

DIAGNOSTIC CHALLENGE

Rash around the mouth

What is it?

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THE CHALLENGE

A 42-year-old woman had a persistent and progressively worsening rash around the mouth (Figures 1 and 2) for several months. She had a history of chronic scaling and erythema of the eyebrows, chin, and scalp that had been diagnosed as seborrheic dermatitis. Treatment with betamethasone dipropionate and clotrimazole cream once or twice daily for 4 to 5 months initially seemed to improve the rash, but the patient soon found it impossible to stop using the medicated cream without the rash getting worse. The patient stated that she had a “terrible flare” when she completely ran out of medication, and that flare is what prompted her visit to the dermatologist (S.E.H.).



Figure 1. Erythema and scaling are associated with scattered 1-millimeter papules on the chin and upper lip.



Figure 2. Lateral and closer view showing extension of the rash onto the cheek; this view also better demonstrates the papular nature of the rash.

CAN YOU MAKE THE DIAGNOSIS?

- A. allergic contact dermatitis
- B. irritant contact dermatitis
- C. perioral dermatitis
- D. acne rosacea
- E. seborrheic dermatitis

THE DIAGNOSIS

C. perioral dermatitis

The dermatologist made the diagnosis of perioral dermatitis (PD) on the basis of the appearance of the rash, the history of long-term use of topical steroids, and the flaring that occurred when the patient discontinued the use of the steroid cream. The patient was instructed to avoid the use of all topical steroids and was treated with tetracycline 500 milligrams twice daily to suppress the acne component of PD for its antibiotic and anti-inflammatory effects. In addition, the dermatologist prescribed ketoconazole cream once daily to suppress *Pityrosporum ovale*, which was the microbiological cause of the patient's underlying seborrheic dermatitis. Finally, it was further explained that improvement would be slow and, indeed, 4 weeks were required to achieve significant clearing (Figure 3).

PERIORAL DERMATITIS

PD also referred to as periorificial dermatitis, is a pruritic, scaly, acneiform eruption that dentists can recognize and treat. Unfortunately, this diagnosis means different things to different practitioners. To some, it is any rash occurring around the mouth, yet to dermatologists, it is a specific clinical entity with a characteristic clinical presentation, which was described first in 1957 by Frumess and Lewis¹ as a "light sensitive seborrheid." Understanding the clinical appearance and the pathophysiologic basis for this condition can prevent its occurrence and guide proper treatment.²⁻⁴

PD may occur de novo, but it is associated commonly with the long-term use of topical steroids applied to the face for a variety of conditions. Combination products that contain a topical steroid with an antifungal agent are particularly common culprits. It also has been described in association with inhaled steroids used for asthma.⁴ More potent steroids may be more likely to cause PD, but even over-the-counter hydrocortisone, the mildest topical steroid, has been implicated with prolonged use.^{5,6}

Manifestations of PD. PD typically manifests as a circumoral eruption composed of 1- to 2-millimeters fine acneiform and scaly erythematous papules with a salmon-colored background. PD generally spares the vermilion border and is accompanied by stinging, burning, and pruritus.^{2,3} Other reported aggravating factors include hand-to-mouth activity, acne rosacea, seborrheic dermatitis, and ultraviolet light exposure.

Pathophysiology of PD. The pathophysiologic basis of the commonly seen form of PD induced by use of topical steroids results from a direct vasoconstrictive effect of the medication and local immune suppression,



Figure 3. Excellent improvement is noted 1 month after treatment with oral doxycycline, clindamycin lotion, and pramoxine lotion.

leading to an overgrowth of various bacteria, yeast, *Demodex* mites, or other organisms within hair follicles.⁷⁻⁹ Withdrawal of the topical steroid medication leads to flaring when suppressed local immunity is reconstituted. The resultant inflammation of hair follicles produces suppurative folliculitis accompanied by erythema, scaling, inflamed perifollicular papules, pustules, and pruritus. Granulomatous changes also have been identified.⁹ This flare stimulates the patient to reapply the topical steroid to calm the inflammation. The topical corticosteroid has direct antierythema (vasoconstrictive), antipruritic, and anti-inflammatory effects that make PD less prominent. When the dermatitis improves, the steroid is discontinued, but the condition recurs as a "rebound phenomenon" that occurs over and over again. A vicious cycle is established, with the PD ultimately getting worse over weeks and months, with patients unable to stop using the topical steroid because it provides temporary relief.¹⁰ We have referred to this cycle as "topical steroid addiction." Topical calcineurin inhibitors (tacrolimus and pimecrolimus) have been reported to cause PD through a similar local immunosuppressive mechanism, but this is not nearly as common as that induced by corticosteroids.¹¹ Several reports have focused on the possibility that PD could be initiated by fluoridated toothpastes or stannous fluoride rinses.¹²⁻¹⁴ In these cases, papules and pustules may appear in the immediate circumoral area without the rim of sparing just outside the vermilion border typical of most cases of PD. The mechanism for this reaction is not clear, although oral fluoride and other halogens have been reported to aggravate rosacea and are considered to be proinflammatory.^{12,14}

Prevention of PD. Prevention of the severe, steroid-aggravated form of PD is possible if health care providers use only the mildest steroids for limited periods when treating patients' facial rashes. This dictum is applicable to dentists who also may use topical steroids to treat cheilitis. Treatment requires the elimination of all topical steroids. Abrupt elimination of the topical steroid leads to uniform flaring, prompting patients to reapply topical steroids, compounding the cycle of local immunosuppression combined with vasoconstriction and rebound vasodilatation that ultimately worsens the condition (topical steroid addiction).¹⁰

Treatment of PD. Treatment with a systemic antibiotic (doxycycline 50 to 100 mg by mouth twice daily, minocycline 50 to 100 mg by mouth twice daily, or tetracycline 500 mg two to four times daily) for its antibiotic and anti-inflammatory effects, a topical antibiotic (clindamycin lotion twice daily or metronidazole lotion), and an over-the-counter topical antipruritic such as pramoxine 1% lotion as needed will lead to improvement in problems with itching.^{15,16}

Topical ketoconazole cream also may be beneficial. It works exceptionally well in patients with associated seborrheic dermatitis. Clinicians should warn patients that, even with proper treatment, flaring of the condition usually will occur in the week following discontinuation of the topical steroid. The clinician must warn the patient about this so that the patient will avoid the tendency to restart topical steroids to gain immediate relief. In patients who have not been using topical steroids on the face, eliminating the use of fluoridated toothpaste and replacing it with a baking soda-based toothpaste, such as one of Tom's of Maine Fluoride Tooth Pastes, Nature's Gate Natural Toothpaste, or Cleure Hypoallergenic, Mint Free, Sensitive Teeth Toothpaste, may be prudent.

DIFFERENTIAL DIAGNOSIS

PD should not be used as the medical diagnosis for every rash around the mouth. True PD is a crossover between an acnelike process and a scaling eczematous process. It should be distinguished from allergic contact dermatitis, irritant contact dermatitis, acne rosacea, and seborrheic dermatitis.¹⁷

Allergic contact dermatitis. Allergic contact dermatitis in the perioral area can be caused by flavoring agents, preservatives, surfactants, and other ingredients in toothpastes, chewing gums, and mouthwashes, as well as cosmetic products.^{15,17,18} Erythema, scaling, and papulovesicular rashing are common. Acneiform papules and pustules of PD are not present. Histopathology of allergic contact dermatitis is characterized by a spongiotic dermatitis with eosinophils.⁸

Irritant contact dermatitis. Irritant contact dermatitis can be caused by soap, detergents, and a wide variety of harsh chemicals. Most interesting to dentists

is the irritant dermatitis commonly caused by pyrophosphates in tartar control toothpaste.^{12,19,20} These conditions are associated with scaly, erythematous patches with fine papules, but not the follicular papules and pustules that define acneiform eruptions such as PD. Histopathology of irritant dermatitis shows a spongiotic dermatitis absent the eosinophils of an allergic contact dermatitis and absent the suppurative folliculitis of PD.

Acne rosacea. Acne rosacea demonstrates acneiform papules and pustules on the nose, cheeks, and chin with background erythema and telangiectasias that often eventuates in a thickening of the nose, termed rhinophyma. The distribution is quite different than the perioral location of PD, although the identical histopathology (suppurative folliculitis) is seen in both PD and acne rosacea.

Seborrheic dermatitis. Seborrheic dermatitis is a condition initiated by the overgrowth of *Pityrosporum* yeast. Laypeople refer to this condition as "dandruff," but erythema and scaling commonly occur in the eyebrows, glabellar area, nasolabial folds, and external auditory canals in addition to the scalp. This distribution is quite distinct from the perioral location of PD. The acneiform lesions of PD also are not seen in this condition. Histopathology demonstrates a spongiotic dermatitis indistinguishable from irritant dermatitis.

CONCLUSIONS

To help patients resolve issues with perioral conditions most effectively, dental professionals should obtain a complete and thorough history of patients' possible topical steroid treatment and review the use of oral health products. In addition, dental professionals must inform patients that continued use of topical steroids will delay the clearing of PD. Dentists may choose to refer patients with PD to a dermatologist or to their primary care physician if the diagnosis is in doubt or the treatment is ineffective. ■

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