

Perioral Dermatitis and Sulfur

Steven L Harlan, MD FAAD

For decades, compounding dermatologists have employed sulfur with topical hydrocortisone acetate for chronic facial conditions like rosacea and seborrheic dermatitis. The addition of precipitated sulfur prevented the development of acneiform eruptions, rebound phenomenon, and perioral dermatitis from the hydrocortisone. Even after long term use, these compounds can be discontinued without rebound phenomenon.

There have been at least two epidemics of steroid induced perioral dermatitis after mid-potency corticosteroids were marketed for treating facial dermatitis. The first was betamethasone valerate 1962-1972, when dermatologists were first recognizing the steroid-induced-perioral dermatitis phenomenon. Another was early 1990s when 0.1% mometasone furoate was promoted as effective for seborrheic dermatitis. These events have contributed to many dermatologists' aversion to treating facial problems like seborrheic dermatitis, retinoid irritation, and rosacea with low potency topical steroids. Some of this related to "mixed fears from mixed discussions" about perioral dermatitis and topical corticosteroids.

For thoroughly understanding the complex subject of Perioral dermatitis, reviewing the dermatologic literature from 1964-1979 is extremely helpful.

Fluorinated topical steroids were introduced circa 1962. After 1964 there was described a new and different type of rosacea-like perioral dermatitis. Daryl S. Wilkinson in a 1979 review of Perioral dermatitis appearing in the British Journal of Dermatology pointed out that the prior usual treatments (pre-1960) for perioral and para-nasal eczematous and papulopustular eruptions (sulfur, ichthyol, and bland creams) failed dramatically in the treatment of the new version of perioral dermatitis. In fact, this new type of perioral dermatitis was extremely intolerant to these previous therapies.

Since this observation from a leading dermatologist in 1979, much knowledge about topical sulfur and perioral dermatitis therapy has been forgotten or dropped from discussions. Without reviewing that experience, the subject of Perioral dermatitis can be confusing and mischaracterized.

It's very helpful to lump together all spontaneous versions, and assert there are *two* kinds of Perioral dermatitis. One type is caused by prolonged use of naked topical steroids on the face, and the other type occurs spontaneously in children, teens and adults. The latter type has always been around, and it often was the reason the topical steroids were used and subsequently caused the steroid induced version of perioral dermatitis. The spontaneous type was often mild eczematous inflammation associated with early onset acne. Mixing these two historically distinct types in a discussion, contributes to poor understanding.

Prior to the advent of topical hydrocortisone in the 1950s, topical sulfur with bland creams and compresses were used to successfully treat the spontaneous forms of perioral dermatitis. Forms that could be related to: barrier problems, atopy, seborrhea, saliva, toothpastes, early rosacea, early acne in children, lower face acne in adult women, irritation from physical touching (hand, telephone, chin strap), and contact dermatitis. Wilkinson described many of these as paranasal dermatitis and paraoral eczematous eruptions in women.

Topical precipitated sulfur is a reducing agent with antimicrobial properties. Above 2% , sulfur preparations exhibit keratolytic properties. Below 2%, sulfur promotes epidermal thickening and barrier function. In the hands of dermatologists, and combined with bland creams, topical sulfur was so successful in treating these spontaneous facial eruptions that the term perioral dermatitis did not exist until 1964 when Mihan and Ayers first gave the disease its present name. By 1966 it was well known that the new version of perioral dermatitis reacted with intolerance and marked worsening when the topical steroid was *withdrawn* and the patient switched to sulfur and bland cream therapy.

The term “rebound phenomenon” became widely used by authors to describe the rapid intense worsening in erythema, burning, itching, and development of pustules that could occur when the potent topical steroid was withdrawn.

From the literature, it appears there was roughly a 10% incidence noted of the original topical hydrocortisone preparations in 1960 causing some acneiform lesions and rebound phenomenon on the face. These cases were managed by the addition of sulfur and continuing the use of hydrocortisone. But it was not until the advent of fluorinated steroids that the 1966 to 1972 epidemic occurred with the refractory virulent form of perioral dermatitis. Surprisingly, from 1966 through the 1970s, there was much disagreement about the etiology of the new form of perioral dermatitis. There was no mechanism to explain how a topical steroid causes inflammation, acneiform lesions, and rebound phenomenon. (There still is not.) Dermatologists were frequently misled by patients insisting they had the perioral problem ‘before’ the topical steroid was initiated. Experiencing the rebound phenomenon, patients would often blame whatever preparation had been substituted as the cause of their problems. Many dermatologists did not fully subscribe to the steroid induced etiology.

But it was the dramatic exacerbation of rosacea by fluorinated steroids that became the most convincing evidence. By 1972 dermatologist Gerhard Weber declared in publication that it was the topical steroids causing the epidemic of perioral dermatitis in Germany. It was not until the late 1970s that most dermatologists were in agreement that topical steroids were the primary cause.

The epidemic began to subside when most physicians fully understood that the fluorinated steroid must be withdrawn, and the patient treated with oral tetracycline 250mg b.i.d. (circa 1969-1972.) Author Ian Sneddon commented in 1976, “this does not seem to be purely an anti-microbial effect.”

Several authors recommended either topical 1% hydrocortisone acetate or 0.1% hydrocortisone butyrate used with oral tetracycline, immediately after discontinuing the potent topical steroid. This was done to mitigate the severity of the rebound phenomenon. The mild steroid was tapered off after a few weeks and the tetracycline continued for six to eight weeks minimum.

It was also reported that many patients improved by simply switching them from fluorinated steroid to either 1% hydrocortisone, or to 0.1% hydrocortisone butyrate. Some patients were able to clear the vicious cycle and get back into immunologic balance without the use of oral tetracycline.

From 1979 forward, many dermatologists used sulfur compounded with hydrocortisone acetate to successfully treat chronic facial conditions like rosacea and seborrheic dermatitis, without inducing perioral dermatitis. They also used this topical strategy to treat the spontaneous banal forms of perioral dermatitis, including those that frequently accompanied acne. Sulfur compounded with topical hydrocortisone could significantly reduce the resolution time of both types of perioral dermatitis, when oral antibiotics were employed. Less than two percent precipitated sulfur was frequently used for perioral and seborrheic dermatitis, and 2% to 5% precipitated sulfur for rosacea patients. When irritant dermatitis occurred from acne medications like retinoids, these compounds were very helpful and safely avoided the 10% risk of side effects seen with 0.1% hydrocortisone butyrate. This author learned to compound sulfur with low potency steroids for facial use from those dermatologists of the early 1980s.

When acne first develops in children, teens, and adult females, there can be a lower facial mildly eczematous, inflammatory component. Often these “sensitive” new acne patients have barrier problems or a background of seborrhea, atopy, or rosacea. These patients are at high risk for both irritation from acne medications, and perioral dermatitis from steroids. They must not be treated with fluorinated steroids or they will very quickly evolve from banal spontaneous perioral dermatitis to steroid perioral dermatitis.

It’s likely the early bad experiences with topical sulfur when the fluorinated steroid perioral dermatitis first appeared, contributed to the subject of topical sulfur being under-discussed in the perioral dermatitis literature. Mixing the two types of perioral dermatitis in discussions has likely contributed to confusion regarding treating the treatment of perioral dermatitis with topical sulfur/hydrocortisone compounds.

In conclusion, multi-factorial spontaneous perioral dermatitis and steroid perioral dermatitis are frequently seen in the same patient, but they should always be separated in discussions. Discussions with clearer separation of these two historically distinct clinical entities serve to place fears about steroids in perspective, and better help our patients.

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