

Steroid-Induced Rosacealike Dermatitis: Case Report and Review of the Literature

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GOAL

To understand steroid-induced rosacealike dermatitis (SIRD) to better manage patients with the condition

LEARNING OBJECTIVES

Upon completion of this activity, dermatologists and general practitioners should be able to:

1. Explain the clinical features of SIRD, including the 3 subtypes.
2. Evaluate the multifactorial pathogenesis of SIRD.
3. Recognize the importance of a detailed patient history and physical examination to diagnose SIRD.

INTENDED AUDIENCE

This CME activity is designed for dermatologists and generalists.

CME Test and Instructions on page 195.

This article has been peer reviewed and approved by Michael Fisher, MD, Professor of Medicine, Albert Einstein College of Medicine. Review date: March 2009.

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Steroid-induced rosacealike dermatitis (SIRD) is an eruption composed of papules, pustules, papulovesicles, and sometimes nodules with telangiectatic vessels on a diffuse erythematous and edematous background. It results from prolonged topical steroid use or as a rebound phenomenon after discontinuation of topical steroid. There are 3 types of SIRD that are classified based on the

location of the eruption: perioral, centropacial, and diffuse. Diagnosis of this disease entity relies on a thorough patient history and physical examination. Treatment involves discontinuation of the offending topical steroid and administration of oral and/or topical antibiotics. Topical calcineurin antagonists should be considered as alternative or adjunctive therapies for patients who do not respond to traditional treatments. Dermatologists may need to provide psychological support during office visits for patients who have difficulty dealing with the discontinuation of topical steroid and/or the psychological impact of a flare. Epidemiology, pathogenesis, histopathology, and differential diagnosis of the entity also are reviewed.

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Case Report

A 42-year-old man previously not seen by a dermatologist contacted our office for an emergent appointment. The patient reported having an excruciating facial eruption of several days' duration that his primary care physician was concerned might have been a serious infection. Because of the urgency of his presentation, the patient underwent immediate evaluation.

On examination, there was diffuse background erythema on the chin, cheeks, and forehead with superimposed erythematous papules and pinpoint pustules (Figure). On further questioning, it was discovered that the patient had been prescribed a topical steroid for a facial rash more than a year ago. The steroid initially worked well, but stronger formulations had been necessary to progressively control the eruption. In the month prior to

presentation, the patient used betamethasone valerate ointment daily. Five days prior to presentation, he ran out of betamethasone valerate. The rash began to appear the day after the steroid was stopped and progressively worsened on a daily basis.

Based on the patient's history and physical examination, steroid-induced rosacealike dermatitis (SIRD) was diagnosed. The patient was counseled on the expected course of the disease. Treatment was initiated with doxycycline 100 mg twice daily, clindamycin phosphate lotion 1% once daily, and tacrolimus ointment 0.1% once daily. On follow-up a month later, the eruption had decreased in intensity by approximately 50%.

Comment

The development of topical corticosteroids in the 1950s opened new doors for dermatologists previously faced with treating intractable dermatoses. Since then and with the introduction of high-potency corticosteroids in the 1970s, a new dermatosis related to the application of topical steroids to facial skin, given several different names, has been described in the dermatology literature. As Weber¹ reported, the first cases of *rosacealike dermatitis* in the United States, Great Britain, Scandinavia, and West Germany each appeared years after the first publication on the clinical use of steroids in the respective countries. The first case was reported in 1957. Frumess and Lewis² described a dermatosis of unknown etiology that resembled seborrheic dermatitis, which they named *light-sensitive seborrheid*. In 1964, the term *perioral dermatitis* was coined by Mihan and Ayres.³ Sneddon⁴ used the term *rosacealike dermatitis* in 1969 because the eruption resembled rosacea, a well-established entity. Leyden et al⁵ named the disease *steroid rosacea*



Diffuse background erythema on the forehead with superimposed erythematous papules.

in 1974. In 1976, the term *steroid dermatitis resembling rosacea* was introduced.⁶ Numerous other terms have been used to describe this disease entity including but not limited to *rosacealike eruption from topical steroid* and *steroid-induced rosacealike dermatitis*. We prefer the latter because it not only indicates the morphology of the lesions but also points out their relationship with topical steroids.

Epidemiology—Steroid-induced rosacealike dermatitis results from repeated application of a topical steroid to the face. The duration of use necessary to produce SIRD can vary from days to several years. Two months is the average, but 6 months or more of application is common.^{6,9} Although it was believed that only high-potency topical steroids could produce SIRD, it is important to note that topical hydrocortisone 1% also can cause such an eruption after prolonged use.⁸

The exact incidence of SIRD is not known, but it is believed to affect women more than men. The most common age at presentation is 40 to 50 years^{1,9}; however, it also has been described in infants, children, and elderly patients.¹⁰⁻¹⁷ Despite its morphologic resemblance to rosacea, SIRD currently is not considered a variant of rosacea.^{9,18}

Clinical Features—Steroid-induced rosacealike dermatitis typically is an uncomfortable and painful condition. Patients experience a sensation of tightness, moderate stinging or burning, and dryness that can be intensely pruritic.⁹ The primary lesion in SIRD consists of pinpoint, red or flesh-colored papules, pustules, or papulovesicles. Further application of the topical steroid leads to a gradual spread of the lesions. The papules then dry up and are replaced by erythema. Eventually, patients develop persistent and diffuse erythematous and edematous skin with numerous telangiectatic vessels as well as deep follicular papules, pustules, and firm nodules.^{9,19} There is a lack of comedones, which is an important differentiating feature, especially from steroid acne.²⁰

Based on the location of the eruption, there are 3 types of SIRD: perioral, centropacial, and diffuse. The perioral type is the most common and presents with discrete papules and pustules with moderate erythema around the mouth, 3 to 5 mm around the vermilion border.⁹ This type of SIRD can be considered a subset of perioral dermatitis that is induced by topical steroid use. However, the percentage of cases of perioral dermatitis that result from a topical steroid versus being idiopathic is not clear. Children often present with the classic perioral type as well as perinasal and periocular eruptions.^{9,13,14} The centropacial type of SIRD involves the cheeks, lower eyelids, nose, forehead, and glabellar region. The perioral region usually is unaffected.

The diffuse type affects the entire face, forehead, and neck.⁹

Pathogenesis—The pathogenesis of SIRD and its rebound phenomenon is multifactorial but can be partially explained by several hypotheses. After prolonged application of a topical steroid, functional and anatomic cutaneous changes begin to occur.

Steroid-induced rosacealike dermatitis has been described as an intolerance reaction of seborrheic skin to topically applied steroids. The seborrheic type of skin seems to be an essential factor because in some experimental studies, application of potent steroids to healthy skin rich in sebaceous glands has resulted in typical rosacealike symptoms.^{1,5,9,20,21}

Topical steroids may inhibit collagen synthesis, leading to dermal atrophy. The decrease in supporting connective tissue allows for the passive dilation of blood vessels and easier visualization of dermal capillaries that clinically manifest as prominent telangiectases and background erythema.^{22,23}

The immunosuppressive effects of topical steroids may facilitate the overgrowth of various bacteria, yeast, *Demodex* mites, or other microorganisms in pilosebaceous glands, resulting in inflammatory reactions that produce papules and pustules.^{22,24} However, no specific organisms have been identified as a definite causative agent.²⁵⁻²⁸

The role of *Demodex* mites in the pathogenesis of SIRD has remained controversial.^{9,29} It has been reported that there is a significant increase in mite densities in patients with SIRD ($P < .001$).³⁰ It is possible the *Demodex* mites can cause an inflammatory or allergic reaction by blocking the hair follicles or acting as vectors for other microorganisms.³¹ However, it is important to remember that *Demodex* mites also are present on the skin of many healthy individuals and may only have a pathogenic role when present in high densities.³²

The immunosuppressive effects of topical steroids also may contribute to the rebound phenomenon. Although no specific organisms have been identified as a definite causative agent, some researchers believe that topical steroids facilitate the overgrowth of various bacteria, yeast, or other microorganisms on treated skin. These microorganisms may subsequently act as superantigens.^{22,24} Withdrawal from use of topical steroids and their immunosuppressive effects then enables a superantigen-mediated immunologic response with an accompanying proinflammatory cytokine release.^{24,33}

Steroids also inhibit the release of a natural dilator called endothelium-derived relaxing factor. Vasoconstriction leads to a buildup of multiple metabolites, such as nitric oxide (a potent vasodilator). When steroids are no longer present,

Differential Diagnosis of Steroid-Induced Rosacealike Dermatitis⁹

Disease	Differentiating Clinical Features
Rosacea	Rhinophyma, centrofacial region, flushing
Steroid acne	Papules and pustules are monomorphic in appearance, no background erythema
Acne vulgaris	Younger population, comedones
Seborrheic dermatitis	Scaling, no pustules, retroauricular, nasolabial region, eyebrows and scalp involvement
Systemic lupus erythematosus	No papules or pustules, systemic symptoms
Discoid lupus erythematosus	No papules or pustules, erythematous patches and plaques
Sarcoidosis	Violaceous plaques or flesh-colored nodules mostly on cheeks or nose, no pustules
Polymorphous light eruption	Specifically related to sun exposure, extrafacial involvement
Tinea faciei	Raised pink scaly edge, skin at lesion center is normal, usually unilateral and asymmetric
Lupus miliaris	Eyelid involvement
Acute eczema	Acute onset, no papules or pustules
Dermatomyositis	Eyelid involvement, extrafacial findings (eg, Gottron papules, macular violaceous erythema), systemic symptoms

the vasoconstrictive effect ceases. As a result, the diameter of blood vessels enlarge beyond their original presteroid diameter because of the accumulation of nitric oxide, which potentiates the erythema, burning, and pruritus seen in SIRD.²⁴

Histopathology—Histopathologic features of SIRD are similar to rosacea, though there is some variation depending on the stage and severity of the disease.⁹

In general, the epidermis shows eczematous changes consisting of edema, acanthosis, and parakeratosis. The histologic changes observed in the follicular epidermis indicate that SIRD is provoked by an external irritant.^{9,34} There are ectatic venules and a sparse perivascular lymphocytic infiltrate in the dermis. Sometimes follicular abscesses containing polymorphonuclear leukocytes can be seen. There also is diffuse hypertrophy of the connective tissue accompanied by sebaceous hyperplasia. Occasionally, noncaseating epithelioid granulomas with multiple foreign body–type multinucleated giant cells are observed.^{9,35,36}

Diagnosis—Steroid-induced rosacealike dermatitis often is diagnosed only after clinical suspicion

based on physical examination, which leads to obtaining a detailed patient history revealing topical steroid use. Biopsy features are not diagnostic and are not helpful.

It can be challenging to diagnose SIRD for several reasons. First, patients may not admit, recall, or even know that they are using a topical steroid on their face. The topical steroid initially may have been prescribed for use on other body parts, or the patients may have obtained topical steroids that had been rightfully prescribed for friends and family members for treatment of other dermatoses. The “magical effect” of topical steroids can lead patients as well as their family members and friends to believe that it is a panacea for all skin ailments.^{4,9} Second, it takes time for patients to recognize the cutaneous changes of SIRD. By the time the patient presents to a dermatologist, it is difficult for the patient to pinpoint the exact temporal relationship with topical steroids or to describe the initial morphology of the lesions.¹ Third, if there is another simultaneous inflammatory dermatosis present, the morphology of SIRD becomes even more difficult,

if not impossible, to appreciate. Therefore, it is extremely important to perform a detailed patient history and physical examination.

Differential Diagnosis—The differential diagnosis of SIRD includes but is not limited to rosacea, steroid acne, acne vulgaris, seborrheic dermatitis, systemic lupus erythematosus, discoid lupus erythematosus, sarcoidosis, polymorphous light eruption, tinea faciei, lupus miliaris, acute eczema, and dermatomyositis (Table). Although a biopsy of a suspected SIRD lesion is not diagnostic, a biopsy may be needed in certain situations to exclude other conditions. Under these circumstances, biopsy specimens should be taken from the chin or nasolabial groove and should include at least one papule.⁹

Treatment—The first and most essential step in the treatment of SIRD involves discontinuation of all topical steroids, which usually leads to a flare of the eruption.^{9,18,37} Patients must be told that the rebound phenomenon is to be expected and the disease regresses slowly, even after the exogenous factors have been removed and the appropriate treatments implemented.

Without any treatment, the severity of the initial rebound tends to subside after 10 to 14 days.³⁸ However, to avoid the rebound phenomenon and/or decrease its severity, some clinicians ask their patients to gradually taper the frequency of topical steroid applications, while others switch their patients to intermittent use of topical hydrocortisone 1% before discontinuation of all topical steroids.^{9,38-40} Goldman²⁴ has found that varying doses (20–60 mg) of prednisone, tapered over 1 to 2 weeks at most, can be helpful in treating the initial flare-ups of SIRD. For patients who have difficulty dealing with the discontinuation of topical steroid and/or the psychological impact of a flare, dermatologists need to provide emotional support. In certain situations, additional referrals for psychological counseling may be needed.^{9,11,41,42}

Although SIRD is not a subtype of rosacea, avoidance of all rosacea-aggravating foods, such as caffeine, alcohol, hot fluids, spicy foods, and fluoride, has been advocated.^{24,37} In addition, some investigators encourage patients to wash their face with water only and to abandon the use of all cosmetics, soaps, moisturizers, lotions, astringents, and day and night creams.⁹

Anti-inflammatory oral antibiotics have played a prominent role in the treatment of SIRD. It is unknown if the antibacterial or anti-inflammatory effects are primarily responsible for the clinical benefit. The preferred oral antibiotics are lipophilic tetracyclines, such as doxycycline and minocycline, in dosages of 100 to 200 mg daily

for 3 to 4 months. Longer duration of treatment rarely is needed.⁹ Oral metronidazole also has been used in patients who are unable to tolerate tetracyclines. If there is no improvement with a full dose of tetracycline, clinicians may attempt treatment with low-dose isotretinoin. An average dosage of 2 to 5 mg daily (often given as a 10-mg dose 2 or 3 times weekly) for 3 months has been found to be effective. As always, precautions must be taken for females of childbearing potential who are receiving isotretinoin.⁹

In addition to oral antibiotics, topical clindamycin, topical erythromycin, topical metronidazole, and sodium sulfacetamide 10% and sulfur 5% lotion all have been used as part of the treatment regimen.^{9,37,40,43-46} There have been no studies directly comparing monotherapy with oral antibiotics versus a combination of oral and topical antibiotics in treating SIRD. In less severe cases, topical therapies can be used alone, while in severe cases, oral and topical antibiotics often are used concomitantly. Oral antihistamines, such as cetirizine or fexofenadine, and/or topical antipruritic agents, such as pramoxine hydrochloride, also can be prescribed for symptomatic relief.^{9,37,41}

More recently, topical calcineurin antagonists, such as tacrolimus and pimecrolimus, have been used in the treatment of SIRD. Compared with traditional treatments using oral and topical antibiotics, topical calcineurin antagonists may offer quicker initial improvement and more rapid eventual resolution of SIRD based on several small case series in the literature.^{22,24,37,47}

In children, the treatment of SIRD is similar to adults and involves cessation of topical steroid use as well as therapy with oral and topical antibiotics. As tetracyclines are contraindicated in children because of discoloration of teeth, oral erythromycin is the antibiotic of choice. A dosage of 30 mg/kg daily every 12 hours for 4 weeks has been recommended.^{9,48,49} There have been no case reports regarding the use of tacrolimus and pimecrolimus in the treatment of children with SIRD.

Conclusion

Steroid-induced rosacealike dermatitis is a symptomatic eruption composed of papules, pustules, papulovesicles, and sometimes nodules with telangiectatic vessels on a diffuse erythematous and edematous background. The disorder results from topical steroid use and can occur from prolonged use or as a rebound phenomenon after discontinuation of topical steroid. Both high- and low-potency topical steroids are known to cause the eruption. Diagnosis of this disease entity relies on a thorough patient history and

physical examination. Treatment involves discontinuation of the offending topical steroid and administration of oral and/or topical antibiotics. Topical calcineurin antagonists should be considered as alternative or adjunctive therapies for patients who do not respond to traditional treatments. Dermatologists may need to provide psychological support during office visits for patients who have difficulty dealing with the discontinuation of topical steroid and/or the psychological impact of a flare.

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