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A sore and sensitive tongue

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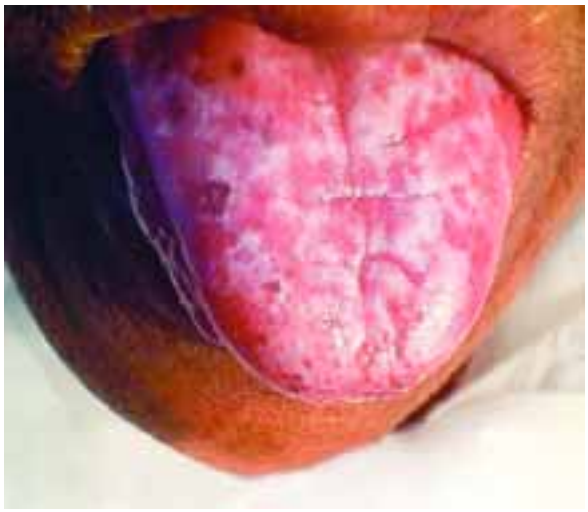
A 40-year-old woman came into the office with a 1-year history of painful red lesions with white striations on her tongue (**Figure 1**). The lesions caused burning after she ate spicy food and an increased sensitivity to mouthwash. She reported a slow, progressive onset and worsening of her condition, with intensifying symptoms during periods of emotional stress.

On physical examination, we observed an erythematous and cream-colored reticulated patch with a few small focal areas of erosion covering the dorsal tongue. The remainder of the oral mucosa appeared normal. She also had thinning and ridging of the toenails (**Figure 2**).

WHAT IS THE MOST LIKELY DIAGNOSIS?

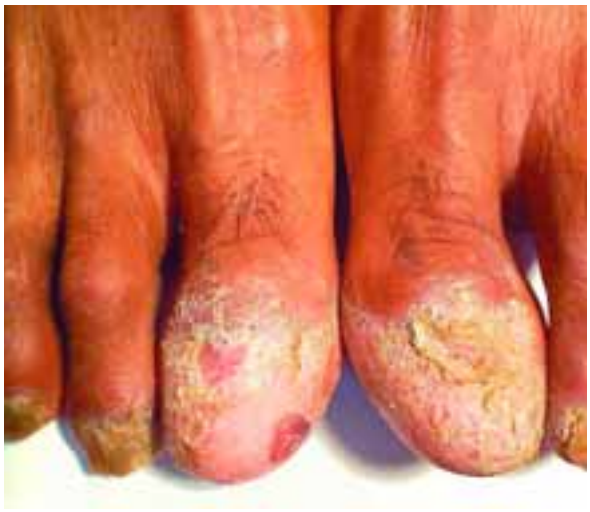
HOW WOULD YOU MANAGE THIS CASE?

FIGURE 1 Tongue with painful lesions



The patient's painful tongue, with erythema, white patches, and erosions.

FIGURE 2 Thinning toenails



The same patient's toenails, showing thinning and ridging.

DIAGNOSIS: ORAL LICHEN PLANUS

Compared with the more self-limited nature of its cutaneous counterpart, *oral lichen planus* (OLP) causes more chronic inflammatory lesions, resulting in increased morbidity and a greater therapeutic challenge for physicians. Of the 3 basic clinical morphologies of the disease (reticular, erythematous, and erosive), the last form tends to be more symptomatic and prompts patients to seek treatment.[1,2] The more asymptomatic reticular form is the most easily recognizable variant and may cause increased symptoms when involving the tongue, typically manifesting on the dorsal surface.[1]

In contrast to reticular lesions in other oral sites, tongue lesions do not usually exhibit the characteristic interlacing pattern of white, raised striae; instead they manifest as well-demarcated areas with a patch-like pattern of erythematous, atrophic, and keratotic regions.[3] Erosive OLP more commonly involves the lateral tongue and displays erythematous or ulcerated areas with peripheral keratosis, forming fine centrifugal striae.[3]

Oral lichen planus affects approximately 1% to 4% of the population[4] and is seen most commonly in older women.[1,5,6] It has a predilection for bilateral involvement of the buccal mucosa but may also affect (in descending order of frequency) the tongue, gingiva, lips, floor of the mouth, and palate.[1,2] Isolated involvement of only 1 oral site is infrequent, with the exception of gingival lesions.[1] Extraoral cutaneous lichen planus has been reported in 16% to 44% of patients with oral disease; it most frequently involves the genital mucosa, as well as the scalp, nails, and esophageal and ocular mucosae.[4] A multidisciplinary approach including generalists, dermatologists, otolaryngologists, ophthalmologists, and gynecologists may be necessary for patient evaluation and management.

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Pathogenesis of oral lichen planus

Although the precise cause of OLP is unknown, its pathogenesis has been linked to an autoimmune mechanism involving autocytotoxic CD8+ T cells, which trigger apoptosis (programmed cell death) of basal keratinocytes.[4,7] An imbalance between T cell helper and suppressor activity has also been observed.[1,7]

Complications: Carcinoma, chronic liver disease

Malignant transformation to squamous cell carcinoma (SCC) is seen in 0.4% to 5% of patients with OLP, particularly those with erosive and erythematous disease.[1,2] Increased risk factors for SCC have not been identified in these patients, but a greater prevalence of *Candida albicans* may be associated with carcinogenesis, as may herpes simplex and human papilloma viruses, immunosuppressive therapy, and an inflammatory cytokine-rich microenvironment.[1]

In Japan and parts of Southern Europe, OLP has been associated with hepatitis C infection and chronic liver disease, but these findings have not been reproduced in patients in the US.

MAKING THE DIAGNOSIS: EXAM, BIOPSY, IMMUNOFLUORESCENCE

A detailed history and physical examination are usually sufficient to diagnose OLP, although laboratory studies and biopsy may be required to exclude malignancy or distinguish OLP from conditions such as pemphigus vulgaris and mucous membrane pemphigoid. A biopsy is rarely needed, and should only be performed by physicians that have training in a tongue biopsy.

If necessary, biopsy samples should be obtained from the most representative sample area of the tongue. After administration of local anesthesia (lidocaine 1:100,000 with epinephrine) near the periphery of the site, biopsy is performed using a #15 blade scalpel or biopsy punch. An assistant may be needed to grasp the tongue by wrapping a gauze sponge over the tip and

securing it firmly. When tongue biopsy sites are closed, deep sutures should be placed if necessary, and mucosal sutures should be placed relatively close together, as inadequately closed wounds on the tongue tend to reopen, resulting in bleeding and prolonged healing time.

Specimens should be placed in a 10% formalin solution for transportation to a pathologist. Patients should return to the clinic after 7 to 14 days for examination of the biopsy site and suture removal.

Microscopically, OLP demonstrates vacuolar degeneration, basal cell lysis, and liquefactive degeneration[1] along with focal hyperkeratosis, a characteristic amorphous eosinophilic band at the basement membrane level, and a dense, bandlike subepithelial lymphocytic infiltrate.[5,6] Direct immunofluorescence, displaying granular fibrinogen and variable immunoglobulin deposited linearly near the basement membrane, and possibly cytoid bodies, is useful in cases of suspected OLP with nondiagnostic histological findings, as well as in those with gingival involvement.[1]

DIFFERENTIAL DIAGNOSIS

The clinical differential diagnosis of OLP includes the autoimmune bullous diseases pemphigus vulgaris, mucous membrane pemphigoid, dermatitis herpetiformis, and linear immuno-globulin A disease, which can all be excluded with direct and indirect immunofluorescence studies. When clinically appropriate, biopsy specimens submitted for direct immunofluorescence must be preserved in Michel solution. Reactive keratoses, chronic hyperplastic candidosis, epithelial dysplasia, discoid lupus erythematosus, anemic states, and several gastrointestinal diseases including oral Crohn disease and chronic hepatic disease, must also be ruled out.

Oral lichenoid reactions may develop as a hypersensitivity to dental materials and drugs, and must also be considered.[1] Patch testing may help identify a contact allergy to dental prosthesis components, including gold, mercury, and palladium salts.

Oral lichenoid reactions may develop as a hypersensitivity to dental materials or drugs

MANAGEMENT: STEROIDS, IMMUNOSUPPRESSANTS, SURGERY

Because fewer than 20% of patients experience total remission,[5] treatment of OLP is chronic and palliative. Most symptomatic patients are taught to avoid exacerbating factors and given topical steroid therapy (dexamethasone rinse, fluocinolone gel, triamcinolone cream). Observation, intralesional and systemic corticosteroids, immunosuppressants, antifungals, retinoids, antimalarials, dapsone, oral psoralen-ultraviolet-light (PUVA) treatment, and surgical techniques (CO_2 and neodymium: yttrium-aluminum-garnet [Nd:YAG] laser therapy, cryotherapy, and excision) are also employed, alone and in combination.[5,8]

Tacrolimus (Protopic), an immunomodulator, administered topically in 0.03% to 0.3% concentrations using Aquaphor or paraffin ointment base, has been shown effective and well tolerated in controlling symptoms,[8,9] and is a less costly alternative to topical cyclosporine.[9] It has been demonstrated effective prospectively,[10] and is safe in long-term therapy[11] of erosive OLP, but it has been reported in one case to cause hyperpigmentation of oral mucosa.[12]

A recent study[13] demonstrated the utility of low-dose 308-nm excimer laser radiation for symptomatic OLP.

PATIENT MANAGEMENT

Our patient was instructed to avoid foods and substances that caused irritation of her tongue and oral mucosa. In addition, she was prescribed topical fluocinolone gel 0.025% 3 times daily, and was given information about alternative treatment options, including tacrolimus and surgical therapy. She was instructed to perform gentle yet thorough daily oral hygiene and to follow-up in 6 months for re-examination.

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CORRECTION

December 2004's Photo Rounds, "Rupturing bullae not respondign to antiobiotics," left out the names of two of the article's authors. The correct authors of the piece are John Sauret, MD, FAAFP, Sandra Yale, DO, and Ahunna Ahiarah, MD. We regret the error.