

# What's Your Diagnosis?®

## Sharpen Your Physical Diagnostic Skills

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### HISTORY

A 14-year-old girl presented with a persistent erythematous rash on her chin. The rash was slightly itchy. She had been treated with 1% hydrocortisone cream and then with an antifungal cream intermittently by her family physician for the past 5 months. The treatment seemed to help, but the rash did not resolve completely. Her past health was unremarkable. All other family members were healthy.

### PHYSICAL EXAMINATION

Erythematous papular rash noted on the chin. Remaining examination findings normal.

## WHAT'S YOUR DIAGNOSIS?

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## What's Your Diagnosis?

ANSWER: STEROID ROSACEA

Steroid rosacea is characterized by a rosacea-like dermatitis that develops in the facial area after prolonged use of topical corticosteroids (particularly fluorinated ones).<sup>1,3</sup> The condition was first described by Sneddon in 1969 who used the term "rosacea-like dermatitis."<sup>4</sup> In 1974, Leyden and colleagues<sup>5</sup> coined the term "steroid rosacea." Other investigators prefer the term "steroid dermatitis resembling rosacea."<sup>6</sup>

### EPIDEMIOLOGY AND PATHOGENESIS

Although usually caused by excessive use of potent topical fluorinated corticosteroids,<sup>1,3,7,8</sup> steroid rosacea may follow use of low-potency topical corticosteroids,

ticosteroids also inhibit collagen synthesis, thereby facilitating passive dilatation of blood vessels and easier visualization of dermal capillaries.<sup>11</sup>

### CLINICAL MANIFESTATIONS

In susceptible persons, steroid rosacea usually develops weeks to months after application of a topical corticosteroid on facial skin.<sup>6,12,13</sup> It characteristically presents with erythematous patches and papules (red face syndrome).<sup>6,12,13</sup> Pustules, telangiectasia, and atrophy may develop in untreated cases.<sup>6,12,13</sup> Sites of predilection include centrofacial, perioral, and periorcular areas.<sup>12,14</sup> Some patients describe itching or burning

sensation in the affected area; these patients often find their skin easily irritated by soaps and skin products.<sup>1,10</sup>

### HISTOPATHOLOGY

Histological features include mild acanthosis, mild edema, parakeratosis, perivascular and perifollicular lymphohistiocytic infiltrate, and vascular ectasia.<sup>6,9</sup>

### DIAGNOSIS

The diagnosis is mainly clinical, based on a history of topical corticosteroid use and the characteristic dermatologic features. Differential diagnosis includes tinea faciei, acne vulgaris, rosacea, folliculitis, seborrheic dermatitis, atopic dermatitis, contact dermatitis, nummular eczema, discoid lupus erythematosus, lupus pernio, and polymorphous light eruption. The distinctive fea-

tures of each condition allow a straightforward differentiation from steroid rosacea.

### COMPLICATIONS

Permanent telangiectasia and skin atrophy may result when steroid rosacea is left untreated.<sup>12</sup> Other complications associated with corticosteroid use include striae, hypopigmentation or depigmentation, telangiectasia, decreased subcutaneous adipose tissue, folliculitis, and steroid acne.<sup>3,14,15</sup> Percutaneous absorption of corticosteroids may, rarely, lead to systemic adverse effects, which include Cushing syndrome, hypothalamic-pituitary-adrenal suppression, cataracts, glaucoma, osteopenia/osteoporosis, and growth retardation.<sup>3,14,15</sup>

including over-the-counter preparations, in susceptible persons.<sup>9</sup> Steroid rosacea may also follow use of systemic or inhaled corticosteroids.<sup>8</sup>

The condition is common among identical twins, which indicates a genetic predisposition,<sup>9</sup> and more common in adults than in children.<sup>10</sup>

The exact pathogenesis is unknown. However, it is believed that the vasoconstrictive effect of corticosteroids leads to accumulation of nitric oxide—a potent vasodilator.<sup>12</sup> The immunosuppressive effect of corticosteroids may facilitate overgrowth of microorganisms (possibly *Demodex* or *Propionibacterium acnes*) on the skin surface. These microorganisms then act as superantigens and cause an inflammatory reaction.<sup>1</sup> Topical cor-

## PREVENTION

A precise diagnosis is required to ensure the need for a topical corticosteroid. When the diagnosis is unclear, consider referral to a dermatologist. Also consider treating facial dermatoses or pruritus with a topical calcineurin inhibitor (eg, tacrolimus or pimecrolimus) rather than a topical corticosteroid. In general, the least potent corticosteroid that can control the dermatitis should be used. Topical corticosteroids should not be applied more than twice a day; frequent use does not improve efficacy and increases the risk of adverse effects.<sup>3,14,15</sup> Topical fluorinated corticosteroids should not be applied to the face.

## MANAGEMENT

For patients with steroid rosacea, first discontinue the topical corticosteroid. In cases of prolonged use, the corticosteroid can be weaned. Topical calcineurin inhibitors, such as tacrolimus and pimecrolimus, have been shown to be effective in the treatment of corticosteroid-induced rosacea.<sup>1,2,16</sup> Aggravating factors, such as caffeine and spicy foods, should be minimized or avoided. In resistant cases or in situations in which topical corticosteroids were used for more than 6 months on the face, oral erythromycin (or oral tetracyclines in children older than 8 years) can be used for 4 to 8 weeks to hasten resolution.<sup>7,9</sup> In some cases, the condition can recur and may require retreatment.

This patient was treated with tacrolimus 0.03% ointment twice daily. The facial rash subsided in 6 weeks.

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