Acad Med J 2025;5(Suppl 1):S139-S145.
UDC: 616.5-002-092-085
https://www.doi.org/10.53582/AMJ2551139dk
Case Report

ADULT-ONSET ATOPIC DERMATITIS: CLINICAL CASE ANALYSIS WITH DIAGNOSTIC AND THERAPEUTIC INSIGHTS

Dohcheva Karajovanov Ivana, Peshikj Nebojsha, Cifrevska Matevska Vesna, Duma Silvija, Breshkovska Hristina, Mirceska Arsovska Elena, Vukovska Rebeka, Nikolovska Suzana

University Clinic for Dermatology, Faculty of Medicine, Ss. Cyril and Methodius University in Skopje, Republic of North Macedonia
email: idohcheva@gmail.com

Abstract

Introduction: Adult-onset atopic dermatitis (AOAD) is a distinct and increasingly recognized condition. This case report presents a patient with severe AOAD, both highlighting diagnostic and therapeutic challenges. Case Report: A 43-year-old male with extensive eczema and elevated IgE levels was treated with systemic corticosteroids, antihistamines, emollients, and phototherapy. Significant clinical improvement was noted. Conclusion: This case illustrates the complex management of AOAD and emphasizes the need for a multidisciplinary and personalized treatment approach.

Objective: This paper discusses a case of AOAD, providing clinical and diagnostic insights while integrating evidence from current literature.

Methods: A 43-year-old male with severe AOAD was evaluated using the Hanifin-Rajka criteria and the SCORAD index for diagnosis and severity assessment. Laboratory, histopathological, and allergological investigations supported the diagnosis. Relevant literature was reviewed to contextualize findings.

Results: The patient presented with extensive xerosis, erythema, and pruritus. Elevated serum IgE and eosinophilia confirmed a heightened atopic state. Management included systemic corticosteroids, antihistamines, emollients, and UVA phototherapy, resulting in symptom improvement. The case highlights the systemic and relapsing nature of AOAD, requiring a multidisciplinary approach.

Conclusion: AOAD differs significantly from COAD in pathogenesis, clinical features, and management. This case contributes to the growing body of literature on AOAD by showcasing its unique clinical features and the complexities associated with managing this under-recognized condition. Personalized, targeted therapies addressing skin barrier dysfunction, immune dysregulation, and systemic inflammation are essential to optimize outcomes.

Keywords: adult-onset atopic dermatitis (AOAD), diagnosis, differential diagnosis, clinical severity, treatment strategies

Introduction

Atopic dermatitis (AD) is a chronic, relapsing inflammatory skin disease that typically presents in childhood but can also develop in adulthood, referred to as adult-onset atopic dermatitis (AOAD). AOAD represents a unique clinical subset characterized by atypical distribution, diagnostic challenges, and distinct comorbidity patterns. This case is presented to highlight the clinical complexity of AOAD and to contribute to the limited body of literature on its diagnostic and therapeutic management. Given its potential overlap with other dermatoses, AOAD warrants careful evaluation and individualized treatment strategies.

Case Presentation

Patient Background

The patient, a 43-year-old male, presented with a well-documented history of multiple allergic and atopic conditions, including food allergies, chronic eczema, asthma, allergic rhinitis, and allergic blepharoconjunctivitis. He had known contraindications (Cave) to acetaminophen (acetylsalicylic acid) and hexoprenaline. His chronic eczema, diagnosed in 2021, manifested as severe pruritus, widespread erythema on the face, neck, décolletage, and extremities, accompanied by marked skin desquamation and fissuring. Approximately one month prior to the onset of dermatological symptoms, the patient experienced an allergic reaction to eggs. His long-standing asthma was actively managed with salbutamol, salmeterol, and fluticasone, under consistent pulmonary follow-up. The patient undergoes regular multidisciplinary evaluations, involving dermatology, pulmonology, otolaryngology (ENT), and ophthalmology, to facilitate coordinated, comprehensive care of his complex conditions.

Clinical Findings

Dermatological Status upon Admission: On admission, the patient exhibited pronounced dermatological abnormalities indicative of chronic atopic and eczematous changes. Diffuse erythema was noted across the face, neck, and décolletage, extending to the retroauricular regions and accompanied by pityriasiform desquamation. The skin showed significant xeroderma and fissuring, suggesting pronounced dryness and barrier dysfunction. Ocular sings included conjunctivitis and a distinct "headlight sign" with marked nasal and perioral pallor. Infraorbital Dennie-Morgan folds were prominent, reflecting chronic atopic influence. The patient also displayed deep neck folds and perioral cheilitis, with erythematous scaling around the mouth. Sunken, darkened "halonated eyes" added to the characteristic atopic facies, while lateral facial folds created a facies leonina (lion-like appearance).

Further examination revealed erythematous-squamous lesions on the flexor surfaces of the elbows and knees, as well as bilaterally on the dorsal hands, areas frequently affected in chronic AD. This comprehensive profile of dermatological findings underscored the severity and extensive distribution of the patient’s dermatological condition, characteristic of chronic atopic disease with systemic involvement. The dermatological findings observed upon admission are illustrated in Figure 1.

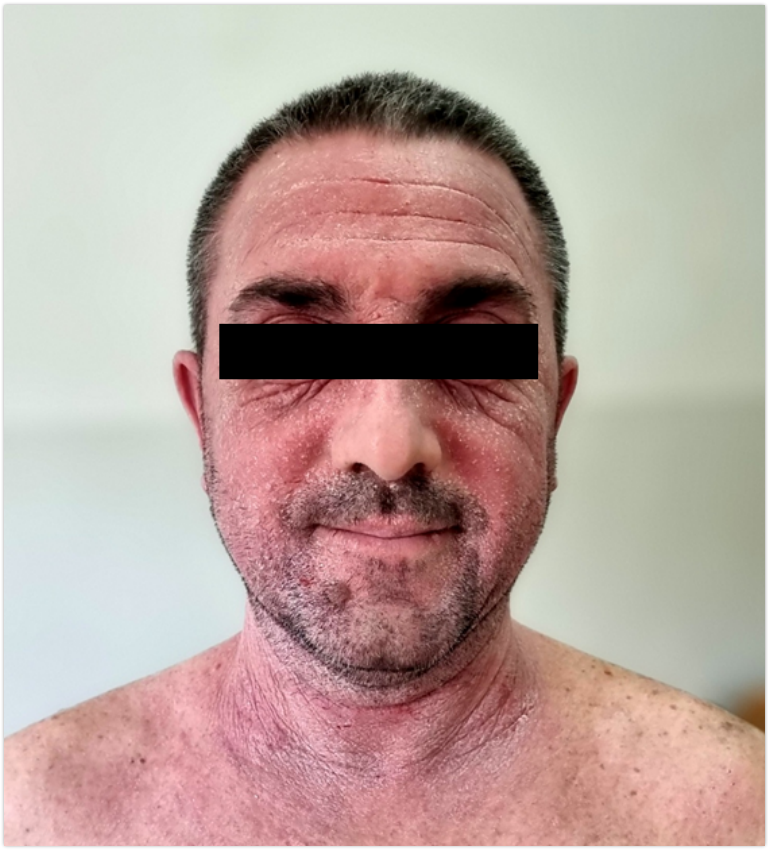

Fig. 1: Dermatological Findings upon Admission

The patient exhibited white dermographism, characterized by delayed blanching in response to cholinergic agents, a hallmark feature often associated with atopic dermatitis (Figure 2).

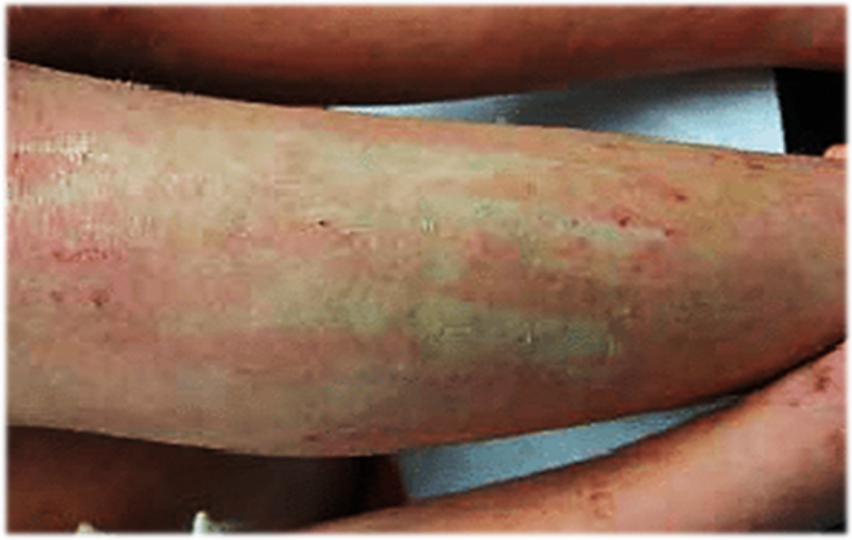

Fig. 2. White dermographism with delayed blanching, characteristic of atopic dermatitis.

Investigations conducted on our patient

A series of laboratory and diagnostic tests were performed to assess the patient's immunological and dermatological status. The complete blood count (CBC) revealed elevated eosinophil levels, consistent with an allergic etiology. The skin prick test yielded positive results, indicating sensitization to various allergens. Specifically, the patient showed reactivity to dust mites and multiple food allergens, including peanuts, egg whites, soy, fish, and milk. Sensitization was also observed for several fruits and vegetables, such as tomatoes, strawberries, pears, sour cherries, melons, and apricots, highlighting a broad spectrum of allergic triggers.

Immunological and rheumatological assessments revealed significantly elevated total IgE levels (2000 IU/mL), far exceeding normal reference values and indicating a heightened atopic state. Additionally, the rheumatoid factor (RF) was elevated (33 IU/mL), suggesting concurrent autoimmune processes or underlying inflammation, though its significance in this context required further clinical correlation.

Histopathological examination of skin biopsies demonstrated features consistent with subacute dermatitis, a finding within the eczema/dermatitis spectrum. Notably, there was no evidence of steroid-induced atrophy or dermal degeneration, underscoring the absence of corticosteroid-associated skin changes. These histopathological findings aligned with the clinical presentation and further substantiate the diagnosis within the eczema/dermatitis group.

Diagnosis and Differential Diagnosis

The diagnosis of atopic dermatitis/eczema was confirmed based on the Hanifin-Rajka (H-R) criteria, with the patient meeting all four major and 16 minor criteria.

Histopathological findings showed characteristic changes consistent with the eczema/dermatitis spectrum, reinforcing the diagnosis. The elevated total IgE levels (2000 IU/mL) and confirmed food hypersensitivity further substantiated the diagnosis, indicating a strong atopic profile.

Table 1. Differential diagnoses of Atopic dermatitis

Condition	Clinical Features	Diagnostic Tools	Key Differentiating Factors
Seborrheic Dermatitis	Greasy scales and erythema on seborrheic areas (scalp, nasolabial folds, chest); less pruritic.	Clinical examination	Not associated with elevated IgE levels.
Contact Dermatitis (Allergic/Irritant)	Erythema and eczema, particularly on face or hands.	Patch testing	Identification of allergic triggers or irritants distinguishes it from AOAD.
Psoriasis	Well-demarcated plaques with silvery scales on extensor surfaces.	Clinical examination, absence of allergen sensitization	Lack of atopic stigmata; no allergen sensitization.
Scabies (with Secondary Eczematization)	Intense pruritus with possible secondary eczematization.	Skin examination, microscopy for mites	Presence of Sarcoptes scabiei mites confirms diagnosis.
Drug-Induced Reactions	Eczema-like eruptions, often after medication use (e.g., calcium channel blockers).	Detailed medication history	Temporal relationship with drug intake; cessation of the drug improves condition.
Impetigo Contagiosa	Crusted lesions often secondary to bacterial superinfection in AD.	Bacterial cultures, clinical observation	Positive bacterial cultures; secondary to AD.

Given the variable clinical presentation, the differential diagnosis included psoriasis, seborrheic dermatitis, and drug-induced eczema [4]. The H-R criteria, recognized as the gold standard for AD diagnosis, were instrumental in establishing the diagnosis, though they have limitations when applied to adult cases [5]. Additionally, elevated serum IgE levels and a history of atopic conditions further supported the diagnosis of AOAD. The United Kingdom Working Party (UKWP) criteria were reviewed as an alternative diagnostic tool [6]; however, these criteria are primarily validated for pediatric cases, making the H-R criteria more applicable in this adult patient. The diagnostic criteria for AD, as outlined by H-R, are presented in Table 1, adapted from Fishbein et al. (2020) [7].

Scoring Severity of Atopic Dermatitis in the Patient

The severity of atopic dermatitis (AD) in the patient was assessed using the SCORing Atopic Dermatitis (SCORAD) index, a validated tool that evaluates the extent and intensity of skin lesions alongside subjective symptoms such as pruritus and sleep disturbance. This method is widely used in both clinical and research settings to quantify AD severity and monitor treatment response [8].

SCORAD Assessment Results

The SCORAD index was calculated as 75.55, indicating severe AD based on extent of lesions, intensity of clinical features, and subjective symptoms. This comprehensive severity score supported the decision to initiate systemic and phototherapy treatments.

Extent of Lesions (Area):
Head: 100%
Upper limbs (left and right): 50% each
Lower limbs (left and right): 50% each
Back: 25%
Genitals: 25%
Trunk: 25%

These values were incorporated into the "rule of nines" to determine the overall body surface area affected by lesions.

Intensity of Symptoms: The intensity of six key clinical features was assessed, each graded on a scale from 0 to 3:

Redness: 2
Swelling: 2
Oozing/Crusting: 3
Scratch Marks: 2
Skin Thickening (Lichenification): 3
Dryness: 3

Subjective Symptoms: The patient's subjective experience of symptoms was recorded, with the following scores:

Itch (Pruritus): 9
Sleep Disturbance: 5

A SCORAD score above 50 is indicative of severe AD, underscoring the extensive nature of skin involvement and the significant impact of symptoms on the patient's quality of life.

Comparative Context

In addition to the SCORAD index, other severity scoring systems such as EASI (Eczema Area and Severity Index) and IGA (Investigator's Global Assessment) are utilized in clinical practice [8]. However, SCORAD remains particularly valuable for its inclusion of subjective symptoms like pruritus and sleep disturbance, offering a holistic view of disease burden in this patient. This comprehensive assessment enabled an individualized treatment plan aimed at reducing inflammation, controlling pruritus, and improving the patient's overall well-being.

Treatment

The findings from this assessment guided the therapeutic approach, including the integration of a short course of systemic corticosteroids, antihistamines, and UVA phototherapy, along with an indicated short hospital stay to address the severity of the condition. In the absence of JAK inhibitors and biologics targeting interleukin pathways in our country, this combination of therapies was selected to manage the patient's symptoms effectively.

The treatment regimen for this patient included both systemic and topical therapies, as follows:

1. Antihistamines: Parenteral and oral antihistamines (amp. Synopen i.m. – in the evening, tbl. Loratadine p.o. – in the morning) were administered to control pruritus;
2. Corticosteroids: A short course of oral corticosteroids (tbl. Decortin 40 mg) were used during the flare-up to reduce inflammation;
3. Topical therapies: Boric acid compresses, emollients (cream Belobaza), topical antibiotic ointment (Ung. Chloramphenicol), medium potency corticosteroid ointment (Ung. Betamethasone) were applied to affected areas;
4. UVA phototherapy: Due to persistent symptoms, UVA phototherapy (0.60 J/cm^2) was employed, offering anti-inflammatory benefits for widespread involvement. This treatment approach aligns with guidelines that recommend phototherapy for moderate-to-severe cases of AD [8].

Over time, the patient experienced gradual improvement in pruritus and erythema, though the chronic-relapsing nature of AOAD required continuous management and adjustments in therapy.

Management of AOAD

The management of Adult-Onset Atopic Dermatitis (AOAD) requires an individualized approach due to its varied presentation and patient responses. First-line treatments include emollients, topical corticosteroids, and calcineurin inhibitors to control inflammation, while systemic corticosteroids and antihistamines are used for severe cases. Phototherapy is an effective adjunct for widespread involvement. For moderate-to-severe AOAD, newer therapies such as JAK inhibitors and biologics targeting interleukin pathways are promising options.

The 2023 JAAD Guidelines recommend moisturizers, topical therapies, and phosphodiesterase-4 inhibitors as standard treatments, with conditional support for wet wrap therapy while discouraging unnecessary antimicrobials or antihistamines. Given the high risk of bacterial and fungal superinfections, antimicrobial treatments such as bleach baths and antifungals may be necessary. Recent advancements include anti-IL-13 agents (tralokinumab, lebrikizumab) and JAK inhibitors (abrocitinib, baricitinib, upadacitinib), which offer targeted treatment for refractory cases, improving symptom control and patient outcomes.

Discussion

Several similar AOAD case reports in the literature describe adult patients presenting with severe xerosis, elevated IgE levels, and history of atopic conditions. Studies such as those by Bannister et al. (2000) and Son et al. (2017) report comparable clinical presentations and successful outcomes with phototherapy and immunomodulatory treatments. Comparative analysis reinforces the need for early identification, differentiation from mimickers, and targeted therapy. This case supports these findings and adds evidence from a multidisciplinary setting in AOAD care.

AOAD presents unique diagnostic and therapeutic challenges compared to childhood-onset AD. This case highlights the importance of early recognition, differential diagnosis, and personalized treatment strategies. While the Hanifin-Rajka criteria remain the gold standard, updated diagnostic frameworks for adults are necessary. Management requires a tailored approach, integrating topical and systemic therapies, phototherapy, and emerging biologics where available. Further research is essential to enhance diagnostic precision and optimize treatment outcomes.

Conclusion

AOAD differs significantly from COAD in terms of pathogenesis, clinical features, and management. This case contributes to the growing body of literature on AOAD by illustrating its unique clinical features and the complexities associated with managing this under-recognized condition. Personalized, targeted therapies addressing skin barrier dysfunction, immune dysregulation, and systemic inflammation are essential for achieving optimal patient outcomes.

Conflict of interest statement. None declared.

References:

1. Bannister MJ, Freeman S. Adult-onset atopic dermatitis. *Australas J Dermatol*. 2000;41(4):225–8. doi:10.1046/j.1440-0960.2000.00598.x
2. Maiello N, Comberiati P, Giannetti A, Ricci G, Carello R, Galli E. New directions in understanding atopic march starting from atopic dermatitis. *Children (Basel)*. 2022;9(4):450. doi:10.3390/children9040450
3. Hill DA, Spergel JM. The atopic march: Critical evidence and clinical relevance. *Ann Allergy Asthma Immunol*. 2018;120(2):131–7. doi:10.1016/j.anai.2017.06.001
4. Son JH, Chung BY, Kim HO, Park CW. Clinical features of atopic dermatitis in adults are different according to onset. *J Korean Med Sci*. 2017;32(8):1360–6. doi:10.3346/jkms.2017.32.8.1360
5. Hanifin JM, Rajka G. Diagnostic features of atopic dermatitis. *Acta Derm Venereol Suppl (Stockh)*. 1980;92:44–7.
6. Rönmark EP, Ekerljung L, Mincheva R, et al. Different risk factor patterns for adult asthma, rhinitis and eczema: results from West Sweden Asthma Study. *Clin Transl Allergy*. 2016;6:28. doi:10.1186/s13601-016-0112-0
7. Fishbein AB, Silverberg JI, Wilson EJ, Ong PY. Update on atopic dermatitis: diagnosis, severity assessment, and treatment selection. *J Allergy Clin Immunol Pract*. 2020;8(1):91–101. doi:10.1016/j.jaip.2019.06.044
8. Alakeel A, Al Sheikh A, Alraddadi AA, et al. Management of atopic dermatitis in adults in Saudi Arabia: consensus recommendations from the dermatological expert group. *Clin Cosmet Investig Dermatol*. 2022;15:1435–45. doi:10.2147/CCID.S376345