A 30-year-old man presented to dermatology outpatients department (OPD) with erythematous papules around the mouth. About 5 months back, he started self-medicating with a topical cream for recurrent acne. After 3 weeks, he noted few papules and pustules on perioral areas. He consulted a physician, who prescribed fluticasone propionate 0.05% cream with initial improvement in the rash. But later, the rash worsened and he was prescribed clobetasol propionate 0.05% cream. After transient improvement, rash further deteriorated. He was asked to stop all topical medications (i.e. zero therapy) by another physician. In few days, there was a sudden flare-up of the rash and visited our OPD.

Examination revealed grouped follicular, reddish papules and papulopustules on an erythematous background around the mouth, on the chin, and nasolabial folds sparing a narrow band around the lips (Figure 1). Biopsy specimen from a papule showed follicular hyperkeratosis with perifollicular and perivascular lymphohistiocytic infiltrates (Figure 2). Differential diagnosis of perioral dermatitis (POD), rosacea, acne vulgaris, seborrhoeic dermatitis, allergic or irritant contact dermatitis, and sarcoidosis were considered. Rosacea was ruled out by the absence of characteristic telangiectasia and flushing. Acne often presents with polymorphic lesions and comedones and the distribution differs. The presence of micropapules and the distribution of the lesions excluded seborrhoeic dermatitis. Sarcoidosis was ruled out on histopathology. A clinico-pathological diagnosis of POD was made.

POD is a chronic eczematous facial dermatitis that characteristically involves areas surrounding the mouth sparing vermillion border of the lips, nasolabial folds, chin, lateral portions of lower eyelids, and periorcular areas. The lesions burn, rather than itch and the course is often chronic and fluctuating. POD occurs mostly in young females, and men constitute less than 10% of the cases suggesting hormonal influences. Nikkels and Pierard noted POD in females after stopping oral contraceptives. These patients also had
premenstrual flares.[1] POD is occasionally seen in children, but it shows histological difference and may be a different condition.[2] The etiology of POD remains unclear. Unscrupulous and unjustified use of potent topical corticosteroids is considered to be the main inducing and aggravating factor. Fluorinated toothpaste,[3] cosmetics (with a petrolatum or paraffin base), chewing gums, and mouthwashes are also incriminated. Attempts to associate POD with infections or infestations (candida or gram-negative bacteria) have been unsuccessful.

POD lesions are seldom biopsied. Histological findings are similar to those of rosacea, but the signs of actinic skin damage are generally less. Follicular hyperkeratosis with perifollicular and perivascular localization consisting predominantly of lymphocytes, histiocytes, and polymorphonuclear leucocytes can be expected with vasodilatation and edema of the papillary dermis. Occasionally, there have been epithelioid or giant cells, sarcoid-like infiltrates, or vasculitis of the leucoclastic type.[4]

Avoidance of topical corticosteroids is the major part of the management. Patients have to be persuaded to stop topical steroids immediately rather than tapering of the dose. Rebound flare-ups may occur but steroid creams should not be restarted. In minor presentations, as well as in children and pregnant women, anti-inflammatory topical therapy with metronidazole[5] and erythromycin is generally recommended. Calcineurin inhibitors such as pimecrolimus and tacrolimus are very effective. In severe conditions, oral tetracyclines or one of its derivatives such as minocycline, doxycycline or lymecycline are prescribed.

He was treated with doxycycline 100 mg daily and tacrolimus ointment (0.1%) application twice a day for 4 weeks.

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Cite this article as: Koley S, Mandal RK. Chronic, fluctuating, grouped, erythematous papules around the mouth. Indian Dermatol Online J 2014;5:229-30.

Source of Support: Nil, Conflict of Interest: Nil.