

Food allergen-mediated exacerbations of oral lichen planus

H. X. Chen,¹ W. J. Yount² and D. A. Culton³

¹School of Medicine, ²Department of Allergy and Immunology, and ³Department of Dermatology, University of North Carolina at Chapel Hill, Chapel Hill, NC, USA

doi:10.1111/ced.12929

Summary

Erosive oral lichen planus (OLP) is a chronic autoimmune condition of unknown aetiology, characterized by periods of exacerbation and quiescence. Many patients with OLP report triggers of flares that overlap with triggers of other oral diseases, including oral allergy syndrome (OAS), an IgE-mediated food allergy. We report a case that, to our knowledge, is the first reported case of concurrent OLP and OAS diagnoses, which provides insight into the triggers of OLP and the role of trigger avoidance. A woman in her 60s presented with erosive OLP refractory to prednisone and azathioprine. She reported that certain food exposures triggered flares of her OLP. She was subsequently diagnosed with concurrent OAS, and avoidance of food allergens resulted in a clinically significant improvement in her OLP, eventually allowing her to taper off systemic treatment altogether. Further studies are needed to pinpoint common triggers and examine the role of trigger avoidance as a management strategy for OLP.

Oral lichen planus (OLP) is a chronic inflammatory disease of the oral mucosa that presents with white striations, white plaques, papules, erosions or blisters affecting the oral mucosa bilaterally and often symmetrically.¹ With a prevalence of approximately 1–3%, OLP is considered the most common noninfectious oral mucosal disease, particularly in women over 40 years old.² Topical corticosteroids are first-line treatment for OLP, with courses of systemic steroids or steroid-sparing immunomodulatory agents for acute or widespread exacerbations.³ Even with more aggressive systemic treatment, disease flares are common. Factors that may trigger flares have not been well studied, but many patients with OLP anecdotally report various food triggers that overlap with triggers of other oral diseases.

We describe a patient with biopsy-proven OLP who experienced acute disease exacerbations and difficulty

tapering systemic corticosteroids and steroid-sparing agents. The patient was subsequently diagnosed with concurrent OAS. After the patient's diagnosis of OAS and subsequent avoidance of food allergens, the patient's OLP improved considerably, suggesting a possible link between the two conditions and their triggers, and highlighting the potential role of trigger avoidance in the management of OLP.

Report

A 66-year-old white woman presented with a several-week history of pain and sores on her buccal mucosa and gingiva. She denied skin or genital lesions, fever, recent dental work, or starting any new medications. Her medical history was notable only for hypertension, allergic rhinitis and intermittent sinusitis. Her medications included atenolol and aspirin, as well as ibuprofen and topical triamcinolone as needed.

On physical examination, superficial erosions and white lacy striations were found to be scattered bilaterally on the buccal mucosa and erythema was observed on the lower gingiva. A 3-mm punch biopsy of the right buccal mucosa revealed a band-like infiltration of lymphocytes in the submucosa beneath an acanthotic epithelium that contained hypergranulosis, compact

Correspondence: Dr Donna A. Culton, Department of Dermatology, University of North Carolina at Chapel Hill, 401 Mary Ellen Jones Building, CB#7287, Chapel Hill, NC, 27599, USA
E-mail: donna_culton@med.unc.edu

Conflict of interest: the authors declare that they have no conflicts of interest.

Accepted for publication 23 December 2015

hyperkeratosis, vacuolar changes and necrotic keratinocytes, diagnostic of OLP.

The patient was started on prednisone 40 mg daily, with triamcinolone gel applied 2–3 times daily to the affected areas in the mouth. Following initiation of prednisone treatment, there was improvement of the patient's symptoms, but she had difficulty tapering the prednisone due to persistent flares. Once she reached a prednisone dose of 10 mg daily, she was started on azathioprine 50 mg daily as a steroid-sparing agent which was later raised to 150 mg daily. Eventually, the patient tapered off prednisone but had difficulty tapering the azathioprine below 100 mg daily. She continued to have active lesions and received intralesional triamcinolone for recalcitrant lesions.

Over the course of multiple visits, the patient consistently reported that certain foods she consumed triggered an immediate burning sensation, tingling and swelling of the oral mucosa, which ultimately resulted in flares of her OLP. She specifically reported sensitivity to strawberries, tomatoes, tree nuts, certain preservatives and soy. Referral to the Allergy Department led to skin prick testing for a panel of inhalants and foods,

which was positive for hickory pecan, oak tree pollen, birch, almond, hazelnut, soybean and lima beans.

Based on the patient's clinical history and allergy testing, she was diagnosed with OAS (2 years after her initial presentation to the dermatology department). In retrospect, the patient remembered tingling of her mouth and lips after eating various foods well before her OLP diagnosis, suggesting that her OAS preceded her OLP diagnosis. Following diagnosis of OAS, she avoided trigger food allergens and noted considerable improvement in both her OAS and erosive OLP. The patient developed no new oral lesions for several months, and was tapered off azathioprine altogether, using only topical corticosteroids as needed (Fig. 1).

To our knowledge, this is the first reported case in the literature of concurrent OLP and OAS. The patient's diagnosis of OAS, identification of triggers/food allergens and subsequent food allergen avoidance considerably improved the management of her OLP beyond the effectiveness of systemic steroids and steroid-sparing agents. Such improvement suggests a link between the two conditions and has implications for trigger avoidance in the management of OLP.

Although the exact aetiology of OLP is unknown, studies have hypothesized that the presence of keratinocyte antigens may activate CD8+ T cells to induce keratinocyte apoptosis.⁴ Several external triggers have been proposed to induce antigen expression or unmasking to elicit acute flares of oral pain associated with OLP. For example, it is believed that medications, psychological stress, infectious agents, trauma, alcohol consumption, systemic illnesses and oral habits such as lip chewing can trigger acute OLP flares.^{5,6}

OAS is the most prevalent food allergy and is characterized by type I hypersensitivity reactions with tingling, pruritus, erythema, or angio-oedema isolated to the oropharyngeal mucosa after contact with certain

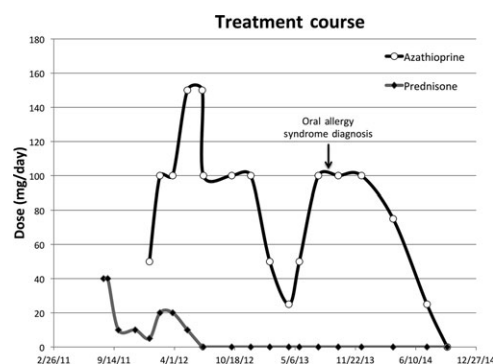


Figure 1 Timeline of the treatment course and oral allergy syndrome diagnosis.

Table 1 Common oral allergy syndrome triggers associated with birch, ragweed, and mugwort pollen sensitization.

Associated triggers			
Pollen	Fruit and nuts	Vegetables	Spices and flowers
Birch	Almond, apple, apricot, cherry,* chestnut, date palm, fig, grape, hazelnut, jackfruit, kiwi,* melon, nectarine/orange,* peach,* pear, peanut, pistachio, plum, prune, walnut	Carrot, celery, fennel, parsley, parsnip, potato, soybean, spinach	Chicory
Ragweed	Banana, kiwi,* (cantaloupe, honeydew, watermelon) peach*	Carrot, celery, cucumber, fennel, peppers, courgette (zucchini)	Chamomile, coriander, dandelion
Mugwort	Apple, mango,* peach,* watermelon	Avocado, carrot, celery, fennel, parsley, peppers	Aniseed, caraway seed, chamomile, coriander, mustard, sunflower

*Triggers that are also reported oral lichen planus triggers.

plant-derived foods.⁷ Nearly all patients with OAS have a history of allergic rhinitis, and reactions are mediated by IgE crossreactivity between pollen and food allergens. Common pollen–food associations include birch pollen and Rosaceae fruits, ragweed–melon–banana and mugwort–celery–spice (Table 1).⁸

The connection between OLP, plant-based food triggers and allergies has not been well explored, although anecdotally, patients with OLP often report food triggers. One retrospective study of 723 patients with biopsy-proven OLP mentioned that certain spicy or acidic foods could exacerbate their OLP.⁵ Another retrospective study found that 42% of patients with OLP reported unverified allergies, including food and pollen allergies.⁹ Food allergens may be able to upregulate or unmask keratinocyte antigen expression to activate an autoimmune response and contribute to acute flares. Additionally, local trauma/inflammation triggered by oral allergens may induce new OLP lesions due to the Koebner phenomenon, which has been reported for cutaneous lichen planus.¹⁰

In summary, avoidance of food allergens considerably improved the management of refractory OLP in a patient with concurrent diagnoses of OLP and OAS. This case demonstrates that trigger avoidance can significantly improve the chronic management of OLP and reduce exacerbations when specific triggers are identified. This report is limited to the description of a single patient, thus further studies are needed to examine the overlap between OLP and OAS, pinpoint common triggers of OLP, and explore the role of trigger avoidance as a potentially effective management strategy.

Learning Points

- OLP is a chronic autoimmune condition of unknown aetiology, characterized by periodic exacerbations refractory to medical therapy.
- Oral allergy syndrome is the most prevalent food allergy and is mediated by IgE cross-reactivity between pollen and food allergens.

- Many patients with OLP report triggers that overlap with triggers of other oral diseases, including OAS.
- Identification and avoidance of food allergens or other known triggers can result in a clinically significant improvement in refractory OLP.
- Trigger avoidance, which is often overlooked by clinicians, can be an effective strategy in the management of OLP.

References

- 1 Gorouhi F, Davari P, Fazel N. Cutaneous and mucosal lichen planus: a comprehensive review of clinical subtypes, risk factors, diagnosis, and prognosis. *Scientific World Journal* 2014; **2014**: 742826.
- 2 Roopashree MR, Gondhalekar RV, Shashikanth MC *et al.* Pathogenesis of oral lichen planus—a review. *J Oral Pathol Med* 2010; **39**: 729–34.
- 3 Thongprasom K, Dhanuthai K. Steroids in the treatment of lichen planus: a review. *J Oral Sci* 2008; **50**: 377–85.
- 4 Sugerman PB, Savage NW, Walsh LJ *et al.* The pathogenesis of oral lichen planus. *Crit Rev Oral Biol Med* 2002; **13**: 350–65.
- 5 Eisen D. The clinical features, malignant potential, and systemic associations of oral lichen planus: a study of 723 patients. *J Am Acad Dermatol* 2002; **46**: 207–14.
- 6 Gavic L, Cigic L, Biocina Lukenda D *et al.* The role of anxiety, depression, and psychological stress on the clinical status of recurrent aphthous stomatitis and oral lichen planus. *J Oral Pathol Med* 2014; **43**: 410–17.
- 7 Konstantinou GN, Grattan CE. Food contact hypersensitivity syndrome: the mucosal contact urticaria paradigm. *Clin Exp Dermatol* 2008; **33**: 383–9.
- 8 Price A, Ramachandran S, Smith GP *et al.* Oral allergy syndrome (pollen-food allergy syndrome). *Dermatitis* 2015; **26**: 78–88.
- 9 Chainani-Wu N, Silverman S Jr, Lozada-Nur F *et al.* Oral lichen planus: patient profile, disease progression and treatment responses. *J Am Dent Assoc* 2001; **132**: 901–9.
- 10 Camargo CM, Brotas AM, Ramos-e-Silva M, Carneiro S. Isomorphic phenomenon of Koebner: facts and controversies. *Clin Dermatol* 2013; **31**: 741–9.