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ATOPIC DERMATITIS: FROM PATHOGENESIS TO TARGETED THERAPEUTIC STRATEGIES

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ABSTRACT

Atopic dermatitis (AD), also referred to as atopic eczema, is a common chronic inflammatory skin disorder characterized by intense pruritus, xerosis, and recurrent eczematous lesions. It significantly affects patients' quality of life and poses a considerable clinical burden. This review aims to summarize current knowledge on the pathogenesis of AD and highlight recent advances in targeted therapeutic strategies. A narrative review of recent literature was conducted, focusing on immunological pathways and modern treatment approaches. The analysis emphasizes the central role of immune dysregulation, particularly involving type 2 helper T-cell responses, impaired skin barrier function associated with filaggrin deficiency, and genetic predisposition in disease development. Environmental factors and microbiome alterations also contribute to disease exacerbation. Recent therapeutic progress has introduced targeted treatments, including biologic agents and Janus kinase inhibitors, which have demonstrated high efficacy and safety profiles. These innovations have significantly improved disease control and patient outcomes. In conclusion, improved understanding of AD pathogenesis supports the development of personalized and more effective management strategies.

KEYWORDS

Atopic Dermatitis; Atopic Eczema; Pruritus; Therapeutics

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Introduction

Atopic dermatitis (AD) is a chronic, recurrent inflammatory skin disease that is one of the most common dermatological conditions, particularly in the pediatric population. It is estimated that the disease affects up to 20% of children and several percent of adults, and its incidence is increasing, making it a significant public health problem [1,2,3]. AD is characterized by severe itching, dry skin, and eczematous lesions with varying clinical presentations, depending on the patient's age and disease stage [4].

The pathogenesis of atopic dermatitis is complex and involves the interaction of genetic, immunological, and environmental factors. A key factor is disruption of the epidermal barrier, leading to increased transepidermal water loss and facilitated penetration of allergens and microorganisms [2]. Dysregulation of the immune response, with a predominance of Th2 responses and overproduction of proinflammatory cytokines such as interleukins IL-4 and IL-13, is also a significant factor [1,2,3]. This leads to persistent chronic skin inflammation.

AD has a significant impact on the quality of life of patients and their families. Chronic itching, sleep disturbances, and visible skin lesions can lead to psychological and social problems, especially in children and adolescents [5]. Furthermore, the disease often coexists with other atopic conditions, such as asthma or allergic rhinitis, further complicating the diagnostic and therapeutic process [1].

In recent years, there has been significant progress in understanding the pathogenic mechanisms of AD and in the development of new treatment methods, including biological therapies and targeted drugs [2,5]. Despite this, treatment of the disease remains a clinical challenge and requires an individualized approach to each patient.

The aim of this paper is to present current methods of treating atopic dermatitis, taking into account both classical therapies and modern therapeutic strategies.

Methods

Review of literature including review articles, clinical guidelines, consensus statements, and peer-reviewed medical studies on atopic dermatitis, with particular emphasis on epidemiology, pathophysiology, clinical presentation, diagnosis, and treatment.

Results

Epidemiology

Epidemiological studies indicate a global increase in the incidence of Atopic Dermatitis. It is the most common eczematous skin disease, with a lifetime prevalence estimated at approximately 15% [7,8]. The condition may occur at any age; however, the peak incidence is observed during infancy [7], typically between 3 and 6 months of life [1]. It is estimated that about 60% of children with atopic dermatitis develop symptoms within the first 12 months of life, and up to 90% before the age of 5 years [1,7].

Among adults, the prevalence is estimated at 1–3% [2,9], and fewer than 2% of newly diagnosed cases occur after the age of 20 years [6]. A higher prevalence of atopic dermatitis has been observed among women [2]. The increasing frequency of the disease has prompted greater attention from clinicians and researchers, leading to a growing number of studies aimed at improving the diagnosis and management of this condition.

Etiology

Atopic dermatitis (AD) is a disease with a complex, multifactorial etiology, involving the interaction of genetic, immunological, and environmental factors, as well as epidermal barrier dysfunction [10,11,12]. The pathogenesis of AD results from the interaction of these factors, leading to chronic skin inflammation.

Genetic Factors

Genetic predisposition plays a significant role in the development of AD. Mutations in the filaggrin gene (FLG), responsible for the proper structure of the epidermal barrier, are particularly important. Defects in filaggrin lead to increased transepidermal water loss (TEWL) and facilitated penetration of allergens and pathogens [10,13]. Furthermore, AD has been shown to occur more frequently in individuals with a history of atopy (asthma, allergic rhinitis), suggesting a hereditary nature of the disease [11].

Epidermal Barrier Disorders

Damage to the skin barrier is a key element in the etiology of atopic dermatitis. Reduced lipid (ceramide) content and abnormal organization of the stratum corneum lead to dry skin and increased permeability to irritants [10,12].

Barrier dysfunction also promotes skin colonization by microorganisms, especially Staphylococcus aureus, which further exacerbates inflammation [11].

Immunological Mechanisms

Atopic dermatitis is associated with an abnormal immune response, primarily Th2. In the acute phase of the disease, the production of cytokines such as IL-4, IL-5, and IL-13 predominates, promoting allergic responses and IgE production [12,13]. In the chronic phase, Th1 and Th17 responses are also observed, indicating the complex nature of the immune response [11].

Environmental Factors

Environmental factors play a significant role in the initiation and exacerbations of atopic dermatitis. These include airborne allergens (e.g., house dust mites), environmental pollutants, tobacco smoke, and climatic factors (low humidity, cold) [10,12]. Lifestyle factors such as diet, stress, and exposure to detergents and irritants also play a role [11].

Skin Microbiome

Increasing attention is being paid to the role of the skin microbiome in the etiology of atopic dermatitis. Microbial imbalances (dysbiosis), especially excessive Staphylococcus aureus colonization, lead to increased inflammation and a worsening of the disease [12,14].

Diagnosis

The clinical diagnosis of atopic dermatitis (AD) is established on the basis of medical history, physical examination, and the exclusion of other conditions in the differential diagnosis [5,6,16]. Laboratory tests and skin biopsy are not required to confirm the diagnosis; however, they may be useful when alternative diagnoses are being considered [5,6]. Biopsy should be considered for lesions that are atypical appearing and/or refractory to conventional therapy [16]. The diagnosis of atopic dermatitis may be challenging due to its heterogeneous clinical presentation, variable severity, and fluctuating course. The diagnostic criteria proposed by Hanifin and Rajka in 1980 (Table 1) remain the most widely used clinical criteria for AD in both children and adults. These criteria consist of four major features (i) pruritus; (ii) typical morphology and distribution of lesions; (iii) chronic or chronically relapsing dermatitis; and (iv) personal or family history of atopy, as well as 23 minor criteria. A diagnosis requires the presence of at least three major criteria and three minor criteria [1,6,8,9,16].

Clinical features

Erythema, papules, and pruritus are the earliest clinical manifestations of atopic dermatitis. Subsequent scratching and rubbing lead to secondary lesions such as erosions, excoriations, and lichenification. In patients with darker skin tones, perifollicular changes may be more pronounced [5,15], whereas erythema may appear more subtle [5]. Eczematous lesions demonstrate an age-dependent distribution [5,9,15]. In infants (≤2 years of age), lesions are typically acute and characterized by pruritic papules and vesicles (elevated lesions <1 cm), often accompanied by serous exudation and crusting. These lesions most commonly involve the face, trunk, and extensor surfaces of the limbs, and occasionally the diaper area. During childhood (≥2 years of age), AD is characterized by xerosis, less pronounced erythema, and lichenified papules and plaques involving the antecubital and popliteal fossae as well as the hands and feet. In adolescents and adults, symmetrical papules and plaques associated with lichenification and excoriations are observed. These lesions most frequently involve flexural areas, the face, neck, and the hands and feet [9,15]. In some individuals, particularly after physical exertion or a hot bath, cholinergic urticaria may occur. Cheilitis, including inflammation of the upper lip extending to the perioral area, is relatively characteristic. In adults, although rarely, involvement of the nipples and eyelids may also occur. Xerosis is a common and widespread feature in patients with AD [15]. When establishing the diagnosis, it is essential to consider other dermatological conditions that may mimic AD and to evaluate a broader differential diagnosis (Table 2) [5,6,15,16].

Table 1. Hanifin and Rajka diagnostic criteria for atopic dermatitis

AT LEAST 3 MAJOR CRITERIA:
PRURITUS
TYPICAL MORPHOLOGY AND DISTRIBUTION
CHRONIC OR CHRONICALLY RELAPSING DERMATITIS
PERSONAL OR FAMILY HISTORY OF ATOPY (ASTHMA, ALLERGIC RHINITIS, ATOPIC DERMATITIS)
AT LEAST 3 OF 23 MINOR CRITERIA:
XEROSIS
ICHTYOSIS/PALMAR HYPERLINEARITY/KERATOSIS PILARIS
IMMEDIATE (TYPE I) SKIN TEST REACTIVITY
ELEVATED SERUM IGE
EARLY AGE OF ONSET
TENDENCY TOWARD CUTANEOUS INFECTIONS/ IMPAIRED CELL MEDIATED IMMUNITY
TENDENCY TOWARD NON-SPECIFIC HAND OR FOOT DERMATITIS
NIPPLE ECZEMA
CHEILITIS
RECURRENT CONJUNCTIVITIS
DENNIE-MORGAN INFRAORBITAL FOLD
KERATOCONUS
ANTERIOR SUBCAPSULAR CATARACTS
ORBITAL DARKENING
FACIAL PALLOR/ FACIAL ERYTHEMA
PITYRIASIS ALBA
ANTERIOR NECK FOLDS
ITCH WHEN SWEATING
INTOLERANCE TO WOOL AND LIPID SOLVENTS
PERIFOLLICULAR ACCENTUATION
FOOD INTOLERANCE
COURSE INFLUENCED BY ENVIRONMENTAL/ EMOTIONAL FACTORS
WHITE DERMOGRAPHISM/ DELAYED BLANCH

IGE- IMMUNOGLOBULIN E

Comorbidities of Atopic Dermatitis

The consequences of atopic dermatitis affect not only the skin but also the entire organism. They also impact quality of life and mental health. The most common complication of atopic dermatitis is secondary infection, resulting from damage to the epidermal barrier. In cases of suspected superinfection, a skin swab should be performed to confirm the presence of infection [4]. Patients with atopic dermatitis are more frequently colonized by *Staphylococcus aureus* than healthy individuals [4, 14]. Patients with severe atopic dermatitis experience recurrent infections; therefore, it is reasonable to assume that infants with exacerbated atopic dermatitis presenting with oozing, crusted, or pustular lesions should be considered infected and treated with systemic antibiotics. If infections are strictly localized, topical agents may be sufficient [14].

Patients with atopic dermatitis, particularly those with more severe disease, require more frequent screening to detect and monitor coexisting atopic conditions. The prevalence of asthma, allergic rhinitis, and food allergies is higher in individuals with atopic dermatitis. Eosinophilic esophagitis is considered a comorbid condition associated with atopic dermatitis and atopic disease [12].

Pruritus is the most significant and debilitating symptom of atopic dermatitis [4]. Patients with atopic dermatitis experience sleep disturbances secondary to intense itching and the itch–scratch cycle [12]. Sleep disturbances affect approximately two-thirds of pediatric patients with atopic dermatitis [5]. It has also been shown that adults with atopic dermatitis have poor sleep quality, with reduced sleep duration, more frequent and prolonged awakenings, overall lower sleep efficiency, and increased daytime dysfunction [12].

Atopic dermatitis significantly affects the quality of life of patients and their families, with its chronic and unstable course manifesting neuropsychologically in several ways, including anxiety, depression, behavioral disorders, neurodevelopmental disorders, attention-deficit/hyperactivity disorder (ADHD), autism spectrum disorders, and suicidal ideation. These associations are particularly evident in individuals with severe atopic dermatitis. Several recent meta-analyses have demonstrated a positive association between atopic dermatitis and symptoms of attention-deficit/hyperactivity disorder (ADHD). It has been found that the risk of developing clinical symptoms of ADHD in a child with atopic dermatitis is 43% higher than in a child without the condition [15].

The presence of atopic dermatitis and associated pruritus is positively correlated with symptoms of depression, suicidal ideation, and/or anxiety and stress [12,15]. Chronic inflammation may lead to serious cardiovascular events (angina pectoris, acute myocardial infarction, cardiac arrhythmias), heart failure, venous thromboembolism, and lymphoproliferative malignancies in patients with atopic dermatitis [15].

Moreover, patients with atopic dermatitis have been found to have an increased body mass index and/or obesity, type 2 diabetes, ocular complications such as atopic keratoconjunctivitis and keratoconus, osteoporosis and bone fractures, dental complications, warts, and extracutaneous infections. Many of these comorbidities are associated with the severity of atopic dermatitis and poor long-term disease control.

Treatment

Treatment for atopic dermatitis (AD) is based on a multifaceted approach, encompassing topical and systemic therapy, as well as addressing triggers. Treatment selection depends on the severity of the disease and the patient's response to current therapy [10,19].

Basic Treatment

The cornerstone of therapy is the regular use of emollients, which improve epidermal barrier function and reduce transepidermal water loss (TEWL). Studies have shown that their systematic use reduces the frequency of flare-ups and the need for anti-inflammatory treatment [20].

Local Treatment

Local treatment primarily uses glucocorticosteroids, which have strong anti-inflammatory effects and are effective in controlling flare-ups. Alternatively, calcineurin inhibitors (tacrolimus, pimecrolimus) are recommended, especially for sensitive areas such as the face and neck [11,20].

Phototherapy

Phototherapy (primarily UVB 311 nm) is an effective treatment for moderate atopic dermatitis, especially in patients who do not respond adequately to topical treatments. However, its use is limited by availability and potential side effects [11].

General Treatment

In moderate to severe cases, systemic therapy is used, including immunosuppressive drugs such as cyclosporine A, methotrexate, or azathioprine. Cyclosporine is considered the first-line treatment for severe disease, but its use requires monitoring for side effects [10].

Biological Treatment

Significant progress in the treatment of atopic dermatitis has been made with the introduction of biologics. Dupilumab, a monoclonal antibody that blocks the signaling of interleukins IL-4 and IL-13, demonstrates high efficacy and a favorable safety profile in patients with moderate to severe atopic dermatitis [13,18].

New targeted therapies, including JAK kinase inhibitors, represent a promising therapeutic alternative, enabling more precise modulation of the immune response [10].

Differential diagnosis

Considering the morphological variability and the spectrum of clinical manifestations of atopic dermatitis, the differential diagnosis is broad and includes, among others, infectious, inflammatory, and neoplastic processes. In infants, atopic dermatitis requires differentiation from seborrheic dermatitis, impetigo, and scabies. Other inflammatory dermatoses that may mimic atopic dermatitis include psoriasis, dermatomyositis, irritant contact dermatitis, and allergic contact dermatitis. Molluscum contagiosum may present with lesions lacking the characteristic central umbilication, instead showing a localized inflammatory reaction that can mimic atopic dermatitis.

Certain neoplastic processes, such as mycosis fungoides, a primary cutaneous T-cell lymphoma, may present with eczematous morphology and mimic atopic dermatitis. It should be noted that severe atopic dermatitis occurring in infants under 3 months of age should alert the clinician to consider primary immunodeficiency syndromes, such as Wiskott–Aldrich syndrome, hyper-IgE syndrome, genetic disorders with impaired barrier function, and metabolic disorders [17].

Table 2. Differential diagnosis of atopic dermatitis

SEBORRHEIC DERMATITIS
SCABIES
NUMMULAR DERMATITIS
ALLERGIC CONTACT DERMATITIS
DERMATOPHYTOSIS
CUTANOUS T-CELL HYMPHOMA/SEZARY SYNDROME
IMMUNODEFICIENCY (WISKOTT-ALDRICH SYNDROME, HYPER-IGE SYNDROME))
PSORIASIS
MOLLUSCUM DERMATITIS
TINEA CORPORIS AND TINEA CAPITIS
CUTANEOUS LUPUS
ECZEMATOUS DRUG ERUPTION
DERMATOMYOSITIS
DERMATITIS HERPETIFORMIS
TRANSIENT ACANTHOLYTIC DERMATOSIS
ASTEATOTIC ECZEMA
SKIN INFECTION (IMPETIGO)
LANGERHANS CELL HISTIOCYTOSIS
ZINC DEFICIENCY

IGE- IMMUNOGLOBULIN E

Discussion

Atopic dermatitis is a complex and heterogeneous inflammatory disease resulting from the interplay between epidermal barrier dysfunction, immune dysregulation, and environmental factors [10,11,12]. Current evidence highlights the key role of filaggrin mutations and increased transepidermal water loss in initiating inflammatory processes [10,13].

From an immunological perspective, the predominance of Th2-mediated responses and cytokines such as IL-4 and IL-13 plays a central role in disease development [1,2,12]. In chronic stages, additional immune pathways, including Th1 and Th17 responses, contribute to disease persistence and variability in clinical presentation [11].

Despite significant progress in understanding the pathogenesis of AD, its management remains challenging. Conventional therapies focus on symptom control and restoration of the skin barrier, while modern treatments, including biologics and JAK inhibitors, offer promising targeted approaches [10,20]. However, long-term safety and accessibility of these therapies remain important considerations.

Conclusions

Atopic dermatitis is a common, chronic, inflammatory skin disease with a complex, multifactorial pathogenesis involving genetic, immunological, and environmental factors. Due to its increasing prevalence, especially among children, its clinical significance as well as its impact on public health are emphasized.

This disease has a significant impact on quality of life, mainly due to persistent pruritus, which may lead to sleep disturbances, but also because of the presence of comorbidities. The diagnosis of atopic dermatitis is primarily based on clinical examination, and effective treatment requires an individualized approach to the patient.

Although current treatment methods focus mainly on symptom control rather than causal therapy, recent advances-particularly in biological and targeted therapies - offer improved outcomes in patients with moderate to severe disease.

Future research should focus on personalized therapeutic approaches and the long-term safety of emerging therapies.

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