

## Perioral Dermatitis

**Suzana Ljubojević<sup>1</sup>, Jasna Lipozenčić<sup>1</sup>, Petra Turčić<sup>2</sup>**

<sup>1</sup>University Department of Dermatology and Venereology, Zagreb University Hospital Center and School of Medicine; <sup>2</sup>Faculty of Pharmacy and Biochemistry, University of Zagreb, Zagreb, Croatia

### Corresponding author:

Suzana Ljubojević, MD, PhD  
University Department of Dermatology  
and Venereology  
Zagreb University Hospital Center and  
School of Medicine  
Šalata 4  
HR-10000 Zagreb  
Croatia  
*suzana.ljubojevic@t-com.hr*

**SUMMARY** Perioral dermatitis is an inflammatory facial skin disorder that predominantly affects women. It is rarely diagnosed in children. The etiology of perioral dermatitis is unknown; however, uncritical use of topical corticosteroids often precedes skin lesions. There is a written diagnostic work-up, differential diagnosis and treatment.

**KEY WORDS:** rosacea-like dermatitis, steroid facial dermatitis, papulopustular facial dermatitis

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### INTRODUCTION

Perioral dermatitis (PD), rosacea-like dermatitis, periorificial dermatitis, light-sensitive seborrheic, chronic papulopustular facial dermatitis, papulopustular facial dermatitis, granulomatous perioral dermatitis, lupus-like perioral dermatitis, stewardess disease are synonyms for a chronic papulopustular facial dermatitis. It mostly occurs in young women. The clinical and histologic features of the lesions resemble those of rosacea. Patients require systemic and/or topical treatment, evaluation of the underlying factors, and reassurance. In 1957, Frumess and Lewis described cyclic dermatitis affecting the skin of the perioral region, principally among young females, by the term "light sensitive seborrhoeid" (1).

### EPIDEMIOLOGY

PD predominantly affects women, who account for an estimated 90% of cases. The number of male patients is assumed to be increasing because of changes in their cosmetic habits. PD may occur but is rarely diagnosed in children (2, 3). The vast majority of patients are women aged 20-45 years (2).

### PATHOGENESIS

There may be more than one cause of PD (Table 1). The etiology of PD remains unknown; however, the uncritical use of topical steroids for minor skin alterations of the face often precedes the manifestation of the disease (5). The underlying cause cannot be detected in all patients. Once

PD has developed, corticosteroid creams seem to help, but the disorder reappears when the treatment is discontinued. In fact, PD usually comes back even worse than it was before the use of steroid creams. The use of inhaled prescription steroid sprays applied into the nose and mouth can also induce PD. Fluorinated toothpaste, overuse of heavy face creams and moisturizers, especially those with a petrolatum or paraffin base, and the vehicle isopropyl myristate are another common cause. Physical factors such as UV light, heat and wind worsen PD. Many investigators have considered that infections may cause PD. Microbiologic factors such as fusiform spirilla bacteria, *Candida* species, *Demodex folliculorum* and other fungi have been cultured from lesions. Their presence has no clear clinical relevance. Hormonal factors are suspected because of the premenstrual deterioration observed. Oral contraceptives may also be a causative factor. Gastrointestinal disturbances such as malabsorption have been considered as well (5).

### CLINICAL FEATURES

The disease is limited to the skin. Skin lesions occur as grouped follicular reddish papules, papulovesicles and papulopustules on an erythematous base with a possible confluent aspect (Figs 1 and 2). The papules and pustules have mainly perioral locations. The predominant locations of PD lesions are the perioral area, nasolabial fold and lateral portions of lower eyelids. In an extreme variant of the disease called lupus-like PD, granulomatous infiltrates have a yellowish aspect at diascopy. A frequently seen feature of PD is a border of normal skin separating lesional skin from the lips (Fig. 1). In the perioral type, discrete to moderate erythematous papules and pustules are found circularly, with a clear zone of 3-5 mm under the lower lip (Fig. 1).



**Figure 1.** A patient with perioral type of steroid dermatitis

### Complications

Although PD is limited to the skin and is not a life-threatening condition, emotional problems may occur because of the disfiguring character of facial lesions and the possibly prolonged course of the disease. An initial rebound effect frequently occurs during the weaning of the steroid. This phenomenon is rare when no underlying cause can be identified. Chronic course is not uncommon. The development of a lupoid dermal infiltrate is considered to be a feature of the maximal variant of the disease. The diagnosis is made on the basis of yellowish discoloration after diascopy. This entity is called lupus-like PD. Scarring may be a problem with the lupoid form of PD.

**Table 1.** Etiology of perioral dermatitis

Drugs	<ul style="list-style-type: none"> <li>• Topical steroids</li> <li>• Inhaled prescription steroid sprays</li> </ul>
Cosmetics	<ul style="list-style-type: none"> <li>• Fluorinated toothpaste</li> <li>• Skin care ointments and creams</li> </ul>
Physical factors	<ul style="list-style-type: none"> <li>• UV light</li> <li>• Heat</li> <li>• Wind</li> </ul>
Microbiologic factors	<ul style="list-style-type: none"> <li>• Fusiform spirilla bacteria</li> <li>• <i>Candida</i> species</li> </ul>
Miscellaneous factors	<ul style="list-style-type: none"> <li>• Hormonal factors (oral contraceptives)</li> <li>• Gastrointestinal disturbances (malabsorption)</li> <li>• Emotional stress</li> </ul>



**Figure 2.** Perioral dermatitis. Diffuse erythematous papule, papulopustules which appear on eczematous skin

### HISTOPATHOLOGY

Histopathologic appearances of the biopsies are similar to those of rosacea (4). Changes in the follicular epidermis are marked by most of the authors (6). They suggest that the disorder might be provoked by some external irritant (6). Biopsies should be taken from the chin or nasolabial groove and should include at least 1 papule. It must be admitted that the changes are not diagnostic. Usually the clinical picture and history of the disease determine the diagnosis (5). Histopathologic examination of early papular lesions shows eczematous changes consisting of mild acanthosis, epidermal edema and parakeratosis. There are mainly ectatic venules and lymphocytes, mild edema and sparse lymphatic perivascular infiltration. Usually, small peripheral areas of the hair follicles are

edematous and are invaded by inflammatory cells. Sometimes follicular abscesses can be seen. The abscess cavity contains many polymorphonuclear leukocytes. Elastic fibers confirm the presence of elastic degeneration. Demodex mite can sometimes be demonstrated as an incidental finding. Examination of late papular lesions reveals diffuse hypertrophy of the connective tissue, accompanied by hyperplasia of sebaceous follicles. In the dermis, occasionally there is discrete epithelioid cell granuloma of the noncaseating type with perifollicular predominance and scanty Langerhans giant cells. Caseating granulomata are characteristic features of granulomatous PD (5).

### DIAGNOSIS

#### Clinical diagnosis

The diagnosis is made clinically. Usually good history of the disease, which reveals prolonged use of local corticosteroids or contact with other potential cause factors (Table 1) is enough. Clinical picture is also characteristic. Predominantly there are erythematous papules and papulopustules, usually localized in the perioral region. In more than 98% of cases, rebound phenomenon occurs (5). There is gradual disappearance of all symptoms, and relapses are rare unless corticosteroids are repeatedly administered.

#### Laboratory diagnosis

No laboratory abnormalities can be expected (4, 5). Prick tests and specific IgE testing against a mixture of aeroallergens have been used to test for skin barrier dysfunction. In a German study, PD

**Table 2.** Differential diagnosis of face rashes resembling perioral dermatitis

Rosacea	<ul style="list-style-type: none"> <li>• usually centofacial disease</li> <li>• no comedones</li> <li>• usually rhinophyma is present</li> </ul>
Seborrheic dermatitis	<ul style="list-style-type: none"> <li>• predominantly retroauricular, nasolabial region, eyebrow and scalp are affected</li> <li>• main symptom is scaling</li> </ul>
Acne vulgaris	<ul style="list-style-type: none"> <li>• comedones, papules, pustules, nodule, cysts</li> <li>• affects younger population</li> </ul>
Facial demodicosis	<ul style="list-style-type: none"> <li>• mycology isolation of <i>Demodex folliculorum</i>)</li> </ul>
Lupus miliaris disseminatus faciei	<ul style="list-style-type: none"> <li>• little scars are present</li> <li>• spontaneous regression</li> </ul>
Polymorphous light eruption	<ul style="list-style-type: none"> <li>• itchy red papules, vesicles or plaques</li> <li>• after sun exposure</li> </ul>
Contact dermatitis	<ul style="list-style-type: none"> <li>• border of the rash immerging into normal skin</li> </ul>
Haber syndrome (familial rosacea-like dermatosis)	<ul style="list-style-type: none"> <li>• begins in childhood, intraepidermal epitheliomas, keratotic plaques and scars</li> </ul>
Granulomatous periorificial dermatitis	<ul style="list-style-type: none"> <li>• in prepubertal children; yellow-brown papules limited to the perioral, perinasal and periocular regions</li> </ul>

patients experienced significantly increased transepidermal water loss compared with rosacea patients and a control group, which indicated a skin barrier function disorder. This type of testing is not routinely used (7).

### Differential diagnosis

PD is usually a straightforward clinical diagnosis (5). However, on differential diagnosis few facial skin diseases should be excluded (Table 2). Facial demodicosis (infestation with *Demodex folliculorum*) clinically resembles PD and should be excluded, especially when anti-inflammatory therapies fail. Patients who are prone to acne or rosacea may experience worsening while undergoing topical immunomodulating therapy (e.g., with tacrolimus ointment). Haber syndrome, or familial rosacea-like dermatosis with intraepidermal epitheliomas, keratotic plaques and scars, is a rare genodermatosis that begins in childhood. Granulomatous periorificial dermatitis manifests most commonly in prepubertal children as yellow-brown papules limited to the perioral, perinasal and periorcular regions. The condition is self-limiting and is not associated with systemic involvement.

### TREATMENT

The first step in therapeutic management should be discontinuation of all suspected topicals which, however, usually leads to relapse of skin lesion. One should insist on abandonment of all cosmetics, soaps, detergents, moisturizers, abrasives, astringents, day or night creams, skin conditioners, etc. Washing with mild water only, using fingers is suggested by some authors. However, this "null (zero) therapy" is hard for many patients, so local neutral treatment such as neutral local creams and compresses (chamomile tea, physiologic solution, etc.) have to be used. The duration of treatment is shorter with men because they give up the idea of ever being cured sooner than the women do. Sometimes the physician must provide a great deal of psychological support during office visits. Some of the patients develop corticosteroid dependence and therefore need medical help including psychological support to break the habit (8).

The patients have to be told that exacerbation is to be expected and that it may take many weeks to purify, and that the disease slowly regresses when exogenous factors are eliminated. Some investigators treat rebound phenomenon patients with hydrocortisone, because hydrocortisone cuts down the violence of the rebound reaction, while

allowing the atrophic collagen to recover (9). Others taper the dose of topical corticosteroids by reducing the frequency of administration (10).

The second factor in treatment is suppression of bacterial infection in hair follicles with systemic antibiotics. The population of *Propionibacterium acnes* within follicles is markedly elevated in patients who apply local corticosteroids. *Propionibacterium acnes* inflame follicles directly by producing agents chemotactic for polymorphonuclear leukocytes. Fusobacteria are often found in PD induced by fluorinated corticosteroids. Besides these two bacteria, in facial dermatoses induced by local corticosteroids one can find gram-negative bacteria, staphylococci or sometimes even streptococci. Preference is given to lipophilic tetracyclines like oxytetracycline, monocyline or doxycycline, 100-250 mg per day for 3-4 months, rarely longer. To prevent poststeroid flare, oral tetracyclines are contraindicated in children younger than 11 years. Acceptable treatment for children includes oral as well as topical erythromycin and topical metronidazole. If there is no response to full dose of tetracyclines, one may have to resort to isotretinoin. Quite low doses are effective; usually 5 mg as a simple daily dose for about 3 months; even 2-3 mg/day may be helpful. Precautions must be taken in women of childbearing potential. In less severe cases only, a neutral local therapy combined with anti-inflammatory agents can be used, mostly local erythromycin and metronidazole, neomycin, clindamycin and oxytetracycline administered in a nongreasy base (e.g., gel, lotion or cream). They have both moisturizing and antibiotic effects. The response of PD to metronidazole is the result of the drug's anti-inflammatory and immunosuppressive effects rather than the direct antimicrobial action (12). Topical antiacne medications such as adapalene and azelaic acid have been used (13) in open studies. Ointments should be avoided. In severe cases of PD, local immunomodulatory creams such as tacrolimus and pimecrolimus can be used (14,15).

Topical antipruritics containing no corticosteroids, such as liquid pramoxine hydrochloride, offer excellent symptomatic relief. The response to local treatment with sulfur, resorcin and ichthyol was very unsatisfactory.

### CONCLUSION

PD has become a quite common facial dermatitis nowadays because of the inappropriate use of topical steroids on the face. Various environmental sensitivities have been reported. The link to

rosacea is not certain but the two disorders occur in the same population and both respond to the same drugs.

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