in terms of timing and formulation), establishing safe protocols for early allergenic food introduction in infants with AD, and exploring novel therapies to induce immune tolerance. Additionally, biomarkers of barrier function or sensitization risk such as transepidermal water loss (TEWL) and filaggrin gene analysis may facilitate more personalized prevention strategies. Long-term studies are also needed to determine whether early interventions in AD can modify the progression of the atopic march.

4. Conclusion

Food allergy is an abnormal or exaggerated specific immune response that occurs repeatedly when exposed to certain foods. Careful anamnesis, clinical manifestations, and confirmation with allergy tests are needed in detecting food allergies. There is a reciprocal relationship between FA and AD, where AD plays a role in the causality pathway of food allergy and AM is associated with the cause of exacerbations, early onset, and severity of AD. Several preventive interventions such as the use of emollients and Vitamin D suplementation have benefited AD and FA in several studies. Elimination diets bring improvement and benefit when certain foods are chronic triggers or in the case of confirmed IgE-mediated food allergies. Although recommended in cases of certain food allergies, this diet has several adverse effects, so careful consideration is required in applying it to patients.

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