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Successful treatment of airborne allergic dermatitis to sesquiterpene lactone with upadacitinib: A case report

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Abstract

This case report describes, to our knowledge, the first known successful treatment of airborne allergic contact dermatitis to sesquiterpene lactone with upadacitinib in an adult patient. Recurrent summertime photodistributed eczematous eruptions should raise suspicion for airborne allergic contact dermatitis to sesquiterpene lactone, and positive patch test results to sesquiterpene lactone and Compositae in these patients should be considered highly relevant. In addition to allergen avoidance, treatment with systemic Janus kinase inhibitors can induce allergic contact dermatitis remission. Our patient did not respond to allergen avoidance or topical corticosteroids; however, he achieved almost complete resolution of his allergic contact dermatitis with a 6-week course of upadacitinib, a Janus kinase 1 inhibitor.

Keywords

allergic contact dermatitis, airborne allergic contact dermatitis, upadacitinib, sesquiterpene lactone, Compositae

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Introduction

Airborne allergic contact dermatitis (ACD) is a type IV delayed hypersensitivity reaction via the skin to a previously sensitized antigen. It can be induced by both plant and non-plant substances. The Asteraceae or Compositae plant family is a commonly reported cause of airborne ACD, with causal plants including but not limited to wildflowers and weeds such as dandelion, herbal medicines such as chamomile, ornamental flowers such as sunflowers and chrysanthemums, and vegetables including chicory and artichokes.[1] The allergen is sesquiterpene lactone (SQL), which is found in many parts of the plant including the leaves, stems, pollen, and flowers.[1] Airborne exposure can occur indirectly via the release of SQL into the air, or directly via contact with SQL coating the pollen.[2] Ideally, patients who are thought to be sensitive to a particular member of the Asteraceae family should be patch tested to portions of the suspect plant. Clinically, it presents as an eczematous eruption on the face (commonly the upper eyelids) and neck, including the submandibular area, the "V" of the chest, forearms, and hands.

The treatment of airborne ACD is multifactorial. Conservative measures include allergen avoidance; however, this can be difficult. Increased frequency of bathing and the use of barrier creams aid in removing antigens and decrease the rate of penetration into the skin.[3] For medical treatment, topical corticosteroids can be used; however, the efficacy can be variable. For severe or recalcitrant cases, immunosuppression with oral steroids and azathioprine has been used.[3]

Upadacitinib is an oral small molecule agent that inhibits the intracellular cytoplasmic Janus kinase (JAK)/signal transduction and transcription activation pathway by selectively inhibiting JAK1.[4] The JAK pathway has downstream dampening effects on the immune system, cell proliferation, and inflammation.[5] Upadacitinib is currently FDA and Health Canada approved for moderate-to-severe atopic dermatitis in patients 12 years and older. To our knowledge, this is the first case of airborne ACD for SQL successfully treated with off-label upadacitinib.

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Figure 1. Sharply demarcated photodistributed erythematous papules and plaques with excoriations. There were vesicles on his dorsal hands.

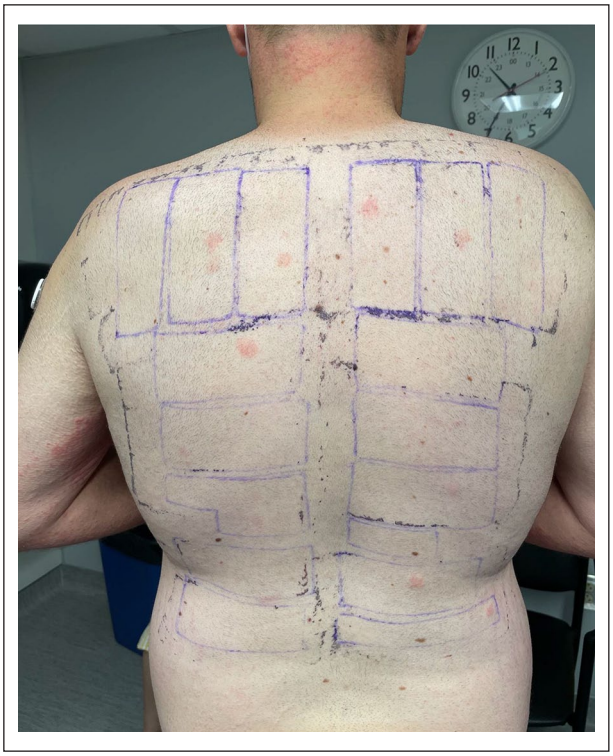

Figure 2. Patch test readings at 96 h were positive for 3+ to SQL and 3+ to compositae mix, in addition to fragrances, preservatives, and metals.

Case report

A 37-year-old male who works as a volunteer firefighter and has a history of atopic dermatitis and seasonal allergies presented with longstanding eczematous eruptions that flared during the summer months. His eruption resolved in winter. Sun-exposed sites on the face, neck, and upper and lower extremities were involved. Exposure to dandelion was noted as a trigger. Treatment with potent topical corticosteroids was ineffective. On examination, there were ill-defined erythematous papules and plaques with erosions, and excoriations over the dorsal hands, forearms, face, and "V" of the neck. There were also vesicles distributed on the dorsal hands (Figure 1). There were sharply demarcated borders at non-sun-exposed areas, including the upper arms and back.

ACD was suspected. He was patch tested to the North American Contact Dermatitis Group Standard Series, Cosmetic Series, Steroid Series, and Plant Series. Patches were read at 48 and 96 h. He had multiple positive allergens to fragrances, preservatives, and metals. His most relevant positive allergen was 3+ to SQL and 3+ to Compositae mix (Figure 2). Phototesting was normal for UVA and UVB. Photo patch testing did not reveal photoallergy.

His marked photodistribution was suspicious for airborne exposure to these plant compounds, as well as possible chronic actinic dermatitis. In addition to behavioural modifications, including allergen avoidance, protective clothing, and use of broad-spectrum sunscreen, oral upadacitinib 15 mg once daily was started. At the 6-week follow up, his skin was almost clear with a body surface area (BSA) <1. He was then transitioned to dupilumab 300 mg subcutaneous every 2 weeks given the safety profile as his past medical history was significant for hypertension, obesity, and type II diabetes. He is on dupilumab year-round given its slower kinetics to reach peak efficacy. For flares in the summer, he occasionally takes a 2- to 4-week course of upadacitinib in addition to his regular dupilumab.

Discussion

Recurrent summertime photodistributed eczematous eruptions should raise suspicion for airborne ACD to SQL. SQL is neither phototoxic nor photoallergic; however, patients allergic to SQLs can develop chronic actinic dermatitis. This is thought to be secondary to autoimmune photodermatosis mediated by sunlight-induced antigens in the skin.[6]

Baseline atopy (asthma, seasonal allergies, and atopic dermatitis) is a risk factor in the development of airborne ACD. The skin barrier dysfunction in atopic dermatitis allows increased penetration of airborne compounds.[7] Interestingly, airborne allergens can also worsen atopic dermatitis (AD) through increased proteolytic enzyme activity, activation of itch receptors and enhancement of the type I hypersensitivity response via binding immunoglobulin E antibodies.[7]

Patients with ACD to SQL should avoid direct contact with these plants, as well as foods, teas, and personal care products containing their essential oils. While allergen avoidance is the most important aspect of management, upadacitinib has been shown in our patient to be an effective treatment option. In ACD, T cells produce type 1 cytokines, including interleukin-2 (IL-2), IL-17, and interferon gamma (IFN-γ), which are cytotoxic.[8,9] Upadacitinib inhibits JAK1 which decreases the downstream effects of IL-2 and IFN-γ.[10] For flares of airborne ACD in patients with a background of AD, it may be useful to use upadacitinib in short courses to achieve disease control, then switch to dupilumab for long-term management, as it has a more favourable safety profile. This would be particularly relevant for patients with comorbidities such as dyslipidemia and hypertension that increase the risk of major adverse cardiac events or in the older adult population, especially in those over 65 years of age.

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Consent for publication

Written informed consent for patient information and images to be published.

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