Chapter 6
Topical Steroid Damaged Face in Females with Skin of Colour

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6.1 Introduction

Topical corticosteroids are among the most commonly used drugs in dermatology. The advent of topical corticosteroids is believed to have revolutionized dermatological therapy. Since the introduction of topical corticosteroids by Sulzberger and Witten in 1952 as Compound F (hydrocortisone), they have been utilized in a wide range of dermatological disorders owing to their anti-inflammatory, antiproliferative and immunosuppressive properties.

The introduction of hydrocortisone also opened up the avenue for the search of more potent molecules with more desirable properties. However shortly after topical corticosteroids were introduced, reports of adverse effects due to their usage started pouring in, the earliest of which were reported in 1955 [1]. Topical corticosteroids were substantially misused over the years which led to a plethora of adverse effects, both cutaneous and systemic, cutaneous or local side effects being more common than the systemic ones [2–4]. Earliest reports of the cutaneous side effects of topical corticosteroids emerged nearly a decade after they were first introduced [5]. The addictive potential of topical corticosteroids was however recognized later in 1970s and the term "steroid addiction" was used by Kligman and Frosh to describe the psychological and physical dependence on these drugs [6]. Face was a common site of adverse effects resulting from this addiction and abuse of topical corticosteroids. Owing to their anti-inflammatory and melanopenic properties, the use of topical corticosteroids on the face as cosmetic and particularly as bleaching

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agents or 'fairness' creams among the population with skin of colour became quite rampant [7]. Multiple studies from different parts of the world report skin lightening and acne as being the major reasons for indiscriminate and unsupervised use of topical corticosteroids on the face [8–12]. Over the years several terms like "red skin syndrome" [13], dermatitis rosaciformis steroidica [14] and steroid-induced rosacea like dermatitis (SIRD) [15] have been used to describe the condition that affects the facial skin due to topical corticosteroid abuse. The entity was labelled as "Topical steroid damaged/dependent face (TSDF)" by Kaushik Lahiri (India) in March 2008 and may be defined as follows: "Semipermanent or permanent damage to the skin of the face precipitated by the irrational, indiscriminate, unsupervised or prolonged use of topical corticosteroids resulting in a plethora of cutaneous signs and symptoms and psychological dependence on the drug." [7].

6.2 Epidemiology

While the exact incidence of TSDF is not known, it has been reported from several parts of the world [8–11, 16, 17]. While in developed countries, TSDF is usually seen consequent to prolonged or injudicious use of prescribed treatment for a steroid responsive dermatosis, the unregulated and non-prescription sale of topical corticosteroids in developing nations leads to misuse of these drugs for cosmetic reasons or steroid non responsive dermatoses [8, 9].

Females have been seen to be much more commonly affected than males in most of the studies, owing to greater cosmetic concerns in females with subsequent misuse of topical corticosteroids on face [8]. In skin of colour, the fairness obsession may be a major factor for misuse of topical steroids. Although any age group may be affected, TSDF has been seen most commonly in the age group of 20–30 years [8, 18, 19]. The misuse of topical corticosteroids in developing countries can be attributed to a number of factors which include

- Self-treatment.
- Prescription by non-dermatologists or even non medicos.
- Reuse of an old prescription for a recurrent or similar rash.
- Ease of availability of topical corticosteroids without prescription.
- Sharing of prescriptions among family and friends.

The lack of awareness among the general public regarding the hazards of self-medication with topical corticosteroids also perpetuates the problem.

6.3 Pathogenesis

Several theories have been put forward to explain the plethora of symptoms that occur in topical steroid damaged face.

1. **Role of nitric oxide**

   Prolonged and repeated application of topical corticosteroids to the face leads to inhibition of endothelial nitric oxide (NO) thus suppressing its vasodilatory effect. This results in a state of chronic vasoconstriction. Upon withdrawal of topical corticosteroids, endothelial nitric oxide is released causing vasodilation and erythema accompanied by itching and burning sensation. Reapplication of corticosteroids for alleviation of these symptoms further leads to vasoconstriction. This repeated cycle of vasoconstriction/vasodilation known as the 'trampoline effect' or 'neon sign' leads to the accumulation of nitric oxide. Release of this accumulated endothelial nitric oxide eventually leads to dilatation of cutaneous vasculature even beyond the original diameter with resultant erythema and flushing [13].
2. **Topical corticosteroid-induced immunosuppression**

   This results in the overgrowth of microorganisms which act as superantigens once the topical corticosteroids are withdrawn and induce an inflammatory reaction, causing the release of proinflammatory cytokines in the skin [20]. The role of *Demodex folliculorum* in the pathogenesis of TSDF is controversial. Bonnar et al. reported a significantly increased density of *Demodex folliculorum* in patients with steroid induced rosacea like dermatitis [21]. It may either cause an inflammatory or allergic reaction by blocking the hair follicles or act as vectors for other microorganisms [15].
3. **Direct effects on follicular epithelium**

   Degradation of follicular epithelium by topical corticosteroids with resultant extrusion of follicular contents is also postulated as a pathogenetic mechanism for occurrence of acneiform lesions in topical steroid damaged face [18].
4. Inhibitory effect of topical corticosteroids on keratinocyte proliferation, collagen synthesis as well as fibroblast and hyaluronic acid synthesis in the extracellular matrix leads to cutaneous atrophy. The lack of support to the vasculature due to dermal atrophy also aggravates the erythema in TSDF [22, 23].

   The pathogenesis of TSDF is summarized in Fig. 6.1.

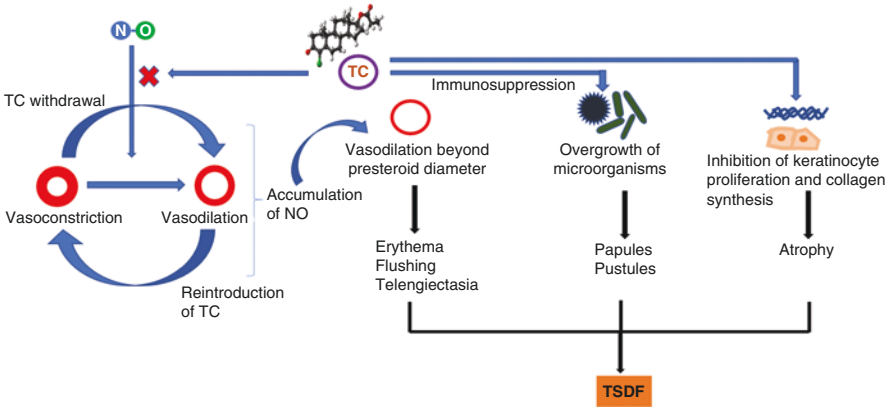

Fig. 6.1 Pathogenesis of Topical Steroid Damaged Face (TSDF), (*TC* Topical corticosteroid; *NO* Nitric oxide)

6.4 Clinical Presentation

Topical steroid damaged face presents as a symptom complex resulting from the numerous adverse effects of topical corticosteroid usage over face. Chronic abuse of topical steroids leads to psychological and physical dependence on the drug. Any attempt to withdraw the drug is faced with exacerbation of the symptoms which compels the patient to restart corticosteroid application to alleviate the symptoms [7]. While most patients develop symptoms after short term usage of potent topical corticosteroids, long term usage of less potent or mild steroids can lead to similar symptoms. Most patients with topical steroid damaged face give a history of topical corticosteroid usage for a prolonged period of time and symptoms may occur either after long-term continued usage or as a withdrawal/rebound phenomenon once the incriminating topical corticosteroid is discontinued.

Various presentations of topical steroid damaged/dependent face seen either alone or in combination include:

1. **Erythema**

   Erythema is a hallmark manifestation of TSDF which becomes more evident upon topical corticosteroid withdrawal. A diffuse erythema accompanied by a burning sensation (which may range from mild to severe) is noted in most patients with topical steroid damaged face. Following topical corticosteroid withdrawal, the erythema usually resolves in about 2 weeks followed by desquamation. Erythema however reappears within the next 2 weeks. These intermittent flares of erythema may also spread beyond the area of corticosteroid application or even to distant sites. The "headlight sign" is often present where nose and the perioral area is spared from erythema which otherwise is present over whole of the face. Erythema may be accompanied by oedema or vesiculation in severe cases. A cycle of flare ups and resolution sets in but the intensity of flares goes on decreasing and the phases of resolution are progressively prolonged till complete resolution occurs [13]. The length of time for which the steroids have been initially used determines the duration of the withdrawal phase.

   While erythema is the principal clinical feature in TSDF, it may be less pronounced in dark skinned individuals where other associated features like dryness, scaling and itching may indicate withdrawal phase or subsequent flare-ups in TSDF. (Fig. 6.2)
2. **Telangiectasia**

   Dermal atrophy in topical steroid damaged face leads to a diminished connective tissue support allowing passive dilatation and easier visualization of dermal capillaries which manifests as prominent telangiectasias [24]. (Figs. 6.3 and 6.4)
3. **Papulopustular lesions**

   Papulopustular and acneiform eruptions are frequently associated with the vascular changes in topical steroid damaged face. This has been attributed to the focal degeneration caused by direct effect of topical corticosteroids on the fol-

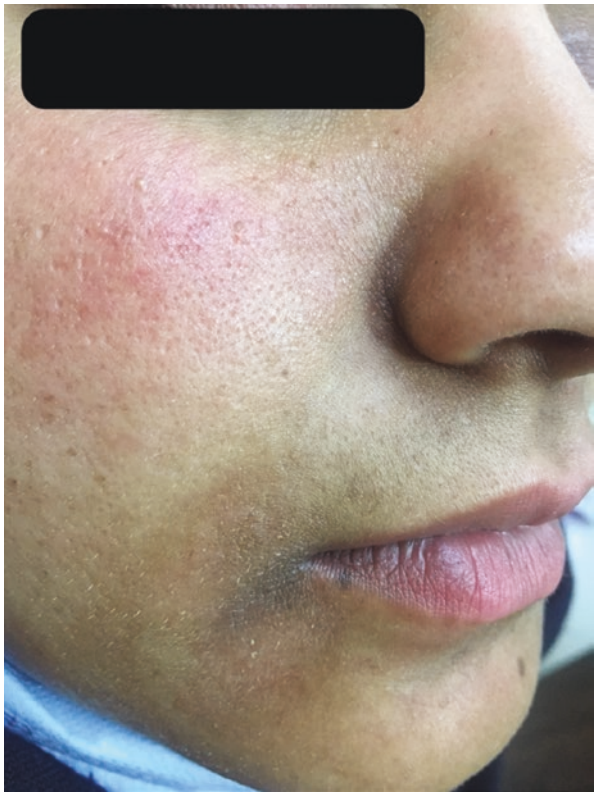

Fig. 6.2 Erythema and hyperpigmentation

licular epithelium leading to localized intrafollicular and perifollicular neutrophilic inflammatory reaction [22]. Topical steroids may also induce comedone formation by rendering the follicular epithelium more responsive to comedogenesis [25, 26]. This however is more common with systemic steroids.

The papulopustular lesions are usually monomorphic in appearance and have an unusual distribution. A rosacea-like dermatitis is commonly seen. Initial lesions may be small, pinpoint erythematous or skin coloured papules, pustules or papulovesicles. On further application of topical steroids, patients may develop diffusely erythematous and oedematous skin with deep follicular papules, pustules and nodules. The lesions may be distributed diffusely or may be localized mainly to the perioral region (resembling perioral dermatitis) and centrofacial area including the cheeks, nose, forehead and glabella [24]. (Figs. 6.5, 6.6, and 6.7)

4. **Atrophy**

A variable degree of skin atrophy is usually associated with other manifestations of topical steroid damaged face. Atrophy of the skin is reflected in the form of increased transparency and shininess of the skin (Fig. 6.8).

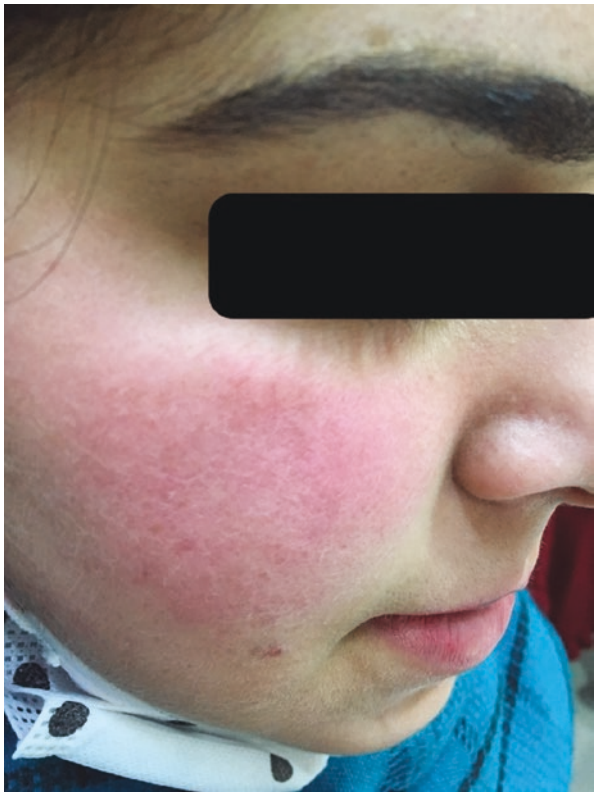

Fig. 6.3 Topical steroid induced telangiectasia

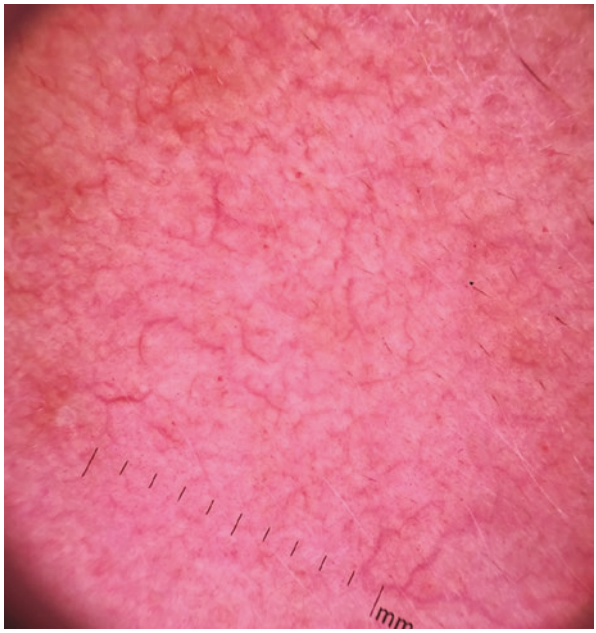

Fig. 6.4 Dermoscopy of Fig. 6.3 showing short, linear, irregular vessels

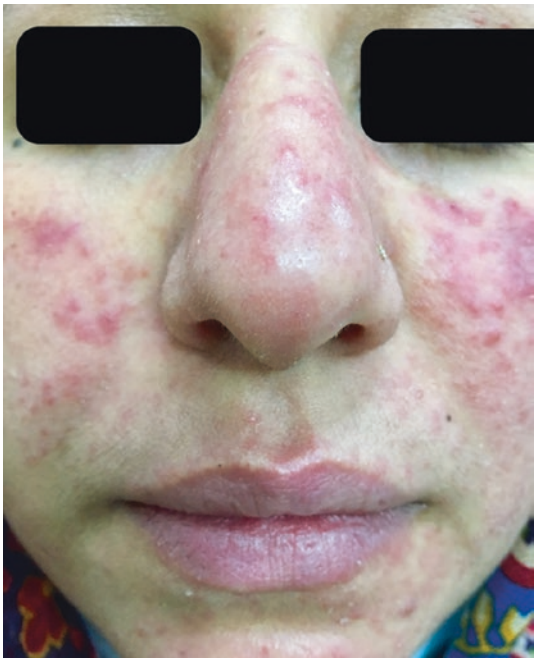

Fig. 6.5 Topical steroid induced papulopustular lesions

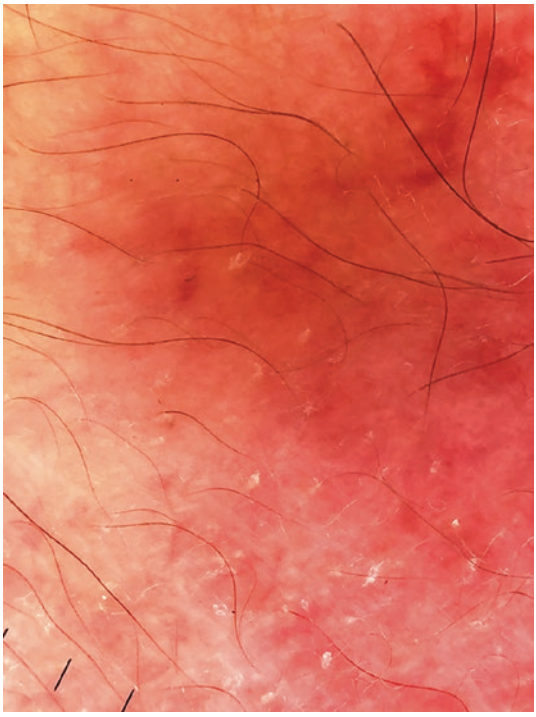

Fig. 6.6 Dermoscopy of Fig. 6.5 showing demodex tails, perifollicular erythema and hypertrichosis

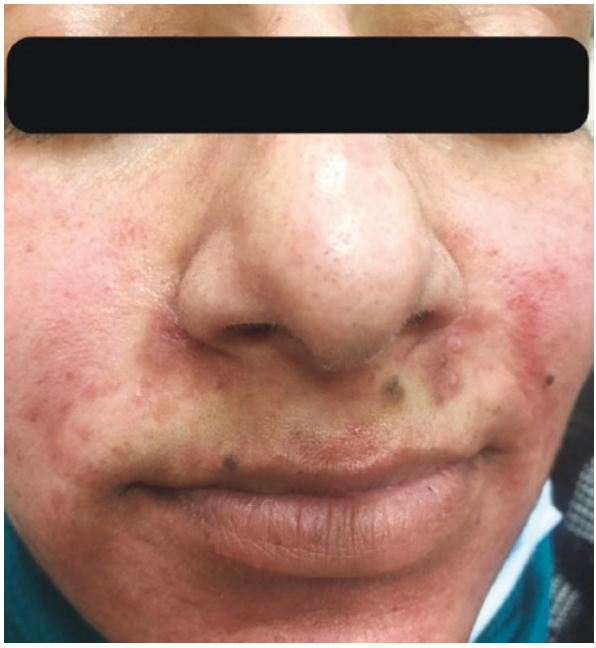

Fig. 6.7 Perioral dermatitis

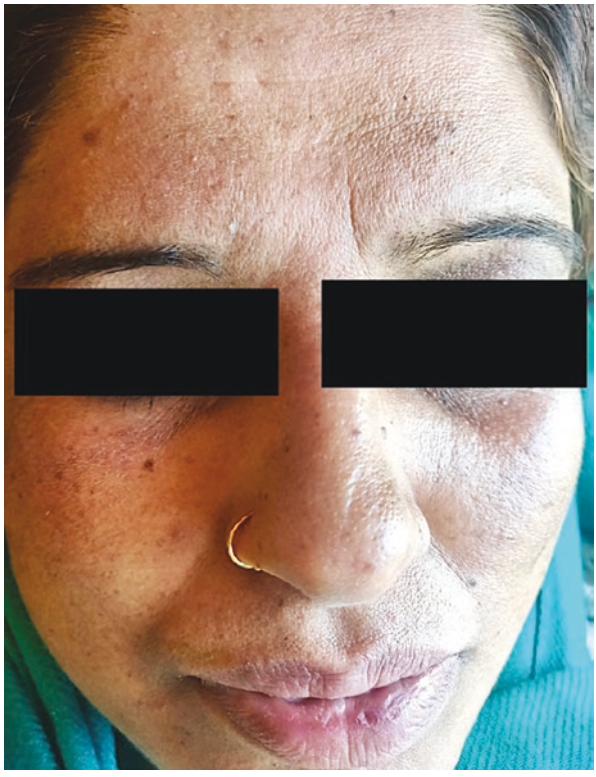

Fig. 6.8 Topical steroid induced atrophy and dyspigmentation

5. **Pigmentary changes**

   Topical corticosteroids are known to cause both hyperpigmentation and hypopigmentation. In people with skin type IV to VI, pigmentary changes especially in the form of hypopigmentation may be a prominent feature of topical steroid damaged face [12, 27]. Topical steroid–induced hypopigmentation is brought about by inhibition of prostaglandin or cytokine production in the epidermal cells which in turn leads to suppression of secretory metabolic products from melanocytes resulting in altered melanocyte function [28]. The mechanism of topical steroid induced hyperpigmentation is not well elucidated but hyperpigmentation has been reported in several cases of topical corticosteroid abuse [29, 30].

6. **Hypertrichosis**

   Hypertrichosis is a common side effect of systemic steroid use but many patients with topical corticosteroid abuse have abnormal facial hair growth.

7. **Other features**

   In addition to the aforementioned ones, topical steroid damaged face can present with several other features like photosensitivity, striae, allergic contact dermatitis and tinea incognito [31] (Figs. 6.9 and 6.10).

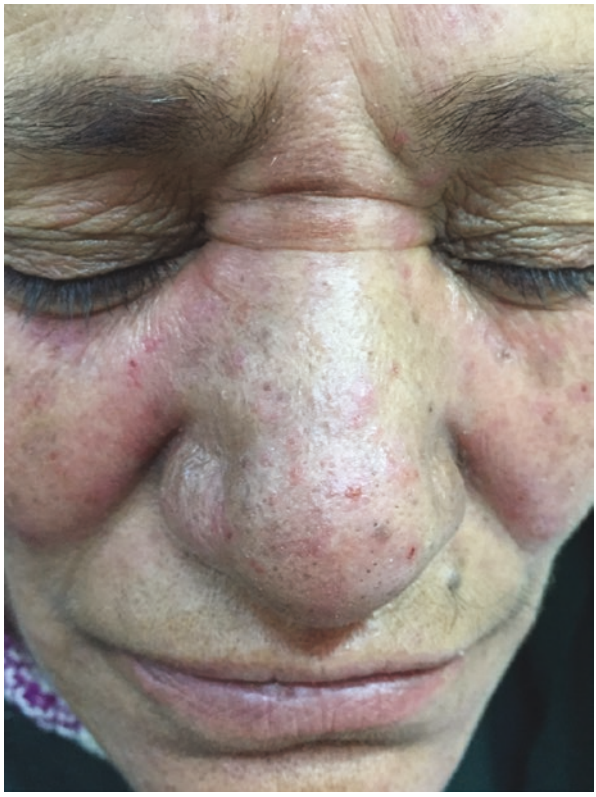

Fig. 6.9 Topical steroid induced dermatitis-rebound phenomenon

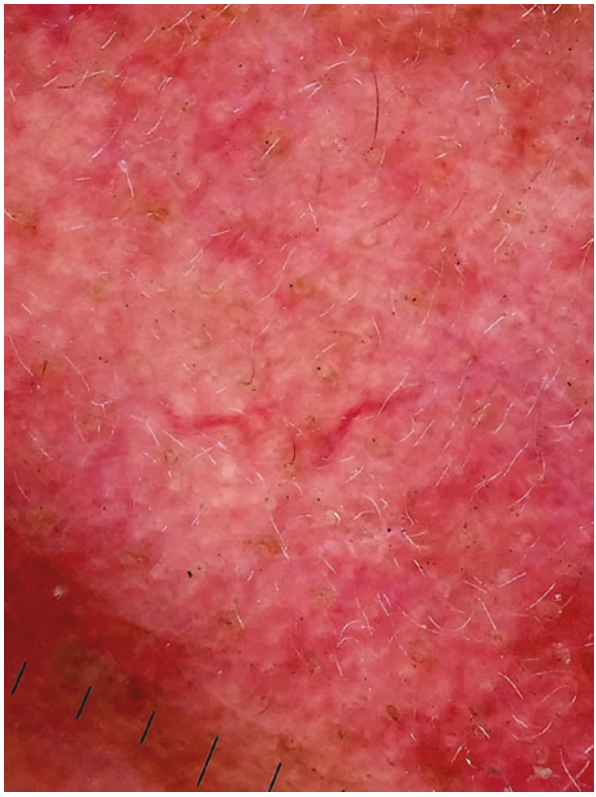

Fig. 6.10 Dermoscopy of Fig. 6.9 showing background erythema, yellow clods and short branching vessels

8. **Status cosmeticus**

   The term was first used by Fisher to describe an extreme form of cosmetic intolerance in which the patient gradually becomes completely intolerant to application of any form of makeup or cosmetic product [32]. Status cosmeticus was originally described in relation to facial dermatoses like atopic dermatitis, seborrheic dermatitis, rosacea and perioral dermatitis. Some patients with TSDF may exhibit symptoms similar to status cosmeticus where application of any cosmetic product to the face leads to severe irritation, erythema and burning sensation.

9. **Dermoscopic features of topical steroid damaged/dependent face**

   Various dermoscopic features suggestive of TSDF include the following [33].

   - Diffuse red areas with multiple, tortuous, polygonal and interconnecting blood vessels.
   - Ivory white, structureless areas to strawberry coloured patches suggestive of dermal atrophy.
   - Yellowish areas suggestive of epidermal atrophy with increased skin transparency.
   - Lesional hypertrichosis.

6.5 Management

Management of TSDF is quite difficult and requires patience on part of the patient as well as the treating physician. Treatment involves use of medication as well as patient counselling. Once the steroids are discontinued, a flare of symptoms is expected in all patients. Patients may develop severe erythema and burning sensation. Thus reluctance on part of the patients to stop corticosteroid application is expected. While some authorities advocate gradual withdrawal of corticosteroids,[15, 34, 35] others are in favour of complete cessation of topical corticosteroid usage on first contact with the patient [36, 37]. Although replacement of the potent topical steroid by a low potency one may alleviate the concerns of severe flares upon topical steroid withdrawal and make the patient more comfortable, some authorities believe that this just perpetuates the problem and hinders a full recovery [36].

Once the topical steroids are withdrawn, a total cure is expected in 6–24 months (with intermittent flares and remissions) without the use of any medication.

Various treatment modalities employed in the treatment of TSDF include:

1. **General measures**

   Repeated cold compresses help alleviate symptoms of topical corticosteroid withdrawal and those of acute flares. Soap free cleansers, bland emollients and calamine may help improve therapeutic outcome in patients with TSDF [29]. Burrow's solution may be used for weepy lesions [13].
2. **Antibiotics**

   In patients with papulopustular lesions, perioral dermatitis and rosacea like features, oral antibiotics with anti-inflammatory properties play a prominent role in the treatment. The preferred antibiotics include lipophilic tetracyclines like doxycycline and minocycline in a dose of 100–200 mg daily and oral azithromycin in a dose of 500 mg given on 3 days a week for 3–4 months. Longer duration of treatment is usually not needed. In patients who are unable to tolerate tetracyclines, oral metronidazole has also been used [38].

   In addition to the oral antibiotics, topical clindamycin, topical metronidazole, topical erythromycin and topical 5% sulphur have also been used in topical steroid induced rosacea and perioral dermatitis like lesions [38–40]. In mild cases of TSDF, topical antibiotics alone may be helpful.
3. **Oral isotretinoin**

   In patients not responding to antibiotics, a very low dose of isotretinoin (2–5 mg daily) for 3 months may be effective [38].
4. **Topical calcineurin inhibitors**

   Topical calcineurin inhibitors like tacrolimus and pimecrolimus have been found to be safe, effective and well tolerated in patients with TSDF. They may be used alone or in combination with oral antibiotics and have been found to offer a quicker initial improvement during the withdrawal phase in TSDF and the eventual resolution of symptoms may also be more rapid [41–43].

5. **Alpha adrenergic agonists**

   α adrenergic agonists like brimonidine, oxymetazoline and xylometazoline act as topical vasoconstrictors and help by reducing flushing and erythema associated with TSDF. Brimonidine tartrate in addition to being vasoconstrictive may also possess anti-inflammatory properties to some extent. Brimonidine 0.33% cream, xylometazoline 0.05% solution or oxymetazoline 0.05% solution applied once daily for variable durations have been shown to significantly improve erythema and flushing in sporadic cases [44, 45].

6. **Other treatments**

   (i) Oral and topical vitamin C and E.

   These have been used in patients with TSDF showing some improvement in vascular symptoms [29].

   (ii) Topical vitamin K. Application of 1% topical vitamin K has been found to be effective in an isolated study where rapid improvement in flushing, erythema and dryness was observed after 4 weeks of treatment [46].

   (iii) Ivermectin 1% cream.

   Ivermectin is a broad-spectrum antiparasitic agent which has been shown to significantly improve papulopustular lesions in rosacea as well as rosacea like dermatitis induced by topical steroids. It is known to act as an anti-inflammatory agent and also has antiparasitic properties against *Demodex folliculorum* [47].

   (iv) Platelet-rich plasma (PRP).

   PRP mesotherapy in the form of intradermal injections was found to be effective in the treatment of TSDF in an isolated study [48].

   (v) Lasers: Intense pulse light and pulsed dye laser have also been used in recalcitrant cases of TSDF and have been found to improve erythema and telangiectasias significantly [49, 50].

6.6 Conclusion

Topical corticosteroids are the cornerstone of treatment for a number of dermatological disorders owing to their anti-inflammatory, antiproliferative and immunosuppressive properties. However, indiscriminate and unsupervised use of topical corticosteroids can lead to a plethora of local side effects especially on the face where the skin is thin and corticosteroid absorption is enhanced. The problem is amplified manifold in females with skin of colour who in an attempt to meet the culturally sanctioned ideals of beauty, tend to misuse topical corticosteroids for skin lightening. The resulting condition in the form of topical steroid damaged/dependent face is very difficult to treat and can lead to irreversible damage to the skin. Considering the magnitude of the problem and owing to the physical, psychological and financial burden of the condition, it is immensely important to raise sufficient

public awareness regarding the ill effects of steroid abuse on face and at the same time limit the unregulated public access to potent topical corticosteroids.

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