ATOPIC DERMATITIS: MODERN APPROACHES TO DIAGNOSIS AND TREATMENT — A DERMATOLOGY PERSPECTIVE

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Abstract. Atopic dermatitis (AD) is a chronic, relapsing inflammatory skin disease with substantial global disease burden and rising clinical significance in pediatric and adult dermatology practice. This narrative review synthesizes current evidence on the pathogenesis, clinical features, diagnosis, and treatment of AD, drawing on systematic reviews, randomized controlled trials, and consensus clinical guidelines, supplemented with epidemiological data and clinical guidance specific to Uzbekistan. The pathogenesis of AD reflects a sequential interplay of skin barrier disruption, Th2-dominant immune activation, and secondary microbial dysbiosis. Diagnosis relies on established clinical criteria supported by standardized severity scoring (SCORAD, EASI), while treatment follows a stepwise ladder from basic skin care and topical anti-inflammatory agents to systemic immunosuppressants, phototherapy, and, in refractory disease, targeted biologic or JAK-inhibitor therapy. Early recognition, individualized barrier-repair and anti-inflammatory strategies, and expanded access to modern targeted therapies are essential to reducing AD-related morbidity, including within the healthcare system of Uzbekistan.

Keywords

Atopic dermatitis, eczema, skin barrier dysfunction, filaggrin, Th2 inflammation, IgE, pruritus, topical corticosteroids, dupilumab, allergic march.

Methods

This work was prepared as a narrative literature review. A literature search was conducted between June and July 2026 using PubMed, the Cochrane Library, and the websites of major dermatology and allergology journals, with the search terms "atopic dermatitis," "eczema," "skin barrier," "Th2 inflammation," and "biologic therapy," combined with filters for publication in English between 2008 and 2025. Priority was given to systematic reviews, randomized controlled trials, and consensus clinical practice guidelines issued by recognized dermatology and allergology societies; original textbook chapters were included for foundational pathophysiological background. Region-specific data and guidance were additionally identified through the official resource portal of the Ministry of Health of the Republic of Uzbekistan and local peer-reviewed pediatric and dermatology journals. Case reports, non-peer-reviewed sources, and articles without accessible full text were excluded. A total of fifteen sources meeting these criteria were selected and synthesized for this review.

Introduction

Atopic dermatitis (AD) is a chronic, relapsing inflammatory skin disease characterized by intense pruritus, eczematous lesions, and a disrupted epidermal barrier [1]. It is among the most common chronic skin disorders worldwide, frequently beginning in early childhood and often preceding the development of other atopic conditions such as food allergy, allergic rhinitis, and asthma — a phenomenon known as the "atopic march" [3].

Disturbances resulting from atopic dermatitis can be broadly classified into two main categories. The first includes barrier-related manifestations, characterized by transepidermal water loss, xerosis, and increased susceptibility to irritants, allergens, and microbial colonization, particularly by Staphylococcus aureus [7]. The second includes immune-mediated manifestations, characterized by Th2-predominant cytokine signaling, elevated serum IgE levels, and chronic pruritus that perpetuates the itch-scratch cycle and worsens barrier damage [4], [11].

According to Global Burden of Disease estimates, AD ranks among the leading causes of non-fatal disease burden attributable to skin conditions worldwide, though the age-standardized disability-adjusted life-year (DALY) rate reported for Uzbekistan (85.1 per 100 000) is among the lowest globally, in contrast to considerably higher rates reported in Northern European countries [13]. National clinical guidance issued by the Ministry of Health of the Republic of Uzbekistan nonetheless identifies AD as the leading allergic skin disorder in the country, affecting an estimated 20% of children and 3–10% of adults — figures broadly consistent with international epidemiological data [14].

Modern technologies — including targeted biologic agents, Janus kinase (JAK) inhibitors, advanced emollient formulations, and digital symptom-tracking tools — are considered promising for improving disease control and for modernizing dermatological care [5], [11].

The aim of this review is to summarize current evidence on the diagnosis and management of atopic dermatitis, with particular emphasis on recent therapeutic advances and their relevance to clinical practice in Uzbekistan.

Understanding the pathophysiology of atopic dermatitis enables clinicians to prevent, diagnose, and manage flares and associated complications effectively. This knowledge is applied across several areas of medicine, including:

* Pediatric Dermatology (early diagnosis and long-term disease monitoring)
* Allergology and Immunology (identification of triggers and comorbid atopic disease)
* Primary Care (initial management and referral triage)
* Dermatologic Surgery (management of chronic lichenified or infected lesions)
* Mental Health Services (addressing sleep disturbance and psychosocial burden)
* Pharmacology (optimization of topical and systemic therapy regimens)

Pathogenesis

The pathogenesis of atopic dermatitis is multifactorial, arising from the sequential interaction of epidermal barrier dysfunction, Th2-dominant immune activation, and secondary microbial dysbiosis. Filaggrin deficiency and reduced ceramide content compromise the skin barrier, permitting increased allergen penetration [7]. Subsequent Th2 lymphocyte activation drives release of interleukin-4, interleukin-13, and interleukin-31, producing the characteristic pruritus and acute inflammatory lesions [4], [11]. Persistent scratching and reduced skin microbial diversity sustain chronic inflammation and, over time, favor a shift toward Th1/Th22-predominant signaling associated with lichenified, chronic plaques [1], [11]. The stages of this cascade are summarized in Table 1.

Table 1. Key Stages in the Pathogenesis of Atopic Dermatitis

Stage	Key Mechanism	Clinical Correlate
1. Barrier disruption	Filaggrin deficiency and reduced ceramide content increase	Xerosis, skin fragility, heightened allergen penetration

Stage	Key Mechanism	Clinical Correlate
	transepidermal water loss [7]	
2. Acute Th2-dominant inflammation	Allergen and irritant penetration triggers IL-4, IL-13, and IL-31 release [4], [11]	Erythema, weeping lesions, acute pruritus
3. Itch-scratch cycle	Scratching further damages the barrier and amplifies cytokine release [1]	Excoriation, secondary lichenification
4. Microbial dysbiosis	Reduced microbial diversity and S. aureus overgrowth on damaged skin [7]	Secondary bacterial infection, flare exacerbation
5. Chronic Th1/Th22 shift	Persistent inflammation shifts toward Th1/Th22 predominance [11]	Lichenification, chronic thickened plaques

Clinical Features

Clinical presentation varies markedly with age, and recognizing these age-specific patterns is important for accurate diagnosis. Infants typically develop facial and extensor lesions, often sparing the diaper area; older children more often present with flexural involvement of the elbows, knees, and wrists; and adults commonly show hand, eyelid, and flexural dermatitis alongside more widespread lichenification from long-standing disease [1], [3].

Table 2. Age-Related Clinical Patterns in Atopic Dermatitis

Age Group	Typical Distribution	Characteristic Features
Infant (0–2 years)	Face, scalp, and extensor surfaces of limbs	Weeping, crusted erythematous lesions; diaper area typically spared
Child (2–12 years)	Flexural creases of elbows, knees, wrists, and neck	Lichenification, excoriation, and dry, scaly plaques
Adult (>12 years)	Hands, eyelids, flexural areas, and head/neck region	Chronic lichenification, hyperlinear palms, more localized or hand-predominant disease

Additional characteristic clinical features across age groups include:

* Intense, often nocturnal pruritus that disrupts sleep quality
* Erythematous, weeping lesions during acute flares
* Frequent secondary bacterial or viral skin infections

Early recognition of these age-specific patterns supports prompt initiation of anti-inflammatory treatment and reduces the risk of chronic barrier damage and infection.

Diagnosis

Diagnosis of atopic dermatitis remains primarily clinical, based on established criteria such as the Hanifin and Rajka criteria or the UK Working Party diagnostic criteria, which

require pruritus plus a defined number of characteristic features including typical morphology and distribution, chronic or relapsing course, and personal or family history of atopy [3].

Diagnostic confirmation and severity assessment rely on:

* SCORAD (Scoring Atopic Dermatitis) index combining extent, intensity, and subjective symptoms [2]

* Eczema Area and Severity Index (EASI) for objective severity quantification [9]

* Serum total IgE and allergen-specific IgE panels when allergic triggers are suspected [4]

* Skin prick testing and patch testing for allergen identification [3]

* Skin swab culture for suspected secondary bacterial infection [7]

Because several other dermatoses can mimic AD, differential diagnosis is an important part of clinical evaluation. Seborrheic dermatitis typically presents with greasy scale in sebum-rich areas without the intense pruritus characteristic of AD; contact dermatitis is usually more sharply localized to the site of allergen or irritant exposure; psoriasis tends to show well-demarcated plaques with thicker silvery scale rather than the ill-defined, weeping lesions of AD; and scabies should be considered when pruritus is severe, widespread, and accompanied by burrows or affects close contacts, warranting microscopic confirmation when suspected [3].

Accurate clinical and scoring-based characterization is essential for classifying disease severity, guiding treatment escalation, and monitoring therapeutic response over time. In pediatric practice in Uzbekistan, dermatoscopy has additionally been studied as a complementary tool for evaluating disease severity alongside the SCORAD index [15].

Treatment

The cornerstone of atopic dermatitis management is restoration of skin barrier function through regular emollient use, combined with anti-inflammatory therapy during flares [2], [9]. Daily moisturization reduces flare frequency and forms the foundation of long-term maintenance therapy.

Key first-line therapeutic strategies include:

* Liberal, regular use of emollients regardless of disease activity

* Topical corticosteroids of appropriate potency for acute flare control [9]

* Topical calcineurin inhibitors (tacrolimus, pimecrolimus) for sensitive areas and maintenance therapy [10]

* Proactive intermittent application of anti-inflammatory agents to prevent flare recurrence [10]

In patients with moderate-to-severe disease inadequately controlled by topical therapy, systemic options become necessary. Traditional immunosuppressants such as

cyclosporine, methotrexate, and azathioprine have long been used, though their utility is limited by cumulative toxicity with prolonged use [10]. Phototherapy, particularly narrowband UVB, remains a useful adjunctive option for widespread, chronic disease not adequately controlled by topical measures alone [10].

Emerging targeted therapies, including biologic agents that inhibit Th2 cytokine signaling and oral Janus kinase (JAK) inhibitors, offer improved efficacy and more favorable safety profiles for long-term use in appropriately selected patients [5], [11].

Table 3. Stepwise (Ladder) Approach to Atopic Dermatitis Treatment

Step	Disease Severity	Recommended Therapy
Step 1	Mild / maintenance	Regular emollients, trigger avoidance, basic skin care [2]
Step 2	Mild-to-moderate flares	Topical corticosteroids or topical calcineurin inhibitors [9], [10]
Step 3	Moderate-to-severe, refractory to topical therapy	Narrowband UVB phototherapy or conventional systemic immunosuppressants (cyclosporine, methotrexate) [10]
Step 4	Severe / refractory disease	Biologic therapy (e.g., IL-4/IL-13 pathway inhibitors) or oral JAK inhibitors [5], [11]

Identification and avoidance of individual triggers — including irritants, specific allergens, climatic factors, and psychological stress — is an important adjunct to

pharmacologic therapy [3]. Coordinated screening and management of comorbid asthma, allergic rhinitis, and food allergy further supports sustained disease control [8].

Conclusion

Effective management of atopic dermatitis depends on recognizing it as a disease driven jointly by skin barrier dysfunction and immune dysregulation, rather than treating flares in isolation. AD-related complications require early diagnosis, individualized barrier-repair and anti-inflammatory strategies, and multidisciplinary management encompassing dermatology, allergology, and primary care.

The integration of modern diagnostic scoring tools, novel biologic and targeted therapeutics, and structured long-term follow-up programs — informed by both international guidelines and locally generated clinical data — holds the potential to significantly decrease disease-related morbidity and improve quality of life for patients in Uzbekistan.

Future strategies must combine early barrier-protective interventions, expanded access to targeted biologic and JAK-inhibitor therapies, and comprehensive dermatology-allergology care networks to mitigate the growing burden of atopic dermatitis-related morbidity.

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