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Seasonal Variation in Urticaria: The Role of Humidity, Pollen, and Environmental Modifiers

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Abstract

Urticaria is a heterogeneous dermatological disorder characterized by transient wheals, angioedema, and pruritus, affecting up to 20% of individuals at least once during their lifetime. While many cases are idiopathic, a substantial proportion of patients report seasonal variation in disease activity, suggesting a contributory role of environmental factors. This narrative review examines the influence of humidity and pollen—two key seasonal environmental modifiers—on the onset and exacerbation of urticaria. Extremes of humidity can disrupt epidermal barrier integrity or promote sweat retention, thereby facilitating mast cell activation in chronic spontaneous and cholinergic urticaria. Seasonal pollen exposure, although classically associated with respiratory allergy, may act as an immune amplifier through IgE-mediated sensitization, cross-reactivity, and heightened mast cell reactivity in predisposed individuals. Additional seasonal contributors such as temperature extremes, viral infections, air pollution, and ultraviolet radiation are also discussed. Recognizing seasonal patterns in urticaria has important clinical implications, enabling anticipatory management through environmental modification, optimization of antihistamine therapy, and control of associated atopic comorbidities. Greater awareness of these modifiers may aid in reducing disease burden and improving quality of life in affected patients.

Keywords: Urticaria; Seasonal Variation; Humidity; Pollen; Environmental Triggers

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Introduction

Urticaria is a common dermatologic condition characterized by the rapid appearance of transient wheals, angioedema, or both, typically resolving within 24 hours. It affects up to 20% of the general population at some point in their lifetime [1]. While acute urticaria is usually self-limiting, chronic urticaria—defined by the persistence of symptoms for more than six weeks—can be particularly distressing and is associated with a significant impairment in quality of life.

Chronic spontaneous urticaria (CSU), the most common subtype of chronic urticaria, occurs in the absence of an identifiable external trigger. Despite extensive evaluation, the underlying etiology remains elusive in a substantial proportion of patients, making management challenging.

In recent years, increasing clinical observations have suggested that disease activity in CSU may exhibit seasonal variation. Environmental factors such as temperature changes, humidity, and exposure to airborne allergens, particularly pollens, have been proposed as potential modulators of disease severity [2, 3]. A better understanding of these influences may help in predicting disease exacerbations and tailoring individualized management strategies.

Seasonal Patterns and Clinical Subtypes of Urticaria

Several subtypes of urticaria demonstrate reproducible seasonal variation reflecting distinct pathophysiological mechanisms. Cold urticaria predictably worsens during winter months, when exposure to low temperatures directly induces mast cell degranulation. In contrast, cholinergic urticaria is more prevalent during warmer seasons and periods of increased sweating, where heat and physical exertion serve as primary triggers [4].

Although CSU lacks a single identifiable environmental stimulus, observational studies have reported increased disease activity during spring and early summer. Seasonal immune stimulation

may lower the activation threshold of cutaneous mast cells through mechanisms such as FcεRI upregulation, cytokine-mediated priming, and neuroimmune interactions, particularly in patients with coexisting atopic disease [5].

Humidity and Urticaria: A Dual-Edged Environmental Modifier

Low Humidity and Dry Climates

Low ambient humidity, commonly encountered during winter or in arid regions, compromises epidermal barrier integrity by increasing transepidermal water loss and disrupting the stratum corneum lipid matrix. Impaired barrier function facilitates penetration of irritants and allergens, promoting nonspecific mast cell activation and histamine release. Such mechanisms are particularly relevant in CSU, where subtle barrier dysfunction may contribute to disease persistence and exacerbations [6].

High Humidity and Tropical Environments

Conversely, high humidity promotes sweat retention and eccrine duct obstruction, which are central to the pathogenesis of cholinergic urticaria. Humid conditions also favor the proliferation of indoor allergens such as molds and dust mites, which have been implicated as aggravating factors in chronic urticaria through both IgE-dependent and IgE-independent immune pathways [7, 8].

Pollen Exposure as an Immune Amplifier in Urticaria

Although urticaria is not classically regarded as an allergen-driven disorder, seasonal increases in airborne pollen frequently coincide with exacerbations, particularly in patients with CSU and overlapping atopic conditions. Studies have demonstrated increased sensitization to aeroallergens among patients with chronic urticaria, suggesting that pollen exposure may amplify systemic immune activation [9].

Pollen-induced Th2 skewing, release of pro-inflammatory mediators, and cross-reactivity with plant-derived food allergens may further lower the threshold for mast cell activation in predisposed individuals. These mechanisms support the concept of pollen acting as an immune amplifier rather than a direct trigger of urticaria [10].

Other Seasonal Environmental Contributors

Seasonal viral infections, particularly during winter months, are well-recognized triggers of acute urticaria, especially in children, through immune activation and mast cell sensitization [11]. In addition, exposure to air pollution has been associated with worsening CSU, potentially mediated by oxidative stress and cutaneous inflammatory pathways [12].

Clinical Implications and Management Strategies

Recognition of seasonal modifiers in urticaria enables anticipatory and preventive management. Maintenance of epidermal barrier integrity through regular emollient use is particularly important during low-humidity seasons, while adequate ventilation and sweat-reducing strategies may benefit patients in humid climates. Reduction of allergen exposure during high-pollen seasons may further mitigate disease activity.

Second-generation H1 antihistamines remain the cornerstone of urticaria management, with guideline-supported updosing recommended in patients with inadequate control [1, 13]. In antihistamine-refractory cases, escalation to biologic therapy such as Omalizumab is recommended, with immunosuppressive agents like Cyclosporine reserved for selected patients. Emerging therapies, including Dupilumab and Bruton's tyrosine kinase inhibitors, are being evaluated and may further expand treatment options in difficult-to-control disease. Optimal management of comorbid allergic rhinitis and asthma is also essential, as reduction of systemic inflammatory burden may contribute to improved urticaria control [14].

Gaps in Evidence and Future Directions

Current evidence linking seasonal factors to urticaria is largely observational. Prospective studies correlating humidity indices, pollen counts, and urticaria activity scores are lacking. Geographic and climatic variability further limit generalizability. Future research should focus on longitudinal cohort studies and integration of environmental and clinical data to develop predictive models for seasonal urticaria exacerbations.

Conclusion

Seasonal variation in urticaria reflects complex interactions between environmental factors and cutaneous immune responses. Extremes of humidity influence barrier function and sweat-mediated pathways, while pollen exposure amplifies immune reactivity in susceptible individuals. Incorporating awareness of seasonal modifiers into routine clinical practice may improve disease control and patient quality of life.

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