Oral Lichen Planus
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For 5 days, a 19-year-old boy had nasal congestion, dry and itchy eyes, edematous lips, nausea, and sore throat. His medical history was significant for meningitis in infancy, herpes simplex virus (HSV) gingivostomatitis, and a tooth extraction about 2 weeks earlier. He was in college and lived with his mother. He was taking no medications other than NSAIDs for pain. He smoked cigarettes and marijuana and was sexually active.

On physical examination, temperature was 38.1ºC (100.6ºF); heart rate and blood pressure were stable. A vesicular lesion was noted in the right superior palpebral conjunctiva; the sclerae were mildly injected. The lips were dry, cracked, and slightly edematous; there was mild pharyngeal erythema, and vesicular lesions were visible on the gums, soft palate, and posterior oropharynx.

The teen was admitted to the inpatient unit, and intravenous acyclovir was started. Over the next few days, the oral lesions began to ulcerate and spread and were extremely painful. He was unable to eat or speak. He was given patient-controlled analgesia with morphine and parenteral nutrition.

A biopsy of one of the oral lesions revealed vascular mural C3 deposition. Oral lichen planus was diagnosed. Serologies were negative for HSV, Epstein-Barr virus, cytomegalovirus, and HIV. Results of all other tests were normal.

Although lichen planus classically manifests as a cutaneous rash of purple, polygonal, pruritic papules and plaques (known as the 5 P’s), the disease can affect the scalp, nails, genitalia, eyes, oral cavity, and even the esophagus. Oral lichen planus typically presents as 1 of 3 major clinical forms: reticular, atrophic, or erosive. This patient had erosive lichen planus, the most severe form of the disease, which exhibits denuding and sloughing of the mucocutaneous surface, represented either by erythematous bullae and ulcers or by a desquamative gingivitis.1 The lesions are most commonly found on the buccal mucosa; however, the tongue and labial mucosa may be involved.

Lichen planus is uncommon in children and adolescents, which may be why it is frequently misdiagnosed as candida infection, HSV infection, and recurrent aphthous stomatitis in this population.1 Confirmation of a clinical diagnosis of lichen planus with mucosal biopsy for histologic examination and direct immunofluorescence is usually necessary. Direct immunofluorescence may show IgM and complement deposition in a subepidermal distribution as well as fibrin and fibrinogen deposited in a linear pattern at the basement membrane.2

Our patient had many of the risk factors associated with lichen planus, including smoking, alcohol consumption, oral trauma (eg, tooth extraction), and NSAID use. Other triggers include citrus and spicy foods, stress, and systemic illness.

The mainstay of treatment is corticosteroids, which alleviate symptoms and alter disease progression. Topical corticosteroids have been shown to be as effective as systemic corticosteroids in decreasing symptoms and improving remission rates and are accompanied by fewer adverse effects.3 Educating patients on how to reduce exposure to offending agents is paramount to decrease the risk of an exacerbation.

This patient was treated with intravenous and topical corticosteroids. A dramatic improvement was seen within 2 days. Before discharge, the treatment regimen was switched to oral corticosteroids. Outpatient dermatologic and dental follow-up were scheduled. Dermatologic follow-up is recommended indefinitely because the risk of malignant transformation in patients with oral lichen planus is reported to be increased compared with that in the general population. In patients with erosive lichen planus, the risk of squamous cell carcinoma may be 0.4% to 5%.1

REFERENCES:

(Photograph courtesy of Anne V. LaRaia, MD.)