International Journal of Research in Dermatology
Odeyinka AZ et al. Int J Res Dermatol. 2025 Nov;11(6):527-529
http://www.ijord.com

DOI: https://dx.doi.org/10.18203/issn.2455-4529.IntJResDermatol20253398

Case Report

The association between atopic dermatitis and cellulitis: a case report and literature review

Abiola Z. Odeyinka*, Ali Khan

Macclesfield District General Hospital, East Cheshire NHS Trust, Macclesfield, U. K.

Received: 08 July 2025
Revised: 07 August 2025
Accepted: 12 August 2025

***Correspondence:**
Dr. Abiola Z. Odeyinka,
E-mail: drbeeorlah@gmail.com

ABSTRACT

There are numerous racial and ethnic differences in atopic dermatitis (AD) morphology, distributions, texture, and pigmentation that make diagnosing AD challenging across Fitzpatrick skin types. This incredibly complex pathophysiology involves interactions between the innate and adaptive immune system, keratinocytes and sensory nerve cells. When patients attend busy clinic setting, dermatologists frequently have insufficient time to educate patients adequately regarding the multiple factors that are important in managing AD. Management involves parental and patient education as well as pharmacological management such as topical creams, calcineurin inhibitors (cyclosporine), methotrexate, azathioprine, mycophenolate mofetil and newer targeted therapies like dupilumab and JAK inhibitors. Recurrent bacterial skin infections and lichenification are typically associated with moderate to severe AD. *Staphylococcus aureus* is a gram positive, beta-hemolytic, catalase positive, coagulase positive cocci occurring in clusters. This organism has a predilection for skin (stratum corneum), heart, and the joints. Dupilumab is a new biologic therapy approved by the food and drugs administration (FDA) for the treatment of moderate- severe AD. It blocks the IL-4 and IL-13 signaling, which are key cytokines driving inflammation in AD.

Keywords: Atopic dermatitis, Skin barrier, Cytokines, Filaggrin, Eczema, *Staphylococcus aureus*

INTRODUCTION

Atopic dermatitis (AD) is a chronic inflammatory skin disease characterized by pruritus and a chronic course of exacerbations and remissions which typically starts in childhood or infancy usually after 2 months of life. About 50% of cases of AD appear in the first year of life, the vast majority within the first 5 years of life, and the remaining cases of adult AD usually before age 30.[1] Most patients with AD describe a vicious cycle, the itch-scratch cycle, as pruritus leads to scratching, and scratching causes secondary changes that in themselves cause itching. The scratching impulse is beyond the control of the patient.[1]

Because many patients with AD suffer with chronic, itchy skin, repeated scratching that breaks the skin can cause open sores and cracks, these increase the risk of infection from *Staphylococcus aureus* bacteria. These skin infections can spread and become life-threatening. Scratching also induces lichenification and may lead to secondary infection, which was demonstrated in the case report.[1] The patient suffered adult-onset AD which impacted her quality of life psychologically causing body image concerns which made her to be socially withdrawn, her history of significant food allergies from childhood and adult-onset multiple drug allergies were potential risk factors. This case highlights the importance of identifying the causal relationship between AD and cellulitis, specifically pathogenic proliferation of *S. aureus* bacteria which normally resides on the epidermis of healthy skin, particularly on the surface layer known as stratum corneum.

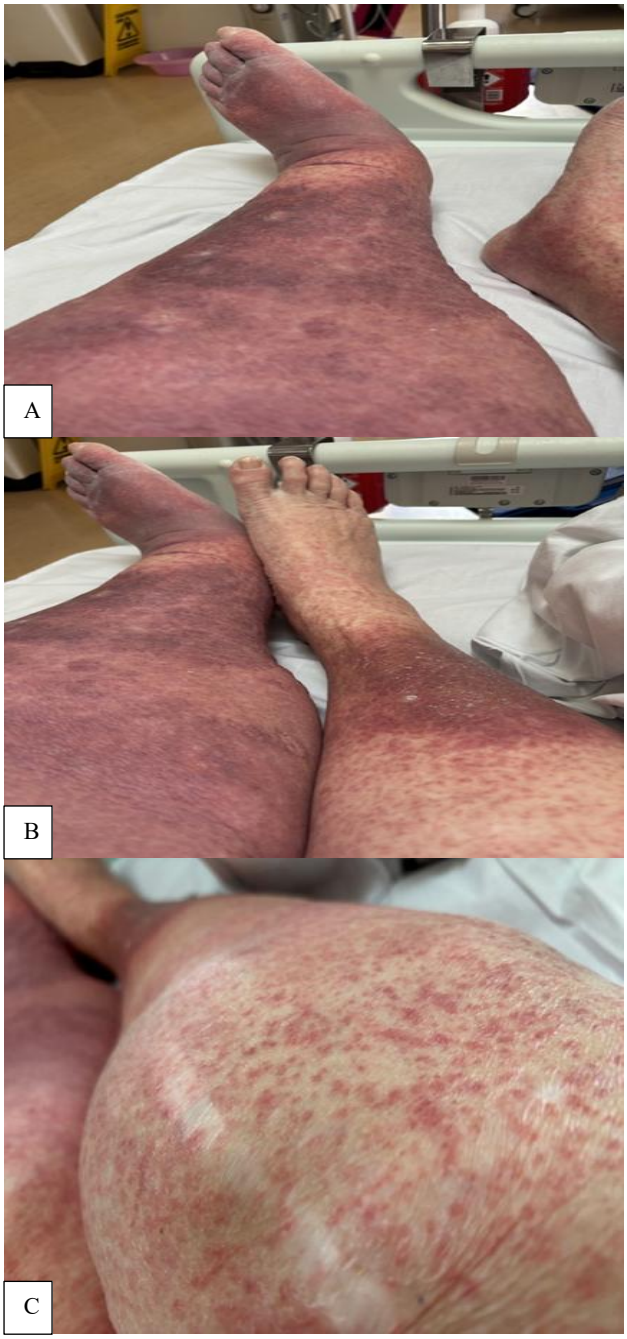

Figure 1 (A-C): Generalised macular rash with intense itching.

A 62-year-old woman arrived the emergency department, clinical examination described erythematous 'pink' swollen left lower limb (Figure 1 A) and generalised, widespread macular rash on the right leg and knee (Figure 1 B and C), trunk, upper back, neck, and upper limbs, and significant distress due to consistent itching. Her vital signs showed significant temperature spikes of 38 degrees Celsius over 48 hours despite broad-spectrum antibiotic medication. Further test investigations revealed raised inflammatory marker, neutrophils and normal serum tryptase level was 6.8 (reference range 1-14 micrograms per litre).

The patient's medical history included multiple food and drug allergies, AD with chronic itch, asthma, type 2 diabetes mellitus, diabetes retinopathy, recurrent cellulitis, paroxysmal atrial fibrillation, left lower leg lymphedema, previous ST elevation myocardial infarction (STEMI) and CKD. The general medicine team sought advice with consultant microbiologist on antibiotics alternatives for a patient with significant allergy to macrolides; erythromycin, azithromycin, clindamycin and penicillin. She was administered daptomycin intravenously 800 mg once daily for seven days to which she responded well to the relief of the team and the infection (white cell count) markers improved significantly. After discharge back to the primary care physician, with advice to book a consultation to see a skin specialist as she complained of 30 years history of 'skin problems' describing very, scaly and itchy skin flare-ups. The pruritus seemed to temporarily subside with Aproderm colloidal oatmeal cream.

DISCUSSION

Urticaria (hives) is a family of disorders characterised by transient wheals. It is characterised by pruritic wheals, sometimes with or without surrounding erythematous flares. Urticarial rash can be classified into acute or chronic, spontaneous or inducible.[2] Wheals are as a result of histamine release, platelet-activating factor, cytokines, tissue mast cells and circulating basophils.[2] Wheals on the skin look round, or form rings, a map-like pattern, or giant macules. It is a type I hypersensitivity reaction often a direct response from the skin's mast cells to medications like antibiotics (penicillin), NSAIDs (aspirin), and hormone therapies. Urticaria can affect any site of the body and is normally distributed widely.[2] Typically, they vanish within 48 hours and should not be misdiagnosed with AD. AD (eczema) is a chronic, inflammatory skin condition characterized by severely dry, itchy, and often red or inflamed, scaly skin affecting paediatric and adult patients. It is often characterized by dry, itchy, and inflamed skin, often with red macules or blisters.[3] Most patients with AD recount triggers such as allergens, irritants, stress, heat, and cold. The main risk factor for AD is having had eczema, allergies, hay fever or asthma in the past.[3] There is also a familial genetic predisposition for developing this skin condition.

Pathophysiology

The main connection between skin bacteria and AD is *S. aureus*, often found on the stratum corneum layer of the epidermis of healthy skin, however the dermis, subcutaneous tissue of AD patients is colonized by staph aureus.[4] Skin problems deriving from AD make it easier for Staph aureus to grow.[5] A lack of Filaggrin usually makes the skin's microbiome more susceptible to *S. aureus*.[6] This colonization prompts keratinocytes to produce proteases, further deteriorating the skin's barrier.[7] The Th2 cytokines IL-4, IL-5, and IL-13 play a pivotal role in the inflammatory process of AD and have

been linked to increased IgE response and eosinophils in AD patients. A study identified that in AD patients, IL-4 and IL-4 receptors on peripheral blood lymphocytes were aberrant, with an increased production of IL-13.[8]

A review of current drug therapy and most important clinical trials in patients suffering from AD shows that due to the chronic nature of AD in patients with Asthma, major research has been conducted in forms of various clinical trials in patients with this condition to identify and monitor clinical responsiveness to new investigational drugs. An example is INTEGUMENT-1 and INTEGUMENT-2 which were identical, parallel group, double-blind, vehicle-controlled randomized trials conducted at 65 and 88 centres, respectively, in the US, Canada, and Poland. A total of 1337 patients were randomized; 654 patients in INTEGUMENT-1 and 683 patients in INTEGUMENT-2. Patients eligible for inclusion were from age 6 years or older, had a history of AD (≥6 months for adults and ≥3 months for children and adolescents) and were in otherwise good health. At baseline, patients had to have an eczema area and severity index (EASI) score of at least 5, validated investigator global assessment for AD (vIGA-AD; score of 2 (mild) or 3 (moderate), and AD involving at least 3% body surface area (BSA) with no upper limit. Patients were randomized 2:1 to receive roflumilast cream, 0.15%, or vehicle cream once daily for 4 weeks.[9]

Roflumilast cream is a phosphodiesterase 4 (PDE4) inhibitor applied directly to the inflamed skin area.

Outcome

Primary endpoint was vIGA-AD success at week 4. worst itch numeric rating scale (WI-NRS) success at week 4 was a key secondary endpoint; roflumilast 0.15% cream=542, vehicle=271. Patients with AD treated with roflumilast cream, 0.15%, demonstrated improvement across multiple efficacy end points, including reducing pruritus within 24 hours after application, with favorable safety and tolerability.[9] vIGA-AD indicates validated investigator global assessment for AD vIGA-AD and WI-NRS are global measures.

Limitations

Limitations of the trials included their short duration, minimum age limit of 6 years, and lack of an active comparator. Extensive research in more European and Asian countries should also be instituted.

CONCLUSION

AD (eczema) involves complex mechanisms, notably Th2 cytokines (IL-4, IL-5, IL-13), which contribute to elevated IgE levels and increased eosinophils. Patients with multiple drug allergies and a history of atopy such as asthma, allergic rhinitis, or AD should consult both allergy/immunology specialists and dermatologists. Most cases benefit from a multidisciplinary management approach. Patients with AD need proper education in outpatient clinics on susceptibility to recurrent bacterial skin infections due to their damaged skin barrier. Topical agents such as roflumilast 0.15% cream are new treatments formulated for the management of moderate-severe AD. Systemic monoclonal antibody dupilumab is administered as a subcutaneous injection, however, socioeconomic factors may affect wider access to this medication due to its high cost.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

REFERENCES

1. James WD, Elston D, Treat JR, Rosenbach MA: Andrew's Diseases of the Skin: Clinical Dermatology, 13th ed. Elsevier, Philadelphia, PA. 2019;86.
2. Oakley A. Urticaria-an overview. Hamilton, New Zealand. DermNet. Available at: www.DermNetnz.org. Accessed on 5 April 2025
3. AskMayoExpert. Atopic dermatitis. Mayo Clinic. 2021. Available at: https://www.mayoclinic.org/diseases-conditions/atopic-dermatitis-eczema/symptoms-causes/syc-20353273. Accessed on 5 April 2025.
4. Gong JQ, Lin L, Lin T, Hao F, Zeng FQ, Bi ZG, et al. Skin colonization by Staphylococcus aureus in patients with eczema and atopic dermatitis and relevant combined topical therapy: A double-blind multicentre randomized controlled trial. Br J Dermatol. 2006;155(4):680-7.
5. Alexander H, Paller AS, Traidl-Hoffmann C, Beck LA, De Benedetto A, Dhar S, et al. The role of bacterial skin infections in atopic dermatitis: Expert statement and review from the International Eczema Council Skin Infection Group. Br J Dermatol. 2020;182(6):1331-42.
6. Clausen ML, Agner T, Lilje B, Edslev SM, Johannesen TB, Andersen PS. Association of Disease Severity with Skin Microbiome and Filaggrin Gene Mutations in Adult Atopic Dermatitis. JAMA Dermatol. 2018;154(3):293-300.
7. Williams MR, Nakatsuji T, Sanford JA, Vrbanac AF, Gallo R.L. Staphylococcus aureus Induces Increased Serine Protease Activity in Keratinocytes. J. Investig. Dermatol. 2016;137:377-84.
8. Ezzedine K, Kechichian E. Epidemiology of atopic dermatitis. Ann Dermatol Venereol. 2017;144(5):VS4-7.
9. Simpson EL, Eichenfield LF, Alonso-Llamazares J, Zoe DD, Laura KF, Seth BF, et al. Roflumilast Cream, 0.15%, for Atopic Dermatitis in Adults and Children: INTEGUMENT-1 and INTEGUMENT-2 Randomized Clinical Trials. JAMA Dermatol. 2024;160(11):1161-70.

Cite this article as: Odeyinka A, Khan A. The association between atopic dermatitis and cellulitis: a case report and literature review. Int J Res Dermatol 2025;11:527-9.