Impetigo Incognito – A Case Series of Steroid Modified Impetigo

Dear Editor,

Impetigo is a common cutaneous bacterial infection caused by *Staphylococcus aureus* or *group A ß-hemolytic streptococci*. The widespread over-the-counter availability and use of topical corticosteroid (TCS) combination creams over the past few years have led to the emergence of modified impetigo lesions, referred to as impetigo incognito.

We report a series of eight children from rural India who presented with modified impetigo (impetigo incognito) after the application of topical agents containing corticosteroids. The clinical details are enlisted in Table 1. All the children had applied either topical steroid creams alone or in combination with topical antibiotics. The clinical morphological patterns observed were eczematous, circinate, and mixed patterns [Figures 1-4]. Gram staining and culture performed from the lesions showed *Staphylococcus aureus* in six patients and *Streptococcus pyogenes* in two patients. The other differential diagnoses were ruled out based on relevant history and examination with appropriate investigations. All patients were treated with oral and topical antibiotics based on their antimicrobial sensitivity.

Impetigo is a common bacterial infection of high burden in childhood in low- and middle-income countries, contributing to more than 162 million cases at any one time.[1] According to a systematic review by Bowen *et al*., the median prevalence of impetigo in the general population and children was found to be 11.2% and 12.3%, respectively.[1] Over a 45-year interval, the impetigo burden has remained relatively unchanged, showing limited progress in the control of impetigo in impoverished settings.[1] But the incidence of impetigo has increased in developed countries like the United Kingdom as evidenced by a study done by Shallcross *et al*.[2]

In a study assessing topical steroid misuse, 80% of people in rural India abused TCS obtained over-the-counter for various dermatological conditions.[3] In this context, one of the diagnostic challenges in impetigo is identifying previously partially treated cases, especially those mismanaged by topical steroids or topical immunomodulators like tacrolimus or pimecrolimus. The

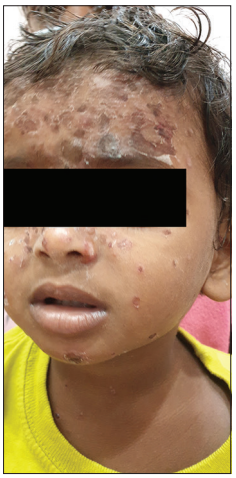

Figure 1: Areas of desquamation with underlying brownish pigmentation present over the forehead, and discretely scattered small erosions with minimal crusting over the central part of the face

Table 1: Demographic, clinical and treatment details of cases

Age	Gender	Duration (from the onset of lesions to presentation)	Topical application	Duration of topical application	Mode of acquisition of topical agent	Presentation pattern (s)	Follow-up (No. of days taken for complete resolution of lesions)
3 years	Male	10 days	Betamethasone valerate + fusidic acid	7 days	Over-the-counter	Eczematous	6 days
5 years	Female	8 days	Beclomethasone dipropionate + fusidic acid	5 days	General practitioner	Circinate	5 days
3 years	Male	5 days	Mometasone furoate	4 days	Over-the-counter	Eczematous	5 days
8 years	Male	7 days	Clobetasol propionate + gentamicin	7 days	Over-the-counter	Mixed eczematous and circinate	6 days
6 years	Male	12 days	Betamethasone valerate + fusidic acid	9 days	Over-the-counter	Circinate	6 days
6 years	Female	6 days	Betamethasone valerate	5 days	General practitioner	Eczematous	4 days
4 years	Male	10 days	Beclomethasone dipropionate + clotrimazole + neomycin sulphate	8 days	Over-the-counter	Circinate (with Id eruption)	7 days
7 years	Male	12 days	Betamethasone valerate + fusidic acid	7 days	General practitioner	Circinate	6 days

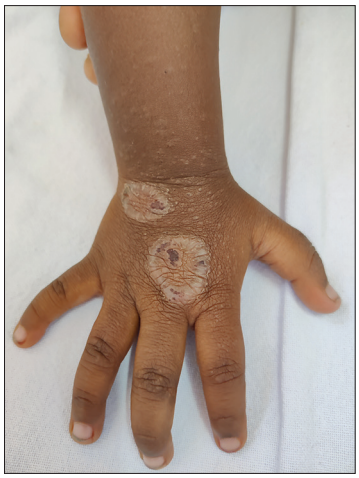

Figure 2: Two skin-coloured annular plaques with raised borders, minimal central crusting, and surrounding ld eruption over the dorsum of the right hand

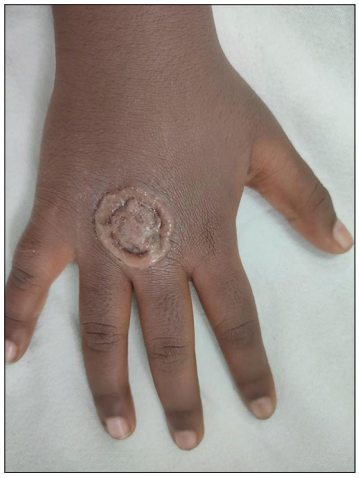

Figure 3: Single skin-coloured erythematous crateriform plaque with raised edematous margin over the dorsum of the right hand

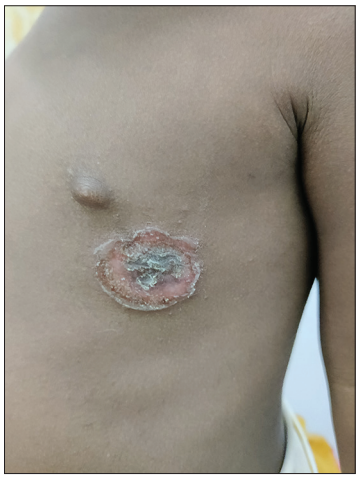

Figure 4: Single annular crusted plaque below the left nipple

last two decades have seen a boom in topical steroid and topical antibiotic combinations and rampant use of the same. In certain Indian studies, topical steroid usage in infectious disorders ranged from 0.3% to 3.6%.[4,5]

According to some studies, topical steroids are known to disrupt the integrity of the stratum corneum, including abnormalities in lipid biosynthesis. This leads to increased transepidermal water loss (TEWL), that in turn triggers the production of pro-inflammatory cytokines like tumour necrosis factor alpha (TNF-α), interleukin (IL)-1, and IL-6, causing inflammation and increased proliferation of the epidermis.[6] The crateriform appearance of lesions as observed in our cases could be probably attributed to the epidermal proliferation induced by the cytokines.

Glucocorticoids are known to suppress macrophage differentiation and its microbicidal action and antagonize the maturation of dendritic cells and their function, leading to poor antigen cell presentation to naïve T cells.[7] This produces a state of immunosuppression leading to the overgrowth of bacterial colonies. Chan *et al*. studied the effect of a topical steroid (triamcinolone) on bacterial flora in the skin and found that the average total microbial counts increased from $3.4 \times 10^2/cm^2$ to $7.4 \times 10^5/cm^2$. They also observed individual rise in colony counts of *Staphylococcus aureus*, *coagulase-negative staphylococci*, *Micrococci*, *Streptococci,* and lipophilic diphtheroids.[8]

Other possible reasons for the worsening of infections because of topical steroids include dawdling mobilization of defenses due to vasoconstriction, reduced epidermal turnover with diminished shedding of scales and surface organisms, increased hydration of stratum corneum, and weakened activity of antimicrobials.[9]

It was found by Wolfgang P. Raab using the Warburg technique that fusidic acid and hydrocortisone combination caused less inhibition of bacterial oxygen consumption than fusidic acid alone. Moreover, antimicrobials and TCS were found to share the same targets, enzymes, membrane structures, and functions resulting in competitive inhibition and culminating in decreased antimicrobial efficacy.[10]

Clinically, erythema is one of the earliest signs observed in impetigo lesions. Erythema was absent in all the patients probably due to the anti-inflammatory effects of TCS. The anti-inflammatory properties of TCS can be due to the reduced number and antigen-presenting function of Langerhans' cells, decreased phagocytic and anti-bacterial capabilities of polymorphonuclear leukocytes, decreased natural killer cell activity, and diminished antibody-dependent cellular cytotoxicity of lymphocytes. TCS also augment vasoconstriction, thereby decreasing vascular permeability.[11]

The application of TCS to impetigo has also led to altered morphology such as eczematous and circinate appearances leading to diagnostic dilemmas and delayed resolution of clinical lesions. The management of impetigo incognito begins with proper counselling regarding the effect of TCS-containing medication on impetigo and the need for discontinuation of the same, followed by appropriate preliminary investigations such as Gram staining. However, bacterial culture and sensitivity

Table 2: Salient features of Impetigo incognito

Features
Use of topical immunosuppressants
Absence of systemic immunosuppression
Absence of inflammation
Persistence of lesions
Asymptomatic

remain the gold standard of diagnostic modality. The features suggestive of impetigo incognito as observed by the authors are mentioned in Table 2.

The differential diagnosis includes circinate impetigo, linear IgA disease, erythema multiforme, discoid eczema, and tinea for the circinate pattern and allergic contact/irritant contact dermatitis, seborrheic dermatitis, herpes labialis, and atopic eczema for the eczematous pattern.

Though steroid and antibiotic combination can be helpful for primary inflammatory diseases like atopic eczema where *Staphylococcus aureus* produces super antigens accelerating inflammatory response and inducing corticosteroid resistance, it is not advisable for primary infectous diseases like impetigo and dermatophytosis. Irrational use of antibiotic and steroid combination creams also predisposes the patient to contact dermatitis and anti-microbial resistance. It requires a sincere effort from the various governmental agencies to curb the over-the-counter availability of TCS and to raise awareness among patients to put an end to this menace.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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