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Year-Round Flares in Atopic Dermatitis: Mechanisms and Strategies for Sustained Control

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Abstract

Atopic dermatitis is a chronic, relapsing inflammatory disorder traditionally regarded as a seasonal disease with exacerbations occurring predominantly during colder months. However, clinical observations and emerging evidence indicate that many patients experience disease flares throughout the year, independent of seasonal variation. This persistent disease activity is driven by ongoing skin barrier dysfunction, immune dysregulation, and continuous exposure to non-seasonal triggers such as sweating, psychological stress, irritants, microbial colonization, and suboptimal treatment adherence. Furthermore, subclinical inflammation persists even during periods of apparent remission, predisposing patients to recurrent flares. Conventional reactive treatment strategies that focus solely on acute exacerbations often fail to achieve sustained disease control. In contrast, proactive and preventive approaches targeting both barrier restoration and persistent inflammation have demonstrated improved long-term outcomes. This review highlights the mechanisms underlying year-round flares in atopic dermatitis and emphasizes evidence-based strategies for sustained disease control.

Keywords: Atopic Dermatitis; Skin Barrier Dysfunction; Subclinical Inflammation; Disease Flares; Proactive Therapy; Chronic Inflammation

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Introduction

Atopic Dermatitis (AD) is a chronic inflammatory dermatosis characterized by pruritus, xerosis, and recurrent eczematous lesions. Traditionally, AD has been perceived as a seasonal condition, with exacerbations commonly attributed to cold weather and low humidity. However, accumulating clinical and epidemiological evidence suggests that a substantial proportion of patients' experience flares throughout the year, independent of climatic changes [1, 2]. This misconception of seasonality often leads to episodic and reactive treatment approaches, resulting in inadequate long-term disease control, frequent relapses, and significant impairment in quality of life.

The pathogenesis of AD involves a complex interplay between epidermal barrier dysfunction, immune dysregulation, and environmental factors [3]. Importantly, these abnormalities persist even in clinically normal-appearing skin, supporting the concept of continuous subclinical inflammation [4]. Recognizing AD as a chronic, year-round disease is essential for shifting management strategies toward sustained disease control rather than intermittent symptom relief.

A flare in atopic dermatitis is defined as an acute or subacute worsening of disease activity, characterized by increased pruritus, erythema, scaling, oozing, or lichenification. Flares may be identified clinically or based on patient-reported symptoms such as worsening itch and sleep disturbance. These episodes significantly impair quality of life and often necessitate escalation of therapy [5].

Mechanisms Underlying Year-Round Flares

The persistence of disease activity in AD can be attributed to several interrelated mechanisms. Epidermal barrier dysfunction is a central feature, driven in part by reduced filaggrin expression, altered lipid composition, and increased transepidermal water loss [6]. This compromised barrier facilitates the penetration of allergens, irritants, and microbial agents, perpetuating inflammation regardless of seasonal variation.

In parallel, immune dysregulation plays a critical role. Atopic dermatitis is characterized predominantly by type 2 helper T-cell–mediated inflammation, with cytokines such as Interleukin (IL)-4 and IL-13 contributing to both inflammation and barrier impairment [7]. Notably, even non-lesional skin in patients with AD exhibits evidence of immune activation, highlighting the presence of subclinical inflammation that predisposes to recurrent flares [4].

Alterations in the skin microbiome further contribute to disease persistence. Increased colonization with pathogenic organisms, particularly Staphylococcus aureus, and reduced microbial diversity promote inflammation and barrier disruption [8]. These microbial changes are not limited to specific seasons and may drive continuous disease activity.

In addition to intrinsic factors, multiple non-seasonal triggers contribute to exacerbations. Sweating, psychological stress, irritant exposure, friction, infections, and poor adherence to therapy can independently or synergistically precipitate flares throughout the year [9]. These factors often play a more prominent role than climatic variation, particularly in tropical and humid environments.

Limitations of Reactive Treatment

Traditional management of AD has largely focused on treating acute flares, with discontinuation of therapy once clinical improvement is achieved. While this approach may provide short-term relief, it fails to address the persistent barrier abnormalities and underlying inflammation. Consequently, subclinical disease activity remains uncontrolled, leading to frequent relapses and a cyclical pattern of exacerbation and remission [10].

Strategies for Sustained Disease Control

Long-term management of AD requires a proactive and preventive approach. Restoration of the skin barrier through regular use of emollients forms the cornerstone of therapy. Emollients improve hydration, reduce transepidermal water loss, and enhance resistance to external irritants [11].

Proactive anti-inflammatory therapy, involving intermittent use of topical corticosteroids or calcineurin inhibitors on previously affected areas, has been shown to reduce the frequency and severity of flares by targeting subclinical inflammation [12]. This approach represents a shift from episodic treatment to continuous disease control.

Identification and avoidance of individual trigger factors are also essential. Strategies such as minimizing irritant exposure, managing sweat and heat, addressing psychological stress, and treating infections promptly can significantly improve disease outcomes [9].

Equally important is patient education. Misconceptions regarding treatment, particularly corticosteroid phobia, often contribute to poor adherence. Educating patients about the chronic nature of AD and appropriate use of therapies is critical for achieving sustained control [13].

Recent advances in targeted therapies, including biologic agents such as Dupilumab and Tralokinumab, as well as oral Janus kinase inhibitors like Upadacitinib and Abrocitinib, have further reinforced the role of persistent immune dysregulation in AD and offer promising options for long-term management in moderate-to-severe disease [14].

Special Considerations

Disease expression varies with age, environmental factors, and lifestyle. In tropical climates, sweating, heat, and infections may play a greater role in disease exacerbation than low humidity. Children are particularly prone to frequent flares due to an immature skin barrier, whereas adults often report stress-related triggers [9].

Conclusion

Atopic dermatitis should be regarded as a chronic, year-round inflammatory condition rather than a purely seasonal disorder. Persistent barrier dysfunction, immune dysregulation, and continuous exposure to triggers contribute to recurrent flares irrespective of climatic variation. Effective long-term management requires a proactive approach that emphasizes barrier repair, sustained anti-inflammatory therapy, trigger avoidance, and patient education. Such strategies can reduce flare frequency, improve adherence, and significantly enhance quality of life.

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