

Perioral Dermatitis

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Figure 1

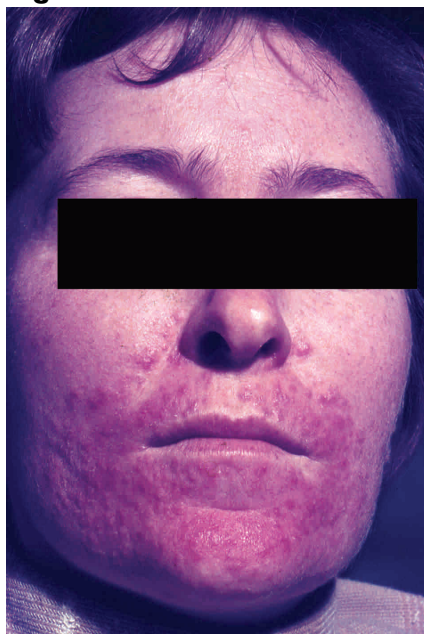


Figure 2

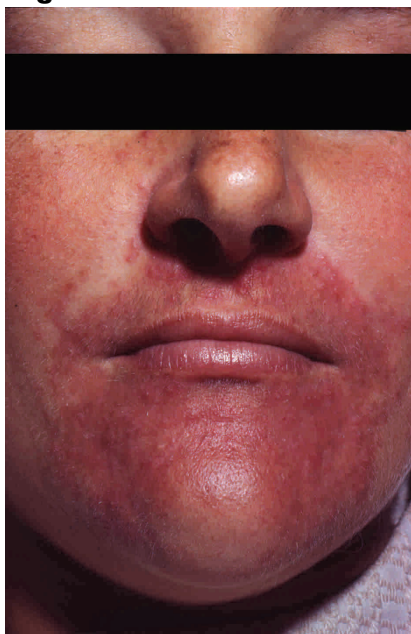


Figure 3



Introduction

Perioral dermatitis has a characteristic clinical picture, small erythematous papules and papulopustules erupt around the mouth, on the chin, in the nasolabial folds. A narrow band around the lips is spared.

(Figures 1, 2 & 3.)

In some patients the ala nasi, the glabella and the periocular areas are also affected while lesions on the cheeks are rare.

An exclusive periocular involvement has occasionally been observed and this form has been rather clumsily named "periocular perioral dermatitis".

The severity of perioral dermatitis varies between the patients. The course is often chronic and fluctuating. The lesions burn, rather than itch.

Epidemiology

Perioral dermatitis occurs mostly in young women (most often in 18-30 year old group). Men constitute less than 10% of the patients. Perioral dermatitis is also occasionally seen in children, but then it often shows some

histological differences and may be a distinct condition.

Perioral dermatitis was first described in the late 1950's¹ and its frequency markedly increased in the 1970's. Over the last 20 years there has been an apparent fall in new cases in the UK while the condition appears still quite frequently in the US, Australia and South Africa. In South Africa, perioral dermatitis is seen almost exclusively in people of European ancestry.

Aetiology and pathogenesis

The aetiology of perioral dermatitis remains unclear. Most likely, it is not a single factor that is responsible. At first, sun exposure was thought to be the cause. Later it was shown that it plays a minor, if any, role. Nowadays, use of potent topical corticosteroids is considered to be the main inducing and aggravating factor. It is thought that severe perioral dermatitis is the result of mild perioral dermatitis incorrectly treated with potent topical corticosteroids.

In many patients this association is obvious, as corticosteroid preparations were used freely for "eczemas", "pimples" and "roughness" of the skin.

The more potent the corticosteroid the more likely that it causes the lesions. Hydrocortisone acetate appears safe, but hydrocortisone butyrate is not. Systemically administered corticosteroids have also been found to be responsible. There is, however, a subgroup of patients denying use of any corticosteroid at any time.² Cosmetics (moisturizers, fatty lipsticks), fluorinated dental pastes, chewing gums and mouthwashes have also been incriminated.

Attempts to associate perioral dermatitis with infection or infestation (candida, or gram negative bacteria) have been unsuccessful.

The high prevalence of the condition in young females may suggest hormonal influences. Nikkels and Pierard³ described patients who developed perioral dermatitis after stopping contraceptive pills. They also noticed premenstrual flares in these

patients. The course of perioral dermatitis in these woman was chronic and recurrent.

Histopathology

The lesions of perioral dermatitis are seldom biopsied and the clinical picture is not diagnostic. The largest study that comprised 26 patients⁴ showed a perivascular and perifollicular mononuclear infiltrate and mild eczematous changes. Sarcoid-like granulomas have been described in some patients, mainly in children.

Perioral dermatitis in children

Perioral dermatitis in children clinically does not differ from its presentation in adults. As in adults, it most often occurs after the topical application of potent corticosteroids. Bubblegum was thought to be a causative factor in children reported in Australia.⁵

Gianotti *et al*⁶ reported a group of children under seven years of age showing, on histology, sarcoidal granulomas. The greater tendency to form granulomas in paediatric cases of perioral dermatitis was later confirmed by others.

Differential diagnosis

The typical appearance of perioral dermatitis usually suggests the correct diagnosis. The condition for differential diagnosis are listed in **Table 1**.

Table 1: Perioral dermatitis-differential diagnosis

- **Rosacea**
- **Acne**
- **Seborrhoeic dermatitis**
- **Sarcoidosis**

Rosacea, acne and seborrhoeic dermatitis may cause different diagnostic problems. In perioral dermatitis there are no telangiectasiae and flushing, characteristic of rosacea, and the lesions seldom spread onto the cheeks. **Fig. 4** and **5** show the difference. Acne in its classic juvenile form, and also other less common types, are easy to differentiate. The lesions in acne are polymorphic, include not only inflammatory papules and papulopustules but also comedones and deep cysts, and their distribution differs.

Figure 4

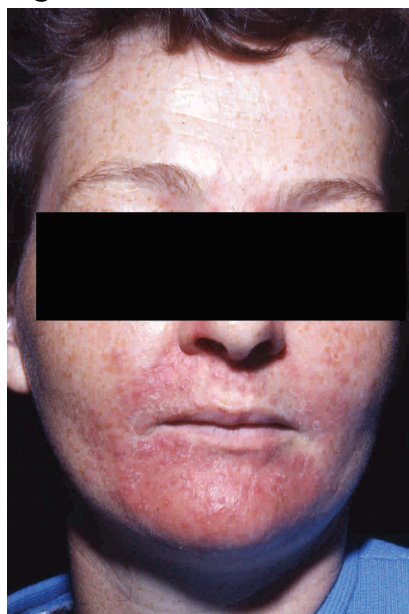


Figure 5

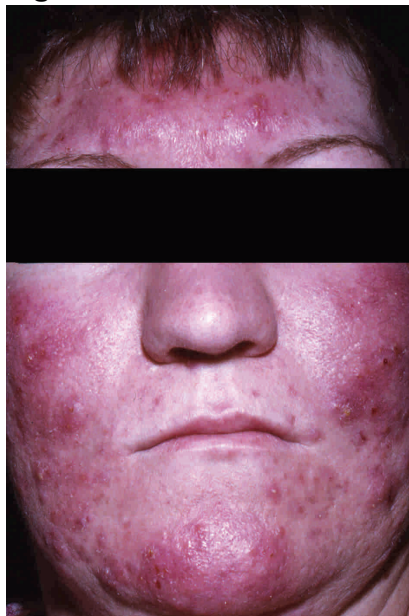


Fig. 4 and 5: Perioral dermatitis (fig4) versus Rosacea (fig5).

The lack of scaling, the presence of micropapules and the distribution of the lesions distinguish perioral dermatitis from seborrhoeic dermatitis.

Treatment

Avoidance of topical corticosteroids is the major part of the management. Patients have to be persuaded to stop all topical corticosteroids, and to stop them immediately rather than taper them by reducing their strength. Rebound flare-ups occur on discontinuation of topical corticosteroids but

these agents should not then be resumed.

In some patients who used topical corticosteroids for long periods, the flare-up may be severe. Antibiotics alone will not control it and a short course of systemic corticosteroids, 2 to 4 weeks with starting dose of about 40 mg/day, is needed. It is of great importance to develop a good relationship with the patients and to give reassurance as to the outcome. Oral tetracyclines have been found to be the most effective, either parenteral tetracycline or one of its derivatives such as minocycline, doxycycline, lymecycline.

Tetracycline is given in full dose (500 mg twice daily) until the patient responds. This usually takes 3 to 4 weeks. Later the dose is halved, until complete resolution. In most cases, the treatment can be stopped after 8 to 12 weeks. Starting doses for doxycycline and minocycline are 100 mg/day, and for lymecycline 600 mg daily.

The mechanism of action of tetracyclines in perioral dermatitis is unclear. For children and pregnant woman, or if tetracyclines are not tolerated, erythromycin 750 – 1000mg daily for several weeks is prescribed. Clarithromycin 250 mg daily for 10 days, followed by 250 mg on alternate days for a further 20 days, has been found to be very effective.⁷

Relapses after treatment with systemic antibiotics occur but they are not common.

Topical treatment of perioral dermatitis is generally much less effective.

Erythromycin solution (1.5-2%) or metronidazole gel (0.75%) are applied two times daily. The latter has to be applied sparingly only to the affected areas.✶

See CPD Questionnaire p.47

References:

1. Furness MG, Lewis MH. Light sensitive seborrhoeid. *Arch Dermatol.* 1957; 75, 245-248.
2. Malik R, Quirk CJ. Topical applications and perioral dermatitis. *Australas J Dermatol.* 2000; 41, 34-38.
3. Nikkels AF, Pierard GE. Control of perimenstrual flares of perioral dermatitis by isotretinoin. *J Dermatol Treatm.* 2000; 11, 97-99.
4. Marks R, Black MM. Perioral dermatitis: a histopathological study of 26 cases. *Br J Dermatol.* 1971; 84, 245-247.
5. Georguras K, Kocsard E. Micropapular sarcoidal facial eruption in a child: Gianotti-type perioral dermatitis. *Acta Dermatol Venerol.* 1978; 58, 433-436.
6. Gianotti F, Ermacora E, Benelli MG, Caputo R. Particulate dermatite peri-orale infantile. *Observations sur 5 cas.* *Bull Soc Fr Derm Syph.* 1970; 77, 341.
7. Vincenzi C, Parente G, Tosti A. Perioral granulomatous dermatitis: two cases treated with clarithromycin. *J Dermatol Treatm.* 2000; 11, 57-61.