Review Article

The unprecedented epidemic-like scenario of dermatophytosis in India: I. Epidemiology, risk factors and clinical features

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Abstract

Dermatophytosis has attained unprecedented dimensions in recent years in India. Its clinical presentation is now multifarious, often with atypical morphology, severe forms and unusually extensive disease in all age groups. We hesitate to call it an epidemic owing to the lack of population-based prevalence surveys. In this part of the review, we discuss the epidemiology and clinical features of this contemporary problem. While the epidemiology is marked by a stark increase in the number of chronic, relapsing and recurrent cases, the clinical distribution is marked by a disproportionate rise in the number of cases with tinea corporis and cruris, cases presenting with the involvement of extensive areas, and tinea faciei.

Key words: Fixed-dose combination creams, superficial dermatophytosis, tinea, topical steroids

Introduction

We aim to comprehensively cover various aspects of the current epidemic-like scenario of dermatophytosis in India. The disease has attained a significant magnitude and is now being observed in other distant nations as well[1,2] often as an extensive and difficult to treat entity with diverse morphology. Recalcitrant superficial dermatophytosis has the potential to become a public health issue in many parts of the world if we do not make an active attempt to understand it in its entirety, take timely measures, and involve public health agencies and the World Health Organization.

This review intends to concentrate on the commonest clinical types of dermatophytoses in the current scenario and will not deal with all clinical variants (e.g., tinea unguium, tinea pedis, tinea capitis). A comprehensive English language literature search was performed across multiple search engines (PubMed, EMBASE, MEDLINE and Cochrane). MeSH terms ("tinea," " Dermatophytosis," "epidemiology," "clinical features," "diagnosis of dermatophytosis,""antifungal resistance," "topical" and "systemic") alone and in combination were considered. Available literature pertaining to dermatophytosis in the last 7 years (2014–2020) with particular emphasis on Indian studies was included.

The phenomenon under review is recent and treatment options are still evolving. Studies on it are lacking, and we have included many previously undescribed but now common clinical observations of the authors and a large number of dermatologists in India. There is a strong need to study this entity and its various treatment options in greater detail.

This review is divided into three parts: Part I covers epidemiology, risk factors and clinical features; Part II will

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discuss diagnostic methods and taxonomical aspects of the causative dermatophytes of this outbreak; Part III will deal with antifungal resistance, a very important feature of this current phenomenon, and prevailing treatment options.

Epidemiology of dermatophytosis in India

Superficial dermatophytosis affects 20-25% of the world population and is a common infective dermatoses in clinical practice.[3] What was once considered an innocuous, easy-to-treat infection of tropical and subtropical countries, mainly seen during the summer and rainy seasons, has now become a perennial and difficult to treat entity in India. According to recent studies, there has been an increase in the incidence of dermatophytosis across the country in the past decade and especially so over the past 5–6 years.[4,5] This increase has been at an alarming rate and has resulted in an epidemic-like situation in the country.

Incidence and prevalence

Though there have been many studies on superficial dermatophytosis, it is difficult to calculate the exact incidence and prevalence owing to a paucity of community-based surveys. The current reported prevalence in India falls in a very wide range (6.09%[6]– 61.5%[7]). A prevalence of 6.09% to 27.6% has been reported in studies from south India,[6,8] while a high prevalence of 61.5% has been recorded in north India.[7] Most of this data comes from hospital-based studies over periods ranging from 1- 2 years[6–8] Interestingly, though dermatophytosis is expected to be more prevalent in the hot and humid climates of south India and less so in north India, no such association is apparent. We feel that there has been a rising trend of dermatophytosis all over the country in the last 5–7 years.

Further, there appears to be a steady increase in the incidence of chronic, relapsing and recurrent dermatophytosis; it is not uncommon to encounter disease durations running into months or years. Agarwal *et al*. in 2014 reported disease of >3 months in 163/300 patients (62.5%).[9] Of the 300 patients, 28 (9.3%) had had similar episodes in the past and 87 (27.7%) patients had been treated previously with medications other than antifungal drugs. Since then, there has been a rising trend of recurrent and relapsing dermatophytosis [Table 1].[9-16] These trends are unique to the present epidemic-like situation of dermatophytosis in India and are not described in the standard dermatology textbooks from the Western world.

Age and sex

In general, dermatophytosis occurs most frequently in postpubertal hosts, with the exception of tinea capitis. Conventionally, men are more frequently affected than women because of the significantly high incidence of tinea cruris, tinea pedis and tinea unguium in men,[17] and outdoor work predisposes men to hot, humid and sweaty conditions conducive to the growth of dermatophytes. However, in the present epidemic, these boundaries are getting blurred with the rising incidence of dermatophytosis in women and children [Table 2].[7,12-15,18-25] It is clear that the male:female ratio is < 2 in all the studies published in the last 3–4 years[12-14,18-20] in contrast to earlier studies where the ratio was 3–5, indicating a rising prevalence in women.[24,25] Recent studies have even shown the reversal of the men:women ratio.[13,19] As most Indian studies on superficial dermatophytosis have included only adult patients, the exact incidence of cases among children cannot be ascertained. Only a single study by Noronha *et al*. from south India has included patients of all age groups.[26] This study reported that 21.3% of patients were in the age range of 0–10 years, similar to the proportions of adolescents and adults (21.3% and 22.7% in the age range of 11–20 years and 21–30 years, respectively).[26] In our experience, it is not uncommon to see infants, toddlers and children of all age groups presenting with all morphological types of dermatophytosis, viz., extensive tinea cruris, tinea corporis and tinea faciei.

Chronic dermatophytosis (disease for more than 6 months with or without recurrence despite being treated) is more common in the late middle age group, around fifth decade, which is attributed to waning immunity, comorbidities like diabetes and other risk factors such as positive family history, use of topical steroid-antifungal creams, immunosuppressants intake etc.[14] However, in a recent study on 150 patients of recurrent dermatophytosis, the mean age of patients with chronic dermatophytosis was lower (32.5 years).[12]

Familial cases

Recent studies have reported a very high frequency of history of dermatophytosis in close contacts (72-82%)[11,12,19] Chronicity and recurrent infection have probably contributed to the rise in the incidence of familial infection. Connubial dermatophytosis is common.[5] The spouse and children, often the whole family, have concomitant dermatophytosis. A high prevalence occurs in people living in overcrowded homes, slums, hostel rooms and dormitories. A careful inquiry into family history has become the norm; all affected family members must be treated simultaneously to prevent recurrences.

Urban versus rural areas and literacy

People from both urban and rural areas are at increased risk of dermatophytic infections.Studies from the first half of the past decade reported a rural predominance, possibly due to the high frequency of outdoor work including agriculture predisposing to increased perspiration.[6,27] However, studies in the last 5 years have shown greater proportions of patients from urban areas (around 80% of patients).[7,22] This change was initially attributed to increased awareness, literacy and cosmetic concerns in the urban population leading them to seek medical attention. However, the predominance seems to be dependent on the place and nature of the study. The relatively easy availability of creams containing irrational

Table 1: Prevalence of relapse and recurrence in superficial dermatophytosis[7-14]

Author and year	Number of patients (*n*)	Place of study	Relapse/recurrent SD (%)
Agarwal *et al*., 2014[9]	300	Jaipur (North India)	Disease duration >3 months - 62.5% Relapse - 9.3%
Sharma *et al*., 2017[10]	192	Sikkim (East India)	Relapse - 60.4% Recurrent - 34.3%
Mahajan *et al*., 2017[11]	265	Varanasi (North India)	Disease duration of 2 years - 35.8%
Pathania *et al*., 2018[12]	150	Chandigarh (North India)	Recurrent - 9.3% (multiple recurrences, (3–10) usually within a few days to 4 weeks of stopping treatment)
Vineetha *et al*., 2018[13]	120	Kottayam (South India)	Chronic - 68%
Tigga *et al*., 2018[14]	300	New Delhi (North India)	Reinfection - 49% Recalcitrant - 44.3%
Rudramurthy *et al*., 2018[15]	195	Chandigarh (North India)	Recurrent - 60%
Singh *et al*., 2019[16]	150	Varanasi (North India)	Chronic - 65.3% Relapsing - 34.6%

Table 2: Trends of superficial dermatophytosis with respect to age and sex in the last 5 years[5,10-13,16-23]

Author and year	Total patients (all adults)	Men: women	Age range
Hazarika *et al*., 2019[18]	130	1.5:1 (60%:40%)	21–30 years (32%) 31–40 years (23%)
Patro *et al*., 2019[19]	294	0.88:1 (47%:53%)	18–40 years (86.7%) f/b 41–60 years (13.2%)
Vineetha *et al*., 2018[13]	120	1:1.1	1st episode - 10–20 years Chronic - fourth to fifth decade
Tigga *et al*., 2018[14]	300	1.97:1	15–30 years (66.3%)
Pathania *et al*., 2018[12]	150	1.7:1 (63%:37%)	Mean age - 32.5 years
Rudramurthy *et al*., 2018[14]	195 Children - 7 (3.6%) Neonates - 4 (2%)	2.8:1 (73.8%–26.1%)	Median age - 33.5 years
Dabas *et al*., 2017[20]	124	1.7:1 (64%–36%)	Mean age - 31.2 years
Ramaraj *et al*., 2016[21]	210	4:3 (57.1%:42.8%)	21-40 years (49%) f/b 41-60 years (29%)
Poluri *et al*., 2015[22]	110	2:1 (67.2%–32.7%)	21–40 (64.5%)
Kaur *et al*., 2015[7]	351	2:1 (67.2%:32.7%)	21–30 years (23.3%) f/b 31–40 years (20.5%)
Maulingkar *et al*., 2014[23]	321	2.4:1	21–30 years (26.5%) 31–40 years (35.5%) Low prevalence - <10 and>50 years
Bhatia and Sharma, 2014[24]	202	5.7:1	21–50 years (64.9%)
Bhagra *et al*., 2014[25]	100	4:1	Third decade (28%)

combinations of potent topical steroids along with antifungal and antibacterial agents in urban areas as compared to rural might be a contributing factor.

Education and literacy have not been commented upon much in reports concerning the increased incidence of dermatophytosis. Kaur *et al*.[7] reported the frequency of literate patients (64.6%) to be more than the illiterates (35.3%). Similarly, a recent study showed that the majority of their patients had medium educational qualifications (60.20%).[19] These findings can also perhaps be interpreted as above.

Socioeconomic status

A higher proportion of patients with dermatophyte infections are still from lower socioeconomic groups, with studies reporting an incidence of 61–67%.[6,22,26] This is followed by lower-middle and medium socioeconomic strata.[9,19,26] Poor standards of living, lack of hygiene, overcrowding and poor nutrition in the lower socioeconomic groups promote the growth of dermatophytes, increasing the risks of infection, chronicity and recurrence.

Occupation

People engaged in outdoor activities in hot and humid environments are at a greater risk of infection since this provides a favorable environment for dermatophytes. Recent studies too have reported that manual laborers are most commonly affected.[13,18,22] Farmers are at an additional risk due to increased exposure to fungal pathogens from the environment and frequent contact with soil and animals.[9]

More homemakers with active infection are seen now.[13] The hot environment of the kitchen with increased sweating favors the growth of dermatophytes, making homemakers susceptible. Rudramurthy *et al*. found homemakers as the most common affected group (25.1%) in their study.[15]

An increased frequency of dermatophytosis has also been reported in students recently.[9,10,22] Increased sweating during school and sport activities along with wearing of school uniform and footwear for prolonged hours might be contributory factors. Moreover, wearing tight-fitting synthetic clothing, common amongst younger individuals, provides damp and warm skin conditions[5,13] possibly contributing to the increased prevalence, recurrence and chronicity of these infections in them.

Topical steroid abuse

Topical steroid misuse may be the most important cause of the current outbreak of chronic and recalcitrant dermatophytosis. A strong temporal association has been observed between the increasing availability and irrational use of the combination creams (antifungal-steroid or antifungal-antibiotic) with the sudden increase in chronic, recurrent and refractory cases in the past 4–5 years.[5] Potent steroid-containing creams are easily available over the counter without prescription. They are often suggested by pharmacists or friends, or are prescribed by general practitioners, and patients keep using them erratically for months to years. At the time of presentation to the dermatologist, most patients are found to have taken some form of treatment [Table 3]. Oral/topical azoles and topical steroids alone or in combinations are commonly self-administered by patients with dermatophytosis.[11] Studies in the last 1-2 years reveal that the frequency of prior application of topical steroid-containing combination creams is very high (42%[16]–81%[4]) among patients. Ironically, the use of topical antifungals was lower, seen in 5.7%[9]–47%[13] of patients [Table 3].

Causative organism

Trichophyton mentagrophytes (mostly *Trichophyton interdigitale*) used to be the predominant organism responsible for superficial dermatophytosis before 1935 worldwide.[28,29] Thereafter, an epidemiological shift began with *Trichophyton rubrum* replacing *Trichophyton. mentagrophytes* and emerging as the predominant organism by 1954 in many countries and continents (United States, Australia, Germany, Switzerland and Netherlands) including India with a reported incidence of up to 80%.[29] The environmental factors (humidity, temperature, trauma), internal factors (host-parasite relationship, host susceptibility) and immunological factors have been speculated for this shift along with the changing virulence of the organism.[4,29] It remained the most prevalent causative agent till the recent upsurge of dermatophytosis, when *Trichophyton mentagrophytes* has again emerged as the predominant organism. The same factors have been thought to be responsible as during the previous epidemiological shift.[4] Moreover, a probability that the zoophilic *Trichophyton mentagrophytes* has acclimatised itself and has undergone anthropization leading to easy transmission among humans has been suggested.[4] In the past decade, and especially from the beginning of 2012, the reported prevalence of *Trichophyton mentagrophytes* has not only increased,[6,7,10,12,18,22,26,30] but it has even replaced *Trichophyton rubrum* in a few studies[Table 4].[9-11,24,26] The most recent studies have found *Trichophyton mentagrophytes* in more than 90% of their dermatophyte isolates. In a recent large multicentric study, *Trichophyton mentagrophytes*/*Trichophyton interdigitale* complex was identified in 93.2 % and *Trichophyton rubrum* in only 6.8%.[4] Yet another study recently demonstrated 97.2% of isolated dermatophytes to be *Trichophyton mentagrophytes* complex with the most significant species being *Trichophyton interdigitale*.[14] Singh *et al*. too identified *Trichophyton interdigitale* in 94% of cases.[16]

Trichophyton mentagrophytes exhibits a rapid growth in primary culture within 5–7 days; this might explain the extensive involvement, inflammatory lesions and fomite transmission frequently seen now.[5]

Immunopathogenesis of Chronic Dermatophytosis

Fungal cells evade host immune responses to survive. Mechanisms include masking of cell wall-associated carbohydrates, shielding of stimulatory surface recognition molecules, prevention of toll-like receptor recognition, shedding of decoy components and downregulation of the complement cascade.[31-33]

A recent study from India demonstrated a higher expression of Th17 and Treg cell markers on CD4+ cells of patients with chronic dermatophytosis as compared to controls.[34] It was speculated that the interaction between the cell wall antigen mannan with the host cell receptor triggers a

Table 3: Pattern of use of topical preparations before presenting to the dermatologist for the treatment of superficial dermatophytosis

Author, year	Topical steroids alone or combination	Topical antifungal alone
Nenoff *et al*., 2019[4]	82 (81.3%)	95 (93.1%) (topical and oral antifungals
Singh *et al*., 2019[16]	63 (42.0%)	31 (20.7)
Vineetha *et al*., 2018[13]	63%	47% (azoles - 28%, allylamines in 19%)
Pathania *et al*., 2018[12]	80 (53.3%)	
Mahajan *et al*., 2017[11]	187 (70.6%)	15 (5.7%)

Table 4: Predominant species isolated in superficial dermatophytosis in studies from 2013 to 2019

Author, year	Place	Predominant species (%)
Nenoff *et al*., 2019[4]	Multicentric	*T. mentagrophytes* (93.21)
Vineetha *et al*., 2018[13]	Kottayam (Kerala)	*T. rubrum* (21)
Tigga *et al*., 2018[14]	New Delhi	*T. mentagrophytes* (97.2)
Pathania *et al*., 2018[12]	Chandigarh	*T. mentagrophytes* (40)
Rudramurthy *et al*., 2018[15]	Chandigarh	*T. interdigitale* (66.1)
Mahajan *et al*., 2017[11]	Varanasi	*T. mentagrophytes* (75.9)
Sharma *et al*., 2017[10]	Sikkim	*T. mentagrophytes* (40)
Dabas *et al*., 2017[20]	New Delhi	*T. interdigitale* (56)
Noronha *et al*., 2016[26]	Mangalore	*T. mentagrophytes* (48.3)
Ramaraj *et al*., 2016[21]	Chennai	*T. rubrum* (48.95)
Poluri *et al*., 2015[22]	Andhra Pradesh	*T. rubrum* (58.06)
Kaur *et al*., 2015[7]	New Delhi	*T. rubrum*
Lakshmanan *et al*., 2015[8]	Tamil Nadu	*T. rubrum* (79)
Surendran *et al*., 2014[30]	Mangalore	*T. rubrum* (67.5)
Maulingkar *et al*., 2014[23]	Goa	*T. rubrum* (38.2)
Bhatia and Sharma, 2014[24]	Himachal Pradesh	*T. mentagrophyte* (63.5)
Bhagra *et al*., 2014[25]	Shimla	*T. rubrum* (66.17)
Agarwal *et al*., 2014[9]	Jaipur	*T. mentagrophytes* (37.9)
Vyas *et al*., 2013[27]	Jaipur	*T. violaceum* (43.3)

T. rubrum: Trichophyton rubrum, T. mentagrophytes: Trichophyton mentagrophytes, T. violaceum: Trichophyton violaceum, T. interdigitale: Trichophyton interdigitale

cascade of dysregulated cytokines eliciting Th17 mediated inflammation but also leads to the unhindered activation of Treg cells undergoing a phenotype change to an alternate lineage. Hence, despite antifungals, the high fungal burden becomes detrimental to a beneficial effector T-cell response. This perpetuates the imbalance with an altered Th17/Treg ratio preventing fungal clearance.

Clinical Features of Superficial Dermatophytosis

The clinical changes in the appearance of dermatophytic infections observed in India have been summarized in Box 1.

Changes in clinical behavior

Quick evolution

Sudden appearance and quick evolution of lesions, sometimes within days, seems to be common, unlike in the past.

Induction of varying degrees of inflammation

It is common now to find moderate or severely itchy, peripherally spreading, flat, whitish or brownish lesions without erythema which yield profuse powdery scales when scraped [Figure 1a]. Such lesions have been called 'non-inflammatory' superficial dermatophytosis[35], but the term may be inaccurate as the absence of erythema does not necessarily mean the absence of inflammation.

Multiple lesions in different anatomical locations exhibiting varying degrees of inflammation are also seen [Figure 1b]. Many patients report noticing diurnal changes, with lesions tending to get more erythematous and raised and the edges becoming more defined at times [Figure 1c]. Heat, humidity, sweating, tight clothing, friction, exercise and topical corticosteroid-containing fixed-dose combinations, are known to modify the appearance of lesions. These observations necessitate further research on host immunity, the virulence of the organism and the nuances of topical steroid use.

Exacerbation of inflammation after starting treatment

An abrupt increase in the number of lesions with increased erythema, edema and occasionally pustulation, is seen on starting oral antifungal drugs like itraconazole in some cases [Figure 2a and b]; many of them have been abusing topical steroid-containing fixed-dose combinations. This phenomenon could be on account of withdrawal of corticosteroid with the administration of oral antifungal agents causing a recovery of the local cell-mediated immunity. Another phenomenon is the abrupt onset of multiple papules, plaques and at times, disseminated eczematous lesions with itching and burning in areas other than those with pre-existing tinea. These lesions appear soon after starting oral antifungal therapy and can cause diagnostic confusion between a dermatophytid and an adverse reaction to the antifungal agent, usually itraconazole.

Chronicity, recurrence and relapse

Chronic dermatophytosis may be defined as disease lasting for more than 6 months in a patient who has been adequately treated. It is common for patients with such longstanding infection to have received treatment comprising fixed-dose combinations as also suboptimal doses and durations of oral and topical antifungal drugs. Recurrent dermatophytosis has been defined as disease in which there is a recurrence of lesions within 6 weeks

Box 1: Summary of the changes seen in the clinical presentation of superficial dermatophytosis in the current epidemic like situation in India

Changes in the distribution of lesions in dermatophytosis

Tinea corporis et cruris - the most common presentation

Tinea facei - much more common than earlier

Multiple lesionsseen, multiple body sites involved

No concomitant increase in tinea unguium, tinea pedis or tinea capitis reported

Erythrodermic variants seen – not infrequently

Changes in the clinical behavior of dermatophytosis

Rapid spread of lesions to form large, bizarre- shaped patches

Early involvement of distant areas compared to the past

Varying degrees of inflammation

Easy spread to other family members often causing the entire family to suffer.

Post tinea eczema/dryness leading to persistence of itch

Flare of lesions with increased inflammation within the lesions of tinea after starting oral potent antifungal, with occasional systematization

Concomitant bacterial infections like furunculosis have become common

Chronic dermatophytosis, recurrences, relapses common

Changes in morphology

Steroid-modified tinea

Varying morphology – arcuate, dumb-bell shaped, bizarre geographic lesions

Double-edged tinea

Tinea pseudoimbricata

Tinea incognita – ill-defined borders, unclear borders – difficult to recognize tinea

Eczematous lesions

Tinea mimicking other dermatoses

Involvement of unusual locations

Genital dermatophytosis of males and females

Superficial dermatophytosis of scalp skin

Tinea auricularis

Tinea labialis

Tinea blepharitis and ciliaris

Tinea of vellus hair

Tinea over immunocompromised districts /sites affected by tinea being recognized as immunocompromised districts

Signs of topical/oral/injectable steroid abuse and irritant applications

Characteristics of itch in dermatophytosis

Often disabling itch ,frequent nocturnal aggravation

Persistent itch after the resolution of lesions

Changes in the impact of disease on society

Distressing to the patient and family; high impact on quality of life

Major financial burden

Confusing profusion in the manufacture of available topical and oral antifungals agents by pharmaceutical companies

after completion of treatment.[36] Relapse refers to lesions occurring more than 6–8 weeks after a patient has been cured clinically. All these presentations have become very common in current practice.[37]

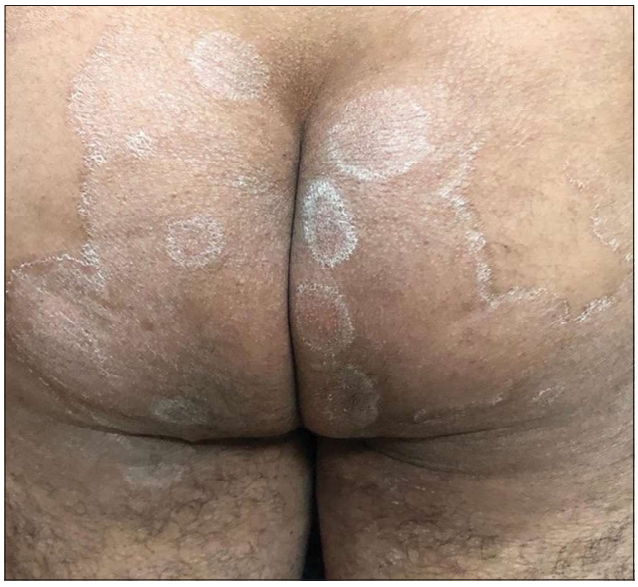

Figure 1a: Annular plaques of tinea corporis with minimal inflammation

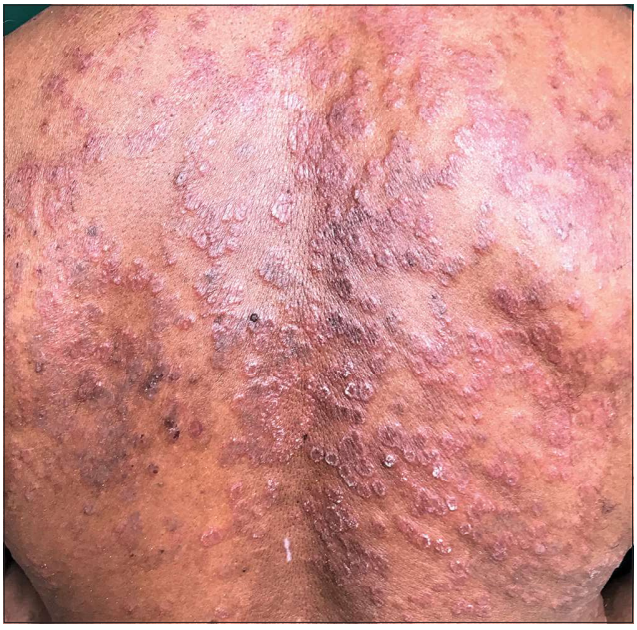

Figure 1b: Multiple annular erythematous plaques of tinea with peripheral pustular borders suggestive of severe inflammation

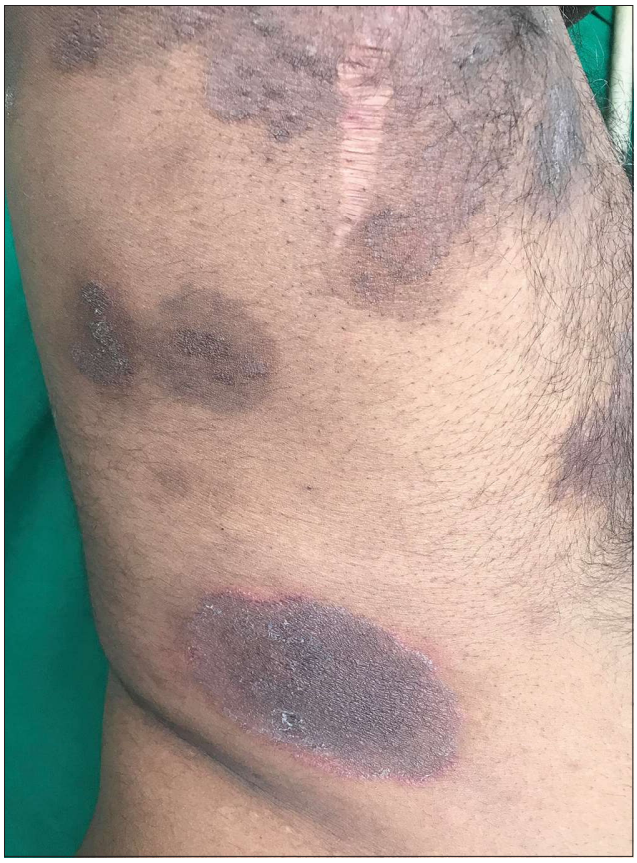

Figure 1c: Tinea corporis et cruris showing varying degrees of inflammation in the same patient

Changes in the morphology of lesions

Well-defined, centrifugally spreading lesions with central clearing and an advancing border, as described in textbooks, are not the rule now. A plausible explanation for this is that treatment-naïve cases are now uncommon.

The following are the morphological forms encountered in the current scenario:

Steroid-modified tinea

Lesions were labeled tinea incognita/tinea incognito in the past because their appearance was different from that of a "classic" lesion of tinea[38] due to the application of steroid creams. Though a majority of patients now present with altered lesional morphology after having used fixed-dose combination creams with steroids (most often clobetasol propionate or betamethasone dipropionate), most such lesions are not "incognito" in a strict sense.[39] It is usually possible in such cases to discern an active border with or without scaling, as typical in superficial dermatophytosis. Therefore, "steroid-modified tinea" is a more accurate term.[39] True cases of tinea incognita are fortunately uncommon.

Varying morphology

Unusually large lesions are seen, either due to centrifugal spread or by coalescence of multiple lesions. Circular or annular lesions, [Figure 3], arcuate and dumbbell-shaped lesions where two annular or circular lesions coalesce, annular lesions with pustular borders [Figure 4], and bizarre geographic lesions are common. Erythrodermic presentations of dermatophytosis are seen in immunosuppressed as well as immunocompetent individuals [Figure 5a and b].[40]

Incomplete central clearance and eczematous appearance

It is common to see incomplete clearing in the centre of lesions leading to variably defined areas of erythema with or

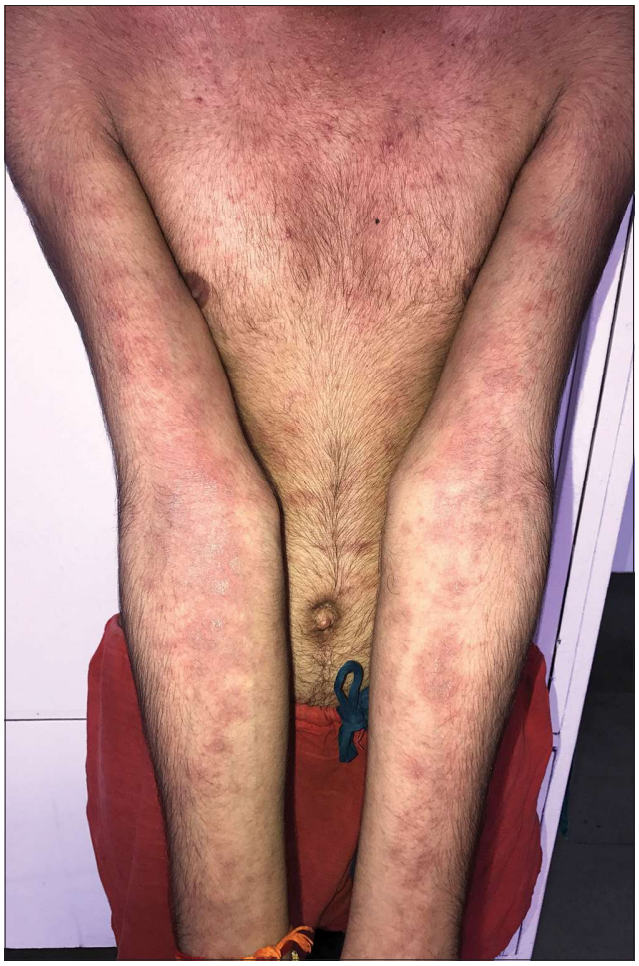

Figure 2a: Sudden exacerbation of tinea with the appearance of multiple eczematous scaly lesions after starting antifungal therapy

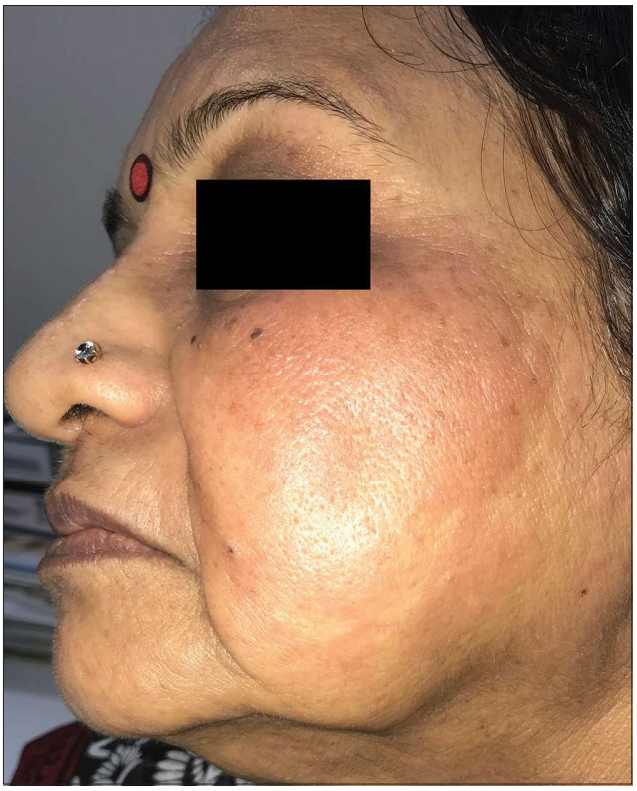

Figure 2b: Sudden appearance of erythema and edema overlying a plaque of tinea faciei after starting antifungal therapy

without scaling i.e.,"eczematous tinea" [Figure 6a and b]. The natural course of the disease is for cell-mediated immunity to mount an inflammatory response and increase the epidermal cell turnover to eliminate the fungus by shedding the stratum corneum[41]. Lesions treated with steroids show activity in the centre as well as in the peripherally spreading margins.[5] Topical steroid abuse slows down cell turnover and downregulates beneficial proinflammatory mediators, aiding survival of the fungus.

"Double-edged tinea" and tinea pseudoimbricata

Erratic application of fixed-dose combinations and steroid creams in tinea switches inflammation on and off, leading to "double-edged" tinea [Figure 7].[42] The degree of inflammation in such cases often is high, and residual post-inflammatory hyperpigmented "double edges" persist for a long time after successful treatment too [Figure 7]. At times, the double edges are well-defined, running parallel to each other and with central crusting and erosions, resulting in a pseudoporokeratotic appearance [Figure 8a and b]. Repeated on-and-off cycles of inflammation for long periods can lead to concentric rings of tinea described as "tinea pseudoimbricata"[Figure 9a and b]. A "rings within a ring" appearance is also not uncommon and is characterized by smaller scaly lesions of tinea within a centrifugally spreading larger annular lesion, often with more than one border.[43] Double-edged tinea, tinea pseudoimbricata and tinea "rings within rings" lesions are all highly suggestive of steroid abuse, especially erratic use of steroid-containing fixed-dose combinations.[44]

Another uncommon entity is tinea recidivans that describes the appearance of lesions at the periphery of a healed lesion [Figure 10a and b].

Pustular lesions in tinea

Pustular lesions are often seen at the periphery of inflammatory erythematous lesions of tinea being treated with potent topical steroids. In some cases, each ring of a tinea pseudoimbricata lesion can be studded with pustules, giving rise to a cockade pattern [Figure 11].

Majocchi's granuloma

Rarely, dermatophytes may invade the dermis owing to the reduced local immunity induced by abuse of topical steroids. Such cases show pruritic papules, pustules and nodules in the areas of preexisting dermatophytic infection [Figure 12], and granulomatous perifollicular inflammation on histology.

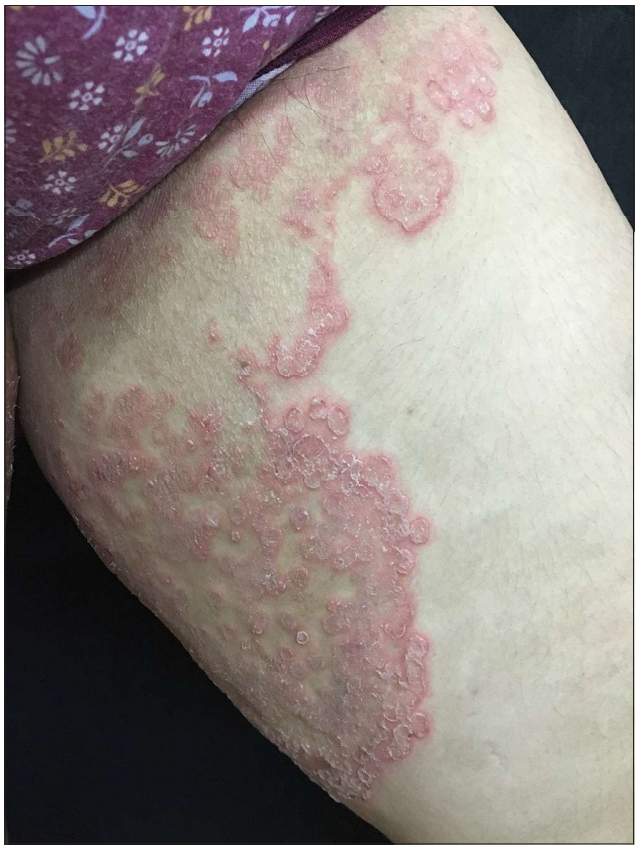

Figure 3a: Multiple annular lesions coalescing to form a plaque

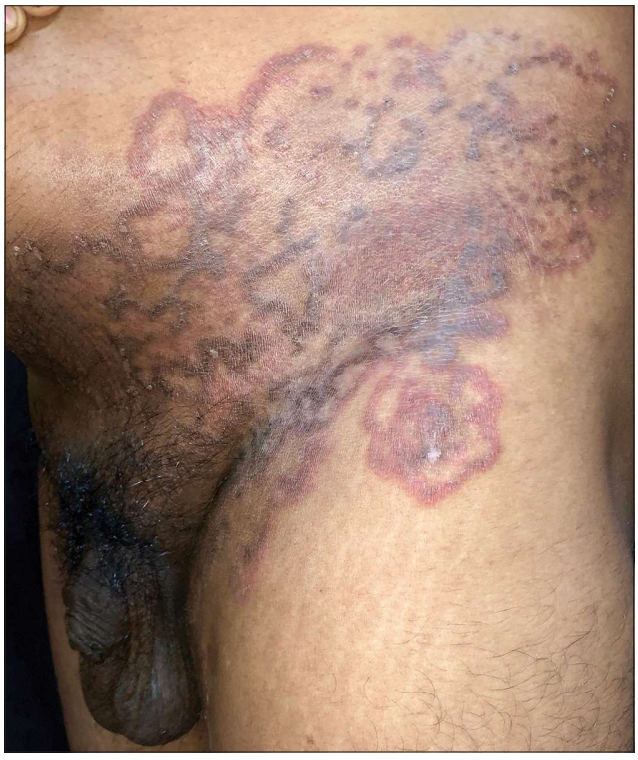

Figure 3b: Multiple annular lesions coalescing to form a plaque

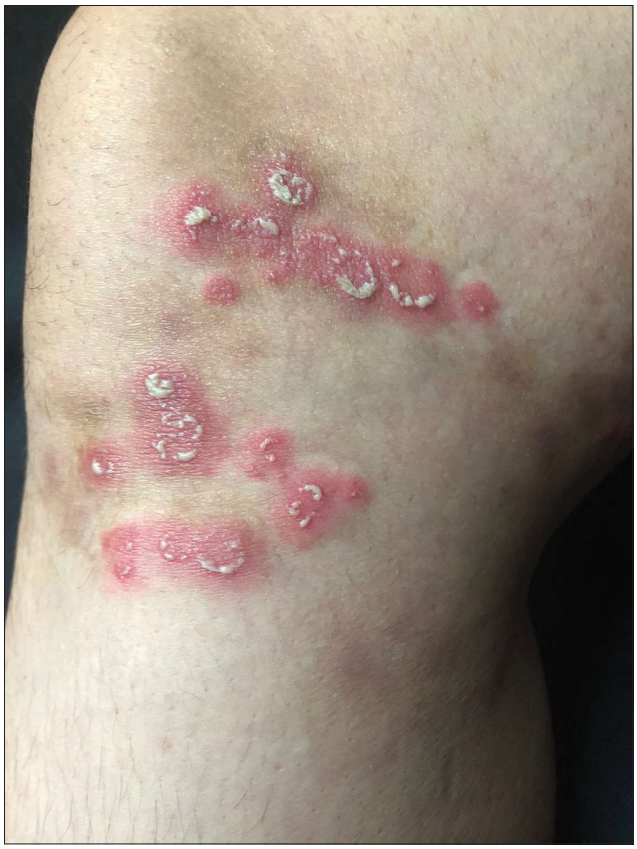

Figure 4: Multiple coalescing lesions with pustules

Bullous tinea

Intense inflammation caused by the zoophilic *Trichophyton mentagrophytes* and a heightened delayed type of hypersensitivity reaction are thought to be causes of a vesicular or bullous presentation. This is characterized by annular lesions with raised, vesicular edges.[45]

Superficial dermatophytosis mimicking other dermatological conditions

Tinea has been observed to mimic a wide variety of conditions including lupus erythematosus,[46] psoriasis, lichenoid lesions, atopic eczema,[47] nummular eczema, erythema multiforme,[48] granuloma annulare, granuloma faciale, lymphocytic infiltration of the skin, pityriasis rosea, seborrheic dermatitis,[49] leprosy,[50] molluscum contagiosum,[51] rosacea[52] and annular secondary syphilis,[37] pustular psoriasis, Sweet's syndrome and impetiginized herpes.[53] Cutaneous dermatophytosis caused by *Trichophyton mentagrophytes* mimicking bullous pemphigoid clinically, histopathologically and on direct immunofluorescence has been recently reported.[54] Though many of these are isolated cases described in the Western literature, it is now perhaps prudent to keep in mind the possibility of dermatophytosis when encountering such presentations in our patients.

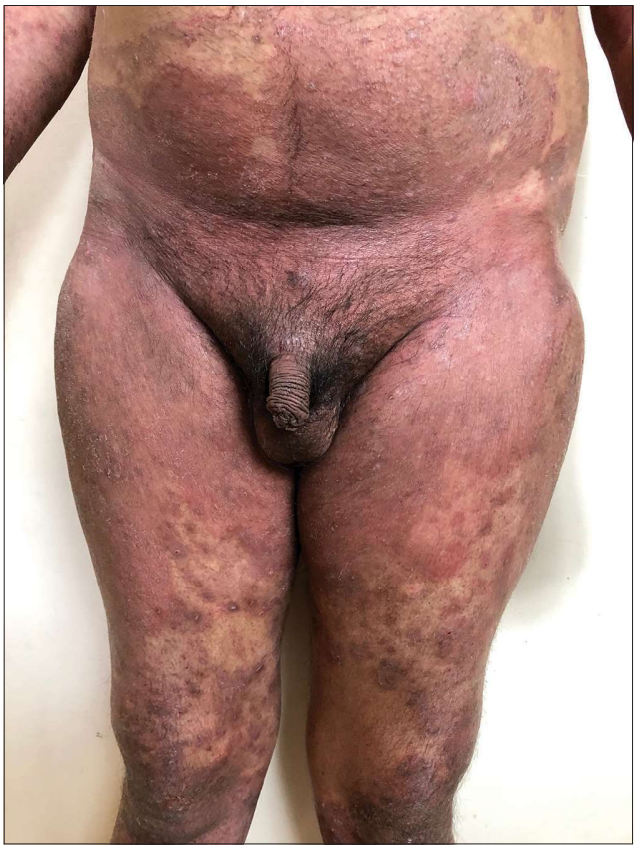

Figure 5a: Erythrodermic variant of superficial dermatophytosis

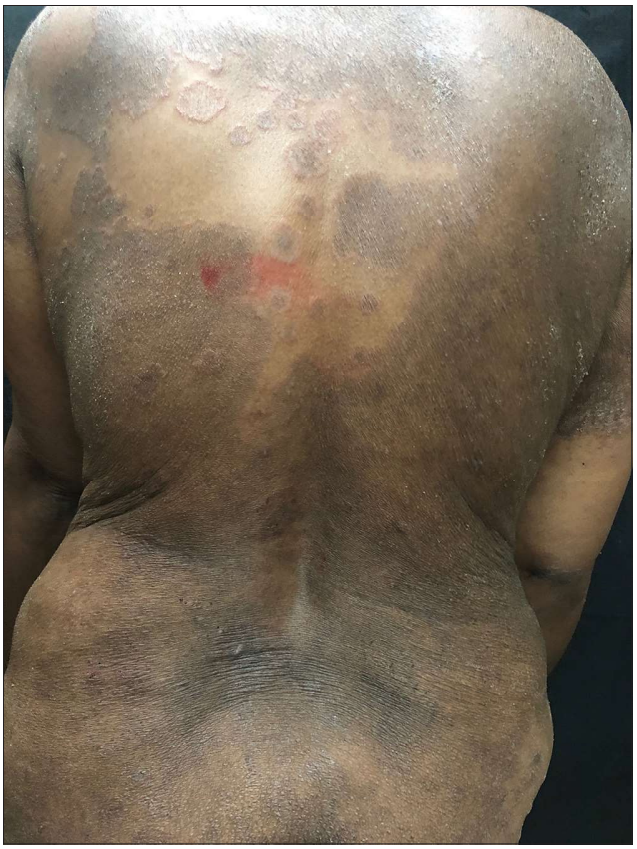

Figure 5b: Erythrodermic variant of superficial dermatophytosis

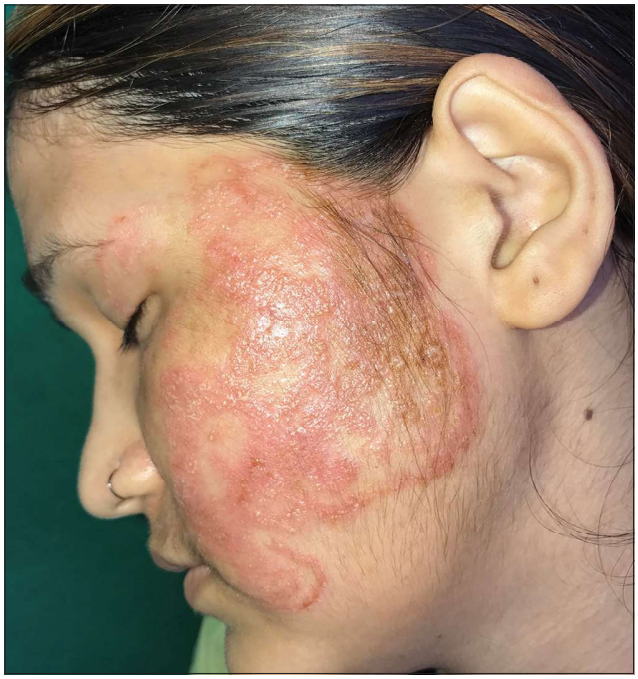

Figure 6a: Eczematous variant of tinea facei

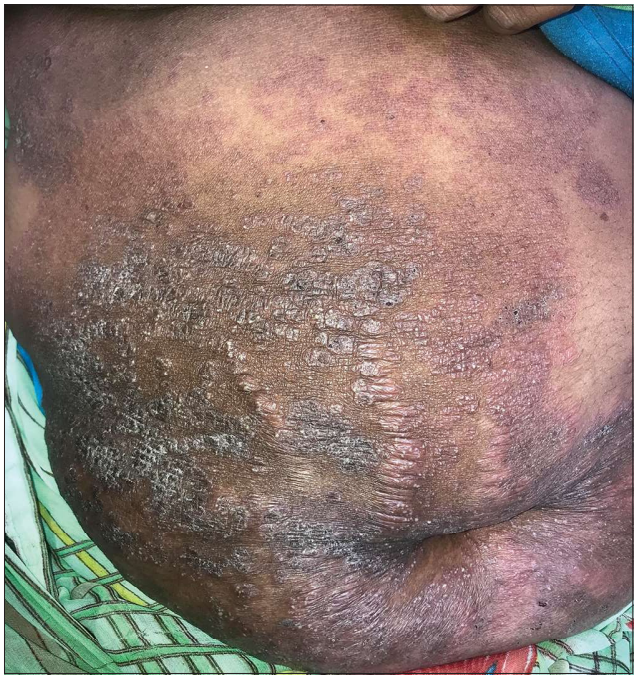

Figure 6b: Eczematous variant of tinea corporis

Change in sites of skin involvement

The involvement of multiple body sites is common. Tinea corporis et cruris is the most common presentation;[3] individual tinea corporis and tinea cruris are the next two commonest. Lesions at sites of occlusion like the waist and inframammary areas are frequent in female patients [Figure 13a,b].

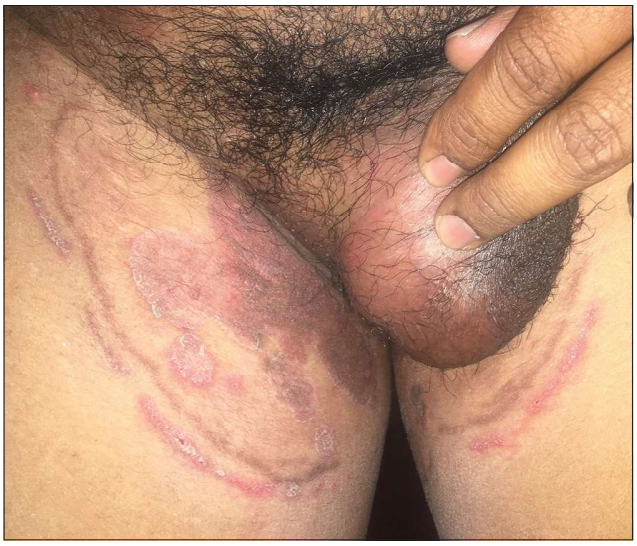

Figure 7: Double-edged tinea cruris

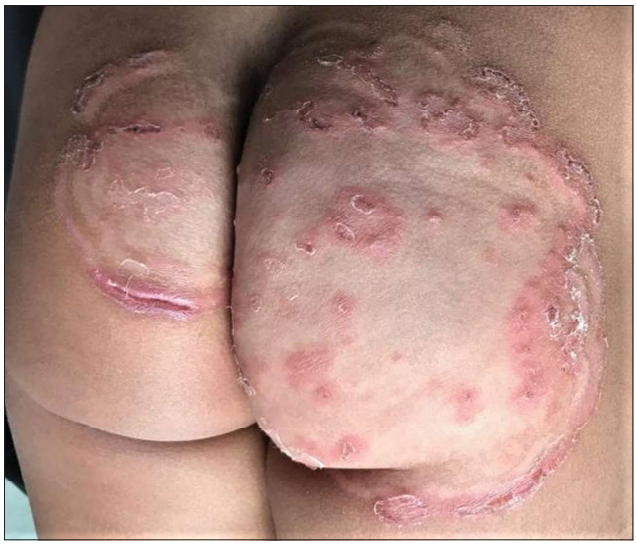

Figure 8b: Steroid modified tinea with pseudoporokeratotic edges

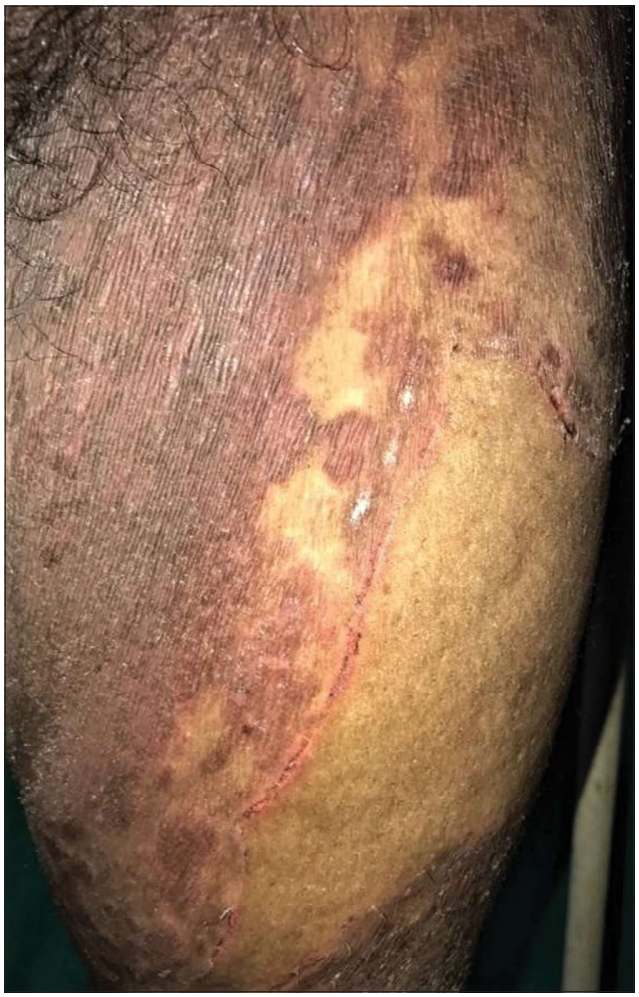

Figure 8a: Steroid modified tinea corporis with pseudoporokeratotic edges

An interesting observation is the absence of a proportionate increase in tinea unguium, tinea pedis and tinea capitis.[5]

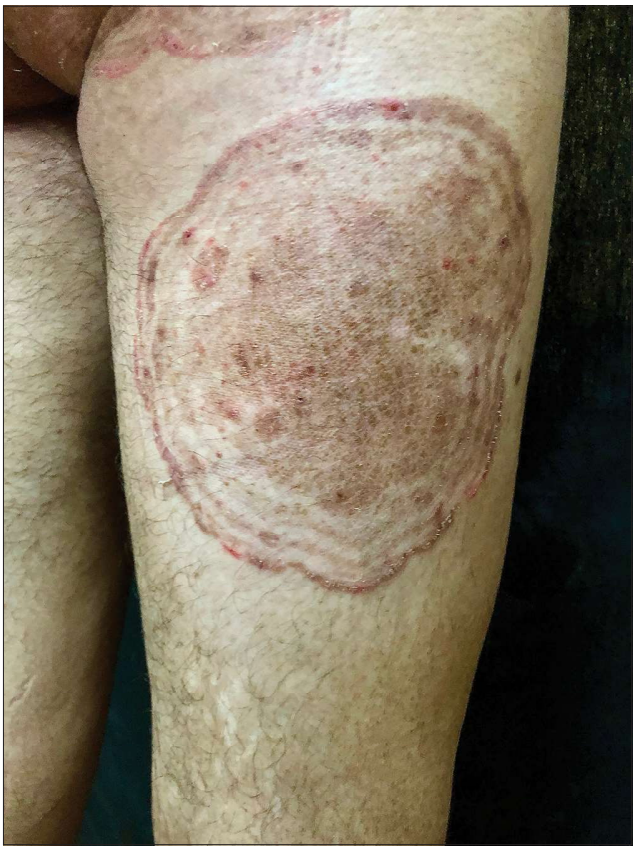

Figure 9a: Tinea pesudoimbricata

Involvement of unusual locations

Genital dermatophytosis

Genital dermatophytosis is not usually mentioned separately in standard textbooks, probably because of its rarity. Together with tinea cruris, it is referred to as tinea pubogenitalis. There is a male predominance with concomitant tinea cruris

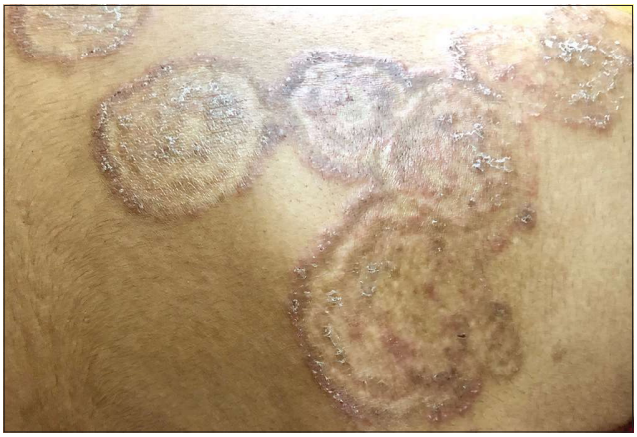

Figure 9b: Tinea pseudoimbricata

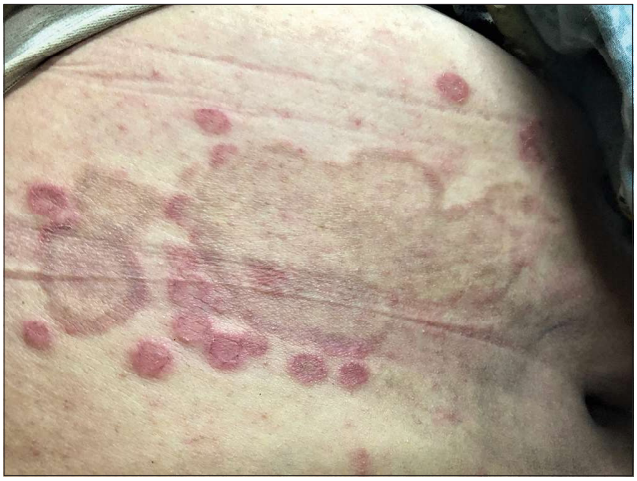

Figure 10a: Tinea recidivans

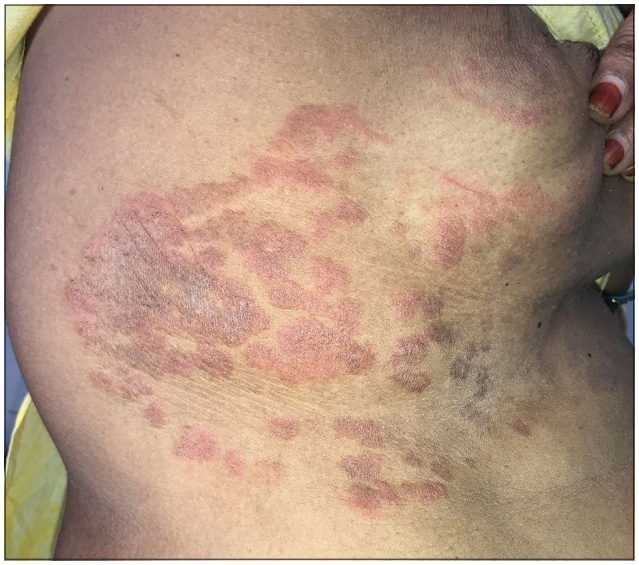

Figure 10b: Tinea recidivans on upper torso

or tinea cruris et corporis, and the vast majority of these patients have a history of applying fixed-dose combinations

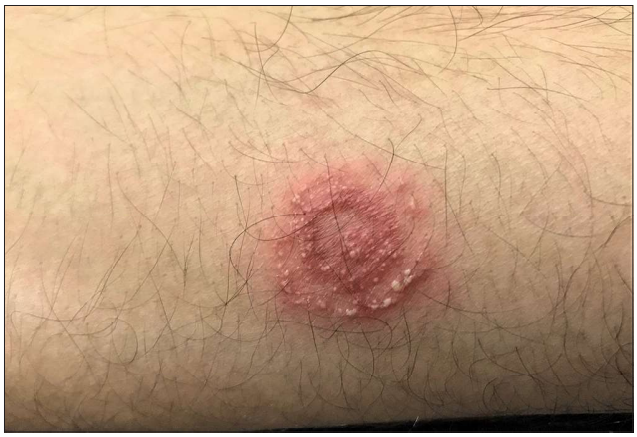

Figure 11: Cockade pattern of pustular lesions in steroid-modified tinea

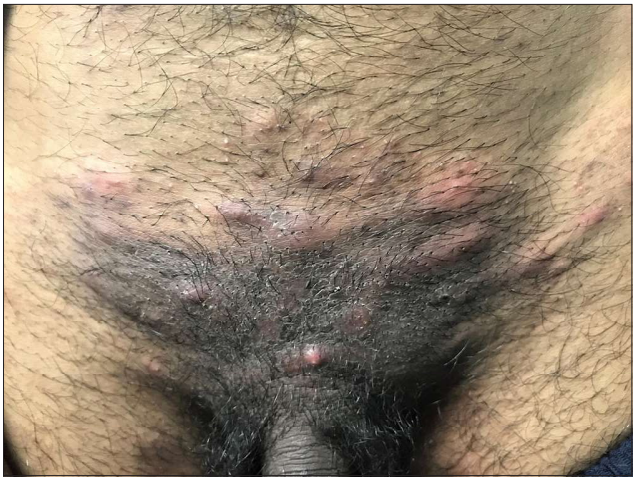

Figure 12: Majocchi's granuloma (Picture Courtesy – Dr. Chetan Rajput)

containing topical steroids.[55] The base of the penis is most often affected, followed by the shaft and rarely, the outer prepuce. The scrotum is affected less commonly. Lesions on the penile shaft vary from well- to ill-defined areas of scaling [Figure 14] to annular lesions with or without pustules, on the dorsal [Figure 15], lateral or ventral aspects. Many cases are detected during the examination because they are asymptomatic and patients have sometimes not even noticed them. These lesions usually resolve within days of application of topical antifungal agents. We would like to stress the importance of identifying and treating them because otherwise they may easily become afocus for recurrence.

A similar entity has been noticed in women with involvement of only the pubic region, or as a part of a larger plaque of tinea cruris [Figure 16].[56] Waxing or shaving the area may allow the dermatophyte deeper and cause extension of the infection. Patients must therefore be instructed that trimming the hair with scissors is preferable instead.

Superficial dermatophytosis of the scalp skin

Tinea of the glabrous scalp is a distinct entity in the current scenario in adults as well as children, where lesions involve the skin of the scalp, most often due to secondary spread, without apparent clinical, dermatoscopic or mycological involvement of the hair [Figure 17a], this last feature differentiating it from tinea capitis. Tinea corporis may extend from the nape of the neck to the lower occipital scalp, and tinea faciei to the lateral and anterior aspects of the scalp. The involvement usually does not extend beyond a centimeter or so into the scalp though occasionally, potassium hydroxide-positive, scaly, well-circumscribed areas may be seen more centrally.[56] This picture defies the earlier notion that sebum is fungistatic and prevents the proliferation of fungus; the higher virulence of *Trichophyton mentagrophytes* may be one explanation.

Lesions are better delineated with the hairs on the area trimmed. Dermoscopy [Figure 17b] and potassium hydroxide examination of the scalp skin scrapings help to differentiate this presentation from psoriasis and seborrheic dermatitis of the scalp. More studies are needed to explore whether there is subclinical involvement of hair in this condition.

Tinea faciei

An increasing frequency of tinea faciei has been noted in adults as well as children.[3] While it is often secondary to tinea elsewhere on the body, it can also be the first to occur, followed by disease elsewhere. The face may be the only site involved and remain so in some patients.

Lesions vary from a well-defined, annular, scaly plaque or multiple scaly plaques studding parts of the face to at times, diffuse erythema and scaling with barely discernible edges. The latter are examples of true 'tinea incognita' as they are difficult to diagnose and a potassium hydroxide examination is necessary. Tinea faciei often shows photoexacerbation with patients complaining of increased erythema, itching and transient edema of the affected areas when exposed to sunlight.

The following entities need special mention here:

a. Tinea facei involving the external ear and "tinea auricularis":
   Tinea faciei often shows extension to the skin of the ear. Lesions often extend to the lateral scalp and beyond when the ear is involved [Figure 18]. Involvement of one ear with a scaly, erythematous, papulosquamous eruption on the same side of the face points strongly towards the diagnosis of tinea faciei. External ear involvement in children with tinea capitis has been referred to in the past as the "ear sign".[57] We propose that this "ear sign" be extended to the context of tinea faciei as well, especially in the current scenario where tinea faciei has become so common.

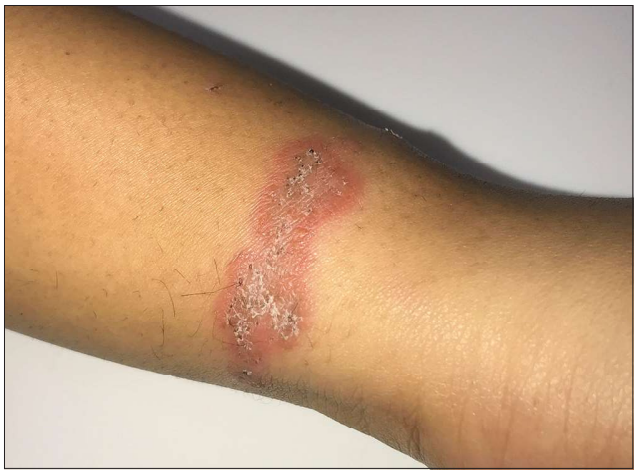

Figure 13a: Tinea corporis limited to the area of occlusion by wrist band

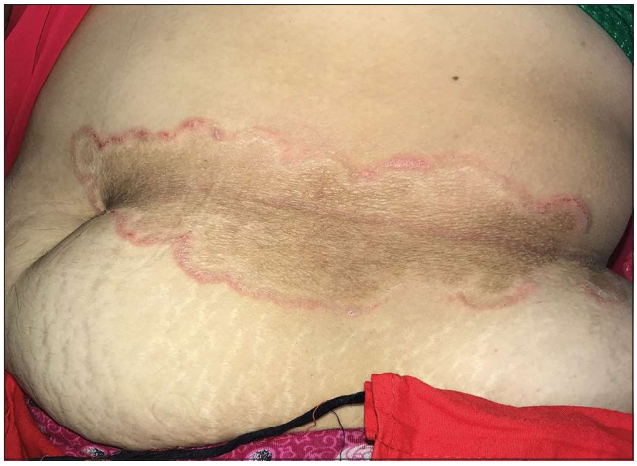

Figure 13b: Tinea corporis presenting as a large annular plaque limited to the area of waistline along the line of occlusion by drawstring

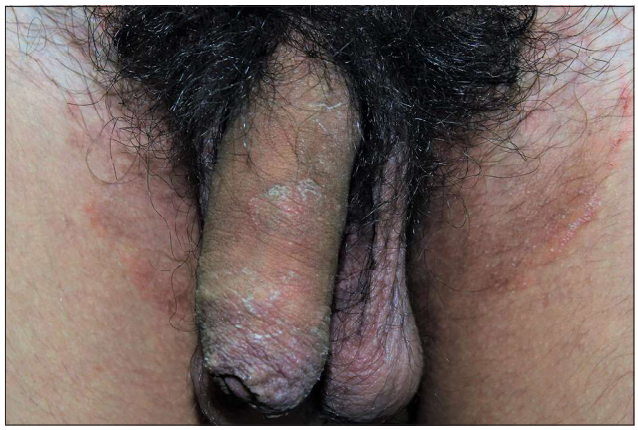

Figure 14: Tinea cruris with male genital dermatophytosis manifesting as ill-defined scaly plaques

b. Very rarely, involvement of only the ear may occur, which can be difficult to recognise with no clinical clue of dermatophytosis elsewhere on the face or the body. We propose the name "tinea auricularis" for this

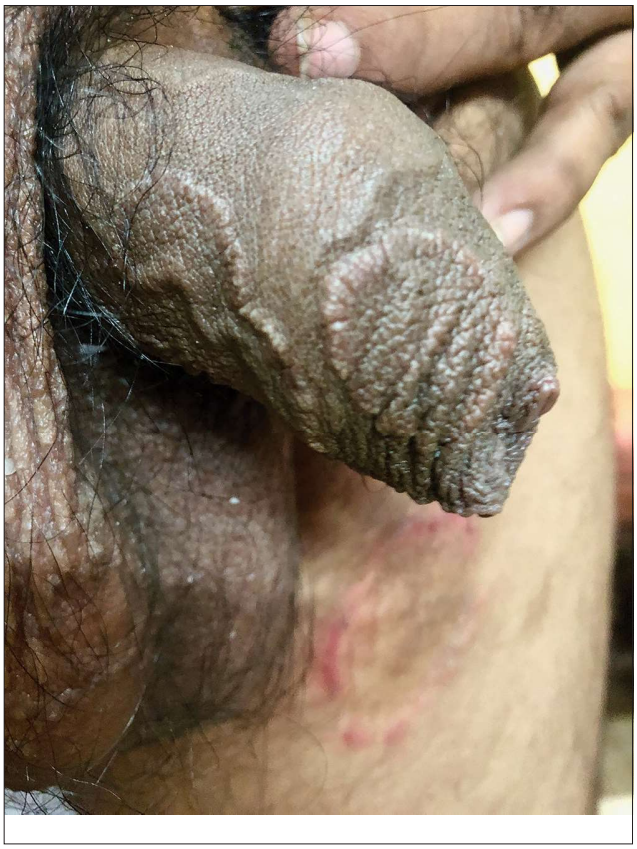

Figure 15: Tinea cruris with male genital dermatophytosis manifesting as well-defined annular plaques

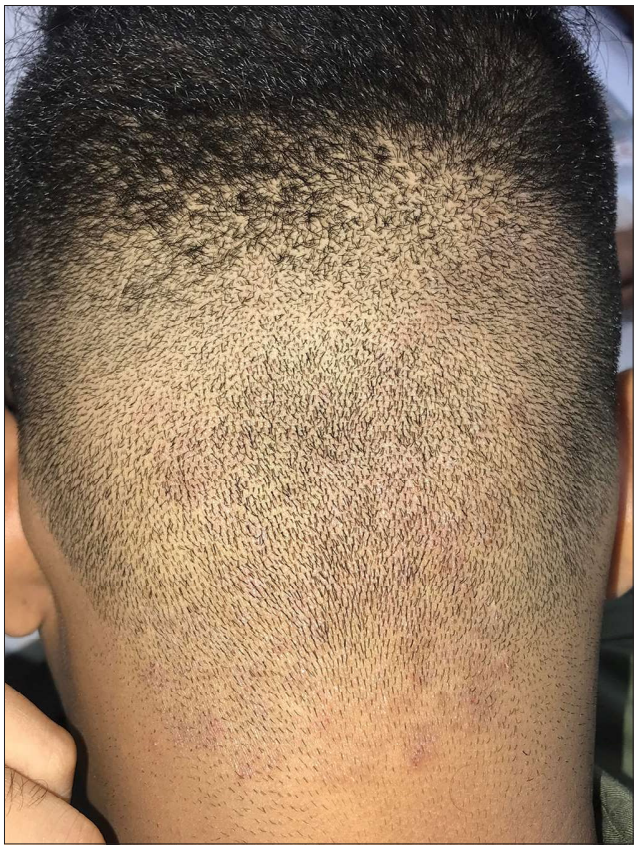

Figure 17a: Dermatophytosis of the scalp skin manifesting as an erythematous scaly, ill-defined plaque on the occipital area

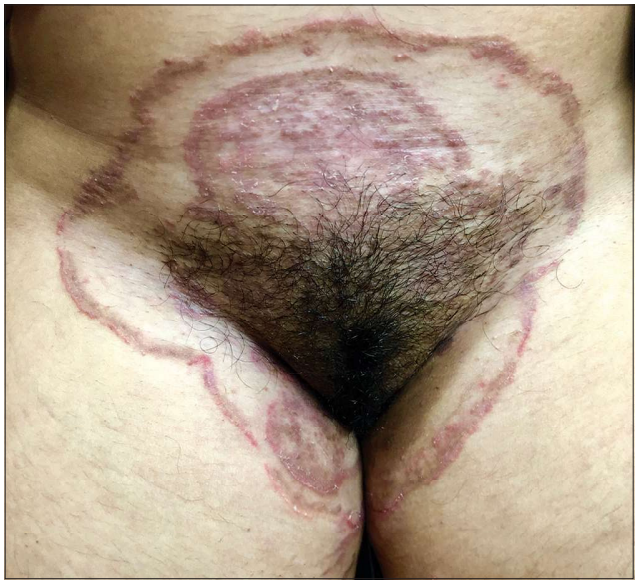

Figure 16: Tinea cruris with female genital dermatophytosis presenting as a double-edged annular plaque involving the pubic area

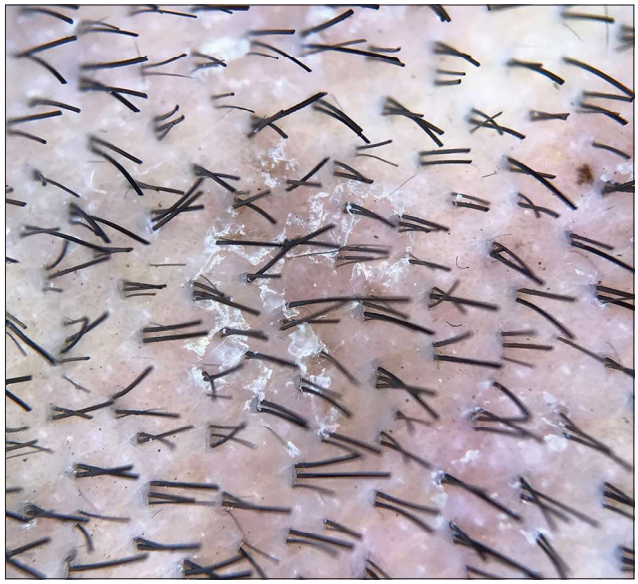

Figure 17b: Dermatoscopy of scalp skin dermatophytosis– manifesting as fine scaling. Dermlite DL4N, polarized mode x10

presentation [Figure 19a and b].[56]

c. Tinea facei extending onto the lips and tinea labialis: Lip involvement can occur due to contiguous spread from tinea faciei [Figure 20 a-c] or as an isolated phenomenon, tinea labialis. It is rare and presents as a well-circumscribed scaly lesion or as diffuse scaling over the entire lip.[56]

d. Tinea blepharitis with tinea ciliaris:

The term tinea capitis by definition, includes dermatophyte infections of the scalp, eyebrows and eyelids.[58] Rarely, dermatophytosis can localize itself to the eyelids[59] [Figure 21a and b], eyelashes or periocular area [Figure 22] without any involvement of the scalp.[56] With signs of rubbing and scratching, it can then easily be misdiagnosed as atopic blepharitis/ allergic contact dermatitis or atopic involvement of the periocular area, and treated with topical steroids. The topical steroid perpetuates the infection which can later involve the skin of the face.

Tinea of vellus hair and subclinical infection of the nails

Subclinical infection of the nail[60] or tinea of vellus hair are thought to be reasons for persistence of the fungus leading to recurrence and relapse.[61] This needs to be validated with large scale studies. While tinea of vellus hair can be documented using a dermatoscope[56,62] and a potassium hydroxide mount, subclinical infection of the nail can be documented by periodic acid–Schiff stain of nail clippings.

Tinea and immunocompromised districts

An immunocompromised district is a localised aberration in the immune control of the skin that has been damaged due to various causes[63]. Many of us are observing superficial dermatophytosis secondarily appearing over amputation sites, surgical incisions and tattoo sites, which are described as immunocompromised districts.[64]

Some dermatologists have also started documenting sites of healed tinea being immunocompromised districts. This could be explained by regional immune dysregulation at sites of tinea, and also related to topical steroid applications. One of us has documented varicella and herpes zoster appearing over partially as well as completely healed sites of dermatophytosis[65] [Figure 23a]. Vitiligo has been known to develop at the sites of tinea corporis [Figure23b] and so has lichen planus.[66].

Signs of topical/injectable corticosteroid use and irritant applications

Patients show much individual variation in the time taken to develop cutaneous adverse reactions, irrespective of the potency of the steroid molecule. It is also common to see patients seeking treatment of multiple stigmata of potent topical steroid abuse, like striae and hypopigmentation.

One of the commonest side effects of topical steroids is striae. They mostly occur early following the application of fixed-dose combinations containing clobetasol propionate and are most pronounced in the flexures and where the skin is thin, like the inner thighs. They are often initially erythematous (striae rubra) and eventually turn whitish (striae alba). Some striae may ulcerate making them prone to secondary bacterial infection [Figure 24].[67] Some gradually worsen and ulcerate because patients continue to apply the same creams that caused them. Some striae assume a pseudoedematous appearance [Figure 25].[68] Others may continue to grow

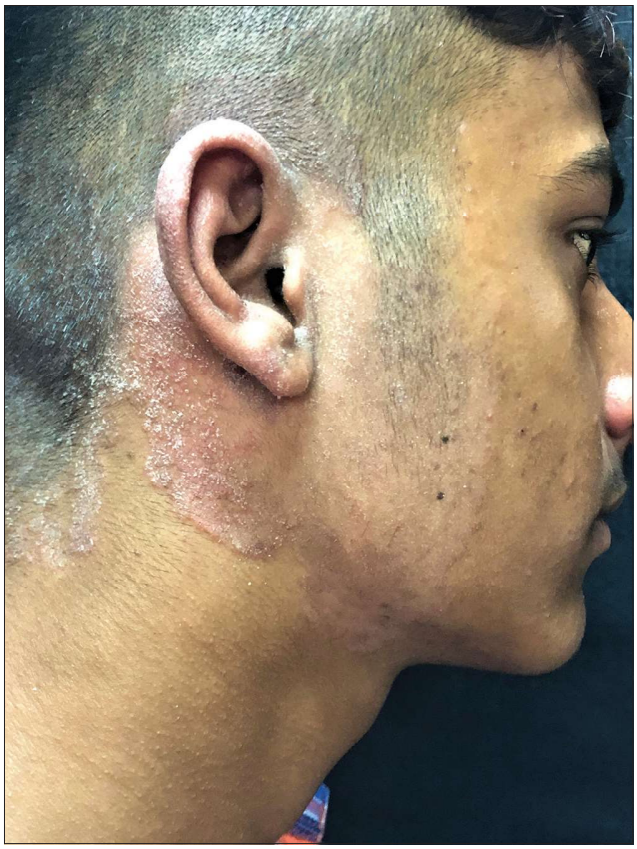

Figure 18: Tinea faciei with extension to the ear

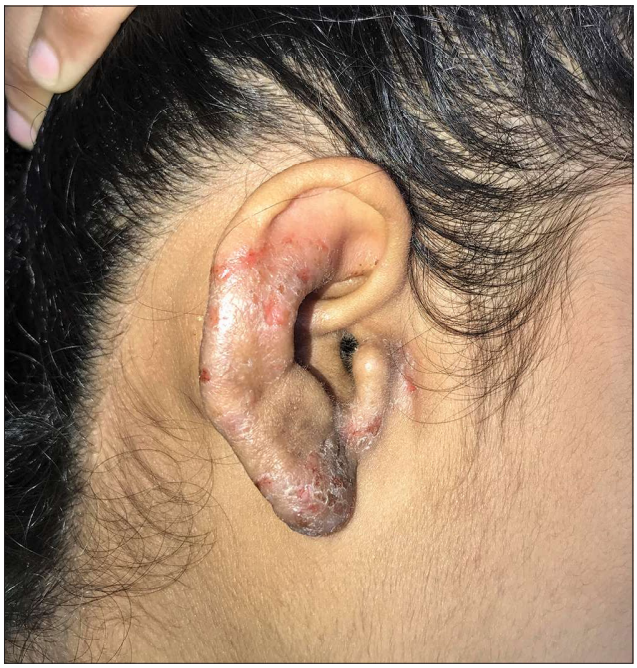

Figure 19a: Tinea auricularis

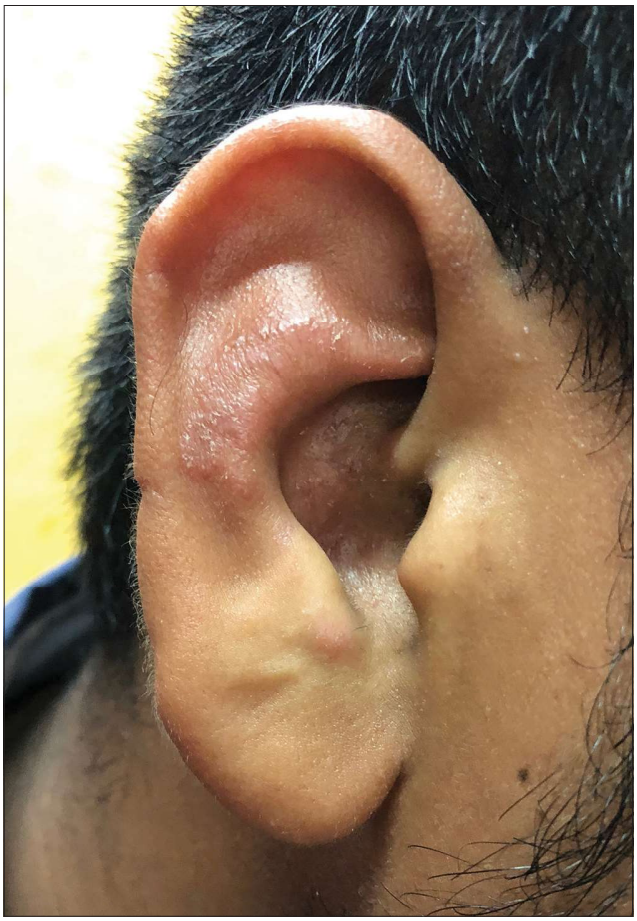

Figure 19b: Tinea auricularis

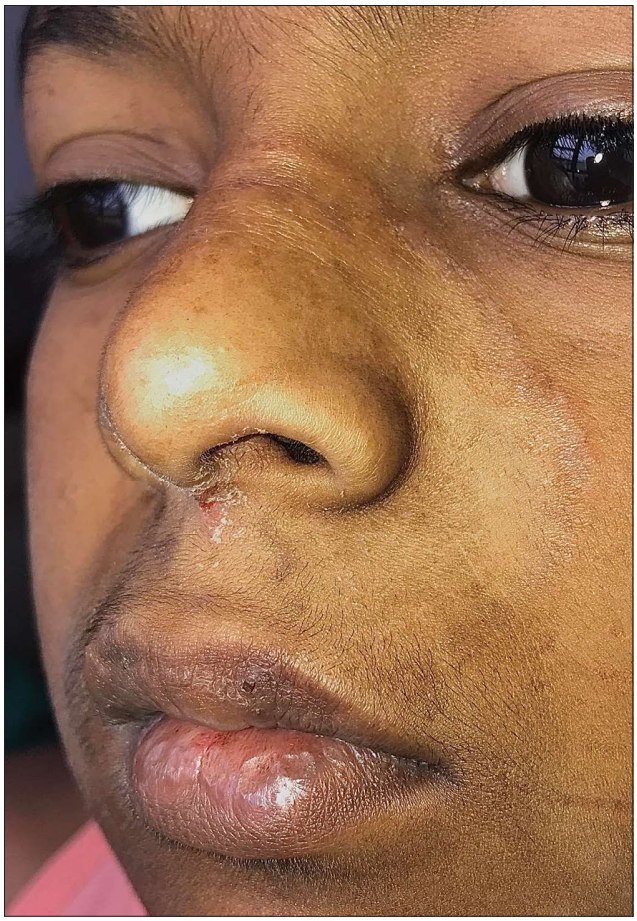

Figure 20a: Tinea faciei presenting as an annular plaque with extension to the upper lip

despite the patient having stopped applying fixed-dose combinations. This is indicative of high potency topical steroids having a depot effect.[69]

Striae may even occur in distal areas, where the fixed-dose combinations have not been applied. A clinical picture akin to red scrotum syndrome is also seen in some patients applying fixed-dose combinations containing topical steroids for tinea cruris, due to passive transfer of the creams.

Another common side effect of fixed-dose combinations with topical steroids is lesional and perilesional hypopigmentation; this can be seen within 3–4 weeks and is likely to be accompanied by atrophy and telangiectasia later on. Hypopigmentation and depigmentation can also be seen in patients treated with intralesional injections of triamcinolone acetonide [Figure 26a and b] by unqualified practitioners. These lesions are often linear or follow the course of lymphatics.

Concomitant bacterial infections such as recurrent furunculosis and abscesses can complicate the dermatophyte-infected sites, due to local immunosuppression induced by topical steroids [Figure 27].

Many patients present with signs of irritant dermatitis over and around the infected sites induced by home remedies made of crushed plants, flowers, balm, battery fluid, or over-the-counter preparations containing salicylic acid, sulfur, or anthralin with salicylic acid. The last combination is particularly irritant, especially in the flexures, leading to erythema, brownish discoloration, scaling and exfoliation, along with burning, pain and tenderness [Figure 28a and b].

Iatrogenic cushingoid syndrome in patients of tinea

Dermatologists are seeing an increasing number of patients, including children, presenting with longstanding lesions of tinea in multiple locations that usually wax and wane. These patients give a prolonged history of applying fixed-dose combination creams containing clobetasol propionate or beclomethasone propionate, sometimes running into hundreds of units. Many also give a history of taking weekly intramuscular injections of triamcinolone for several weeks or small doses of oral prednisolone, methylprednisolone or betamethasone for weeks to months. Many are overweight with truncal obesity and have striking striae (often in areas away from the sites of application of creams), hypopigmentation, telangiectasias, acneiform eruptions,

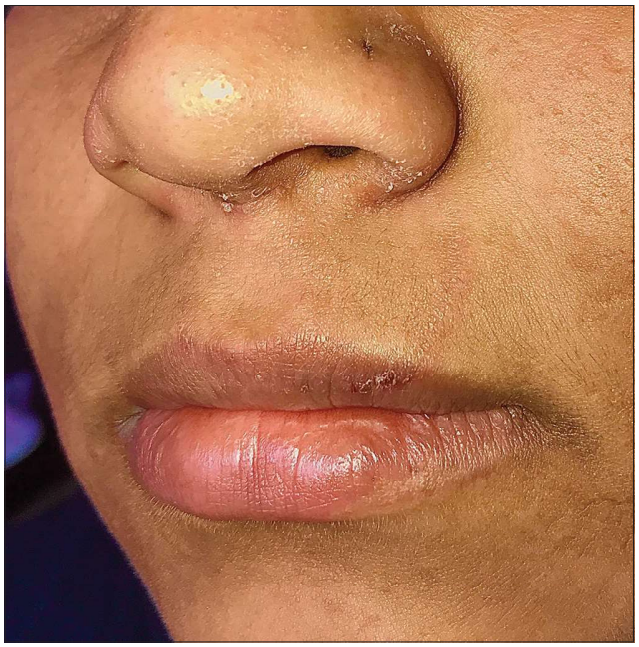

Figure 20b: A close-up view of tinea faciei presenting as an annular plaque with extension to the upper lip

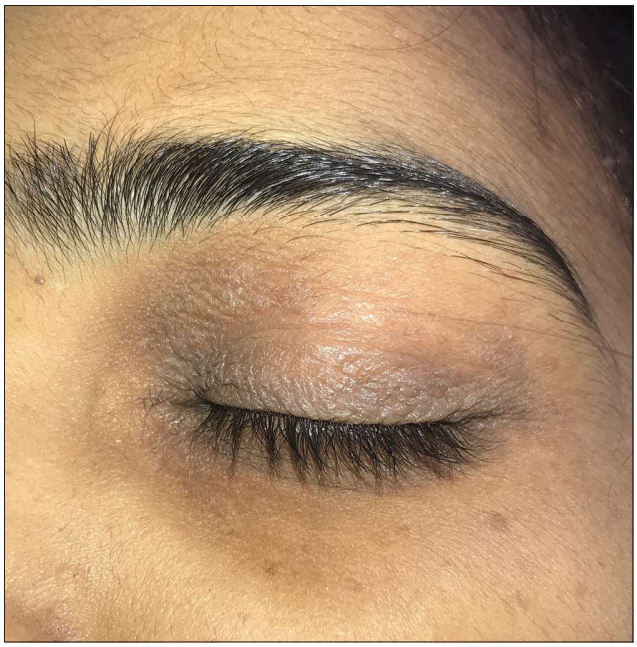

Figure 21a: Superficial dermatophytosis of the upper eyelid with involvement of the eyelid margin

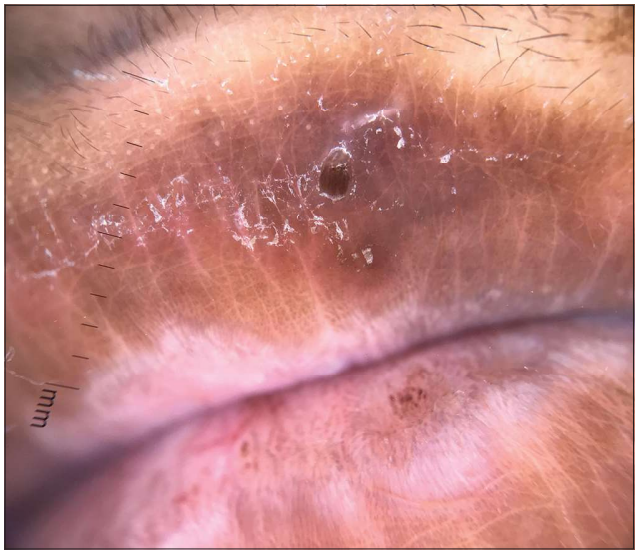

Figure 20c: Dermatoscopy of involvement of lip by superficial dermatophytosis showing scaling at the active border with crusting. DermLite DL4N, polarised mode, ×10

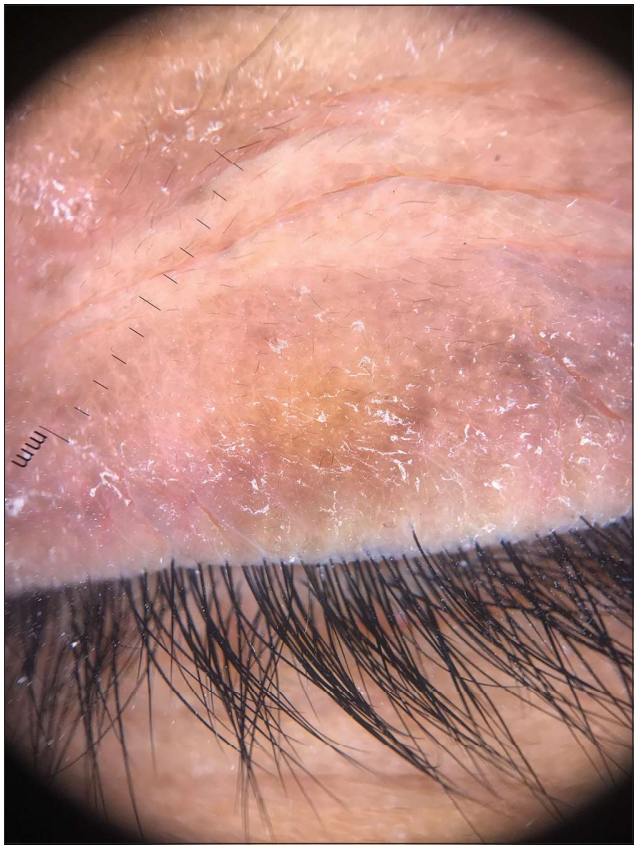

Figure 21b: Dermoscopy of superficial dermatophytosis involving the eyelid margin presenting with fine scaling along the eyelid margin sparing the hair shafts

local hypertrichosis and sometimes hirsutism [Figure 29]. Their 8 am and 4 pm serum cortisol levels are often very low, even less than 1 microliter per decaliter.[70] Further studies are needed to quantify the proportion of patients so affected and the amount/duration of application of topical steroids to induce this phenomenon.

Stopping steroid creams and instituting appropriate antifungal therapy is recommended in these patients. In our observation of a limited number of cases, the serum cortisol levels then return to normal within 3 to 5 months. The infection in such cases may take a much longer time to fully clear despite appropriate treatment.[71] We have had

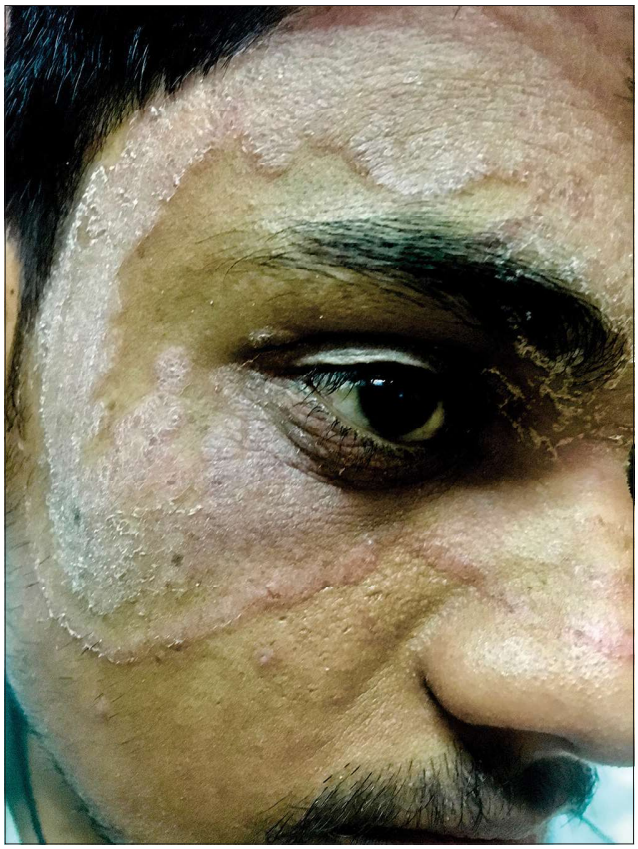

Figure 22: Tinea faciei involving the upper and the lower eyelids as well as the periocular area by extension

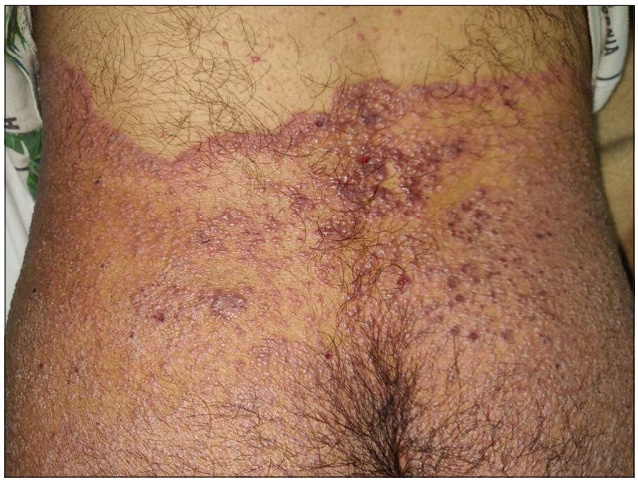

Figure 23a: Vesicles in a case of varicella predominantly localized to the site of tinea corporis that serves as an immunocompromised district (Photo courtesy - Dr. Sanjeev Gupta)

some patients needing treatment for 6–9 months before the infection finally resolved.

Characteristics of itch

Itching is one of the cardinal features of superficial dermatophytosis. It is graded severe in studies and

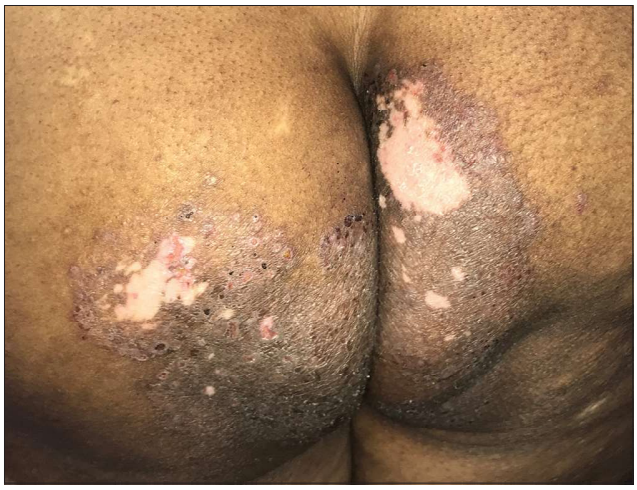

Figure 23b: Vitiligo presenting primarily at the site of active tinea corporis

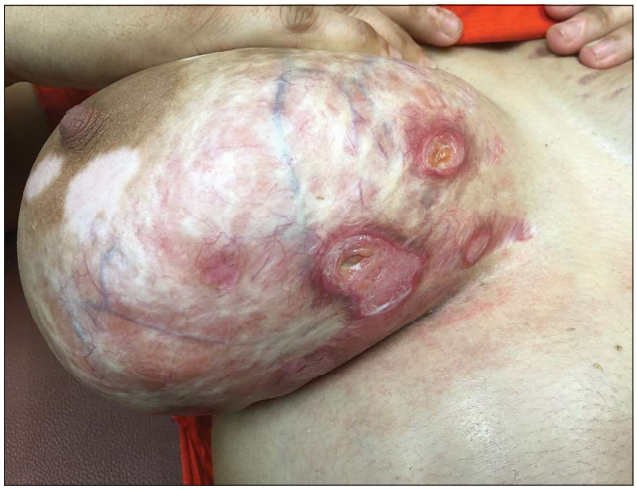

Figure 24: Steroid abuse in dermatophytosis leading to hypopigmentation, striae, atrophy, telangiectasia and ulceration

can be very bothersome for adults and children alike. Patients complain of paroxysms of pruritus lasting for a few minutes a few times a day; some have it for longer periods. Another common symptom is 'burning and itching." In many cases, it gets aggravated by sweating, heat, hot water and after disrobing (atmokinesis), and can disturb sleep.[72] A significant number of patients complain of nocturnal exacerbation of pruritus.[73] Some fully treated patients complain of persistent itching which is often due to xerosis or impaired barrier function due to scratching and topical corticosteroid abuse. Topical antifungal agents like luliconazole have been anecdotally observed by many dermatologists to cause xerosis which can contribute to itching.

Change in the quality of life of patients

Superficial dermatophytosis is now to be viewed like other chronic and recurrent disorders because of its altered nature. The disease is now seen to have a significant bearing on the quality of life, emotions and personal relationships of affected

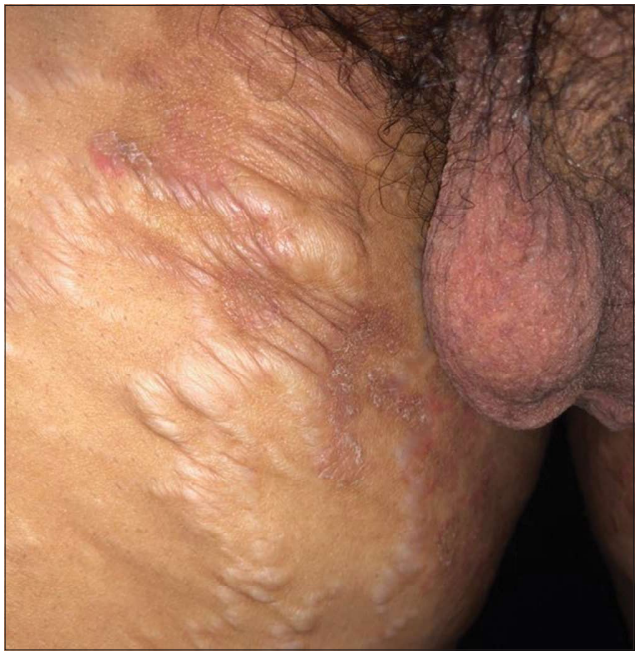

Figure 25: Pseudoedematous appearance of striae

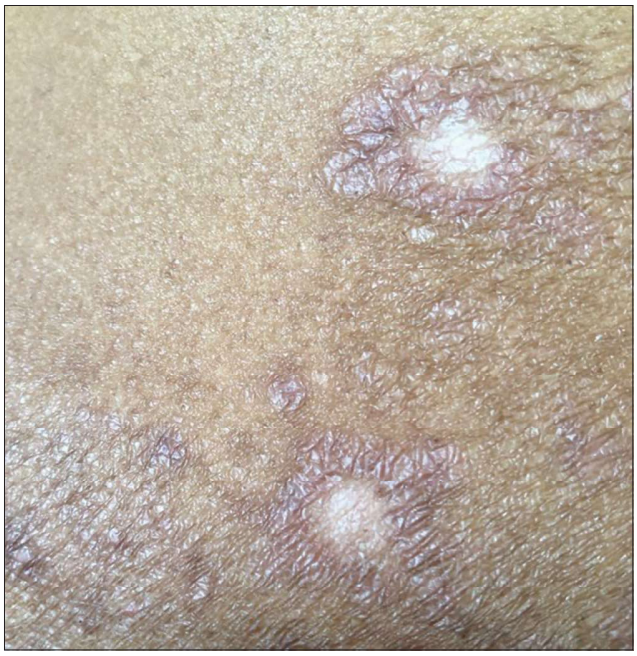

Figure 26b: Close-up of depigmentation at the site of intralesional injection

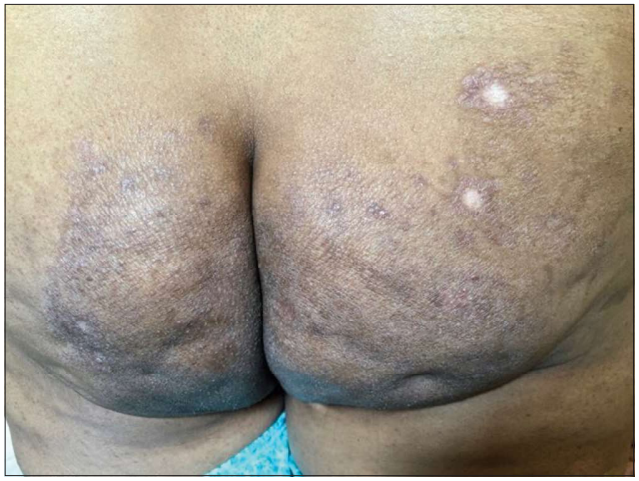

Figure 26a: Depigmentation at the site of intralesional steroid injection

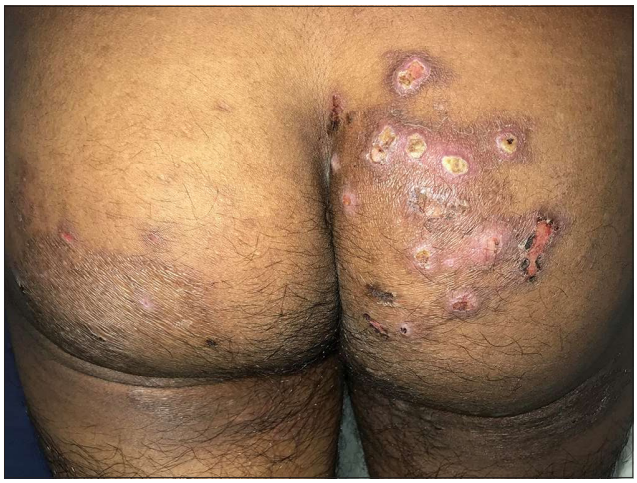

Figure 27: Superficial dermatophytosis with superadded bacterial infection

individuals. It is the experience of dermatologists that many patients with chronic recalcitrant dermatophytosis develop feelings of hopelessness, shame and anger; a minority even express suicidal ideation. It is important to give adequate time to each patient, understand disease impact, and educate the patient about the disease, its management and prognosis in order to improve adherence to treatment and hence their quality of life.[19]

Financial impact

Though dermatophytosis in the current context does not spare any socioeconomic group, it is a common observation that chronic widespread dermatophytosis often affects those from the lower socioeconomic strata of society. A majority of these patients have multiple, similarly afflicted household contacts, resulting in a magnification of the financial burden of treatment. Such patients usually start with self-treatment and are later advised inappropriate medication (including steroid-containing creams) by non-dermatologists or unqualified individuals. The treatment continues with little or no benefit, compelling the patient to finally visit a dermatologist. Oral antifungals like itraconazole and topical molecules like luliconazole, sertaconazole, oxiconazole, amorolfine and ciclopirox olamine are currently preferred by dermatologists. They are perceived as more efficacious but are also much more expensive than the older antifungal drugs and are a significant financial burden to the patient, especially when multiple family members are affected. Cheaper brands manufactured by companies with uncertain credentials may lack efficacy, necessitating multiple visits to dermatologists and adding

to the financial burden. In widespread disease, extensive application of the newer topical antifungals listed above is inconvenient and expensive. Patients often buy less than the required quantity to cut costs and discontinue treatment upon getting symptomatic relief. Other affected members of the household also may use these medications erratically. Such erratic treatment ironically results in their spending larger amounts of money over long periods. This particular patient population harbors smoldering, inadequately treated tinea that becomes an important pool of infection in the community.[74]

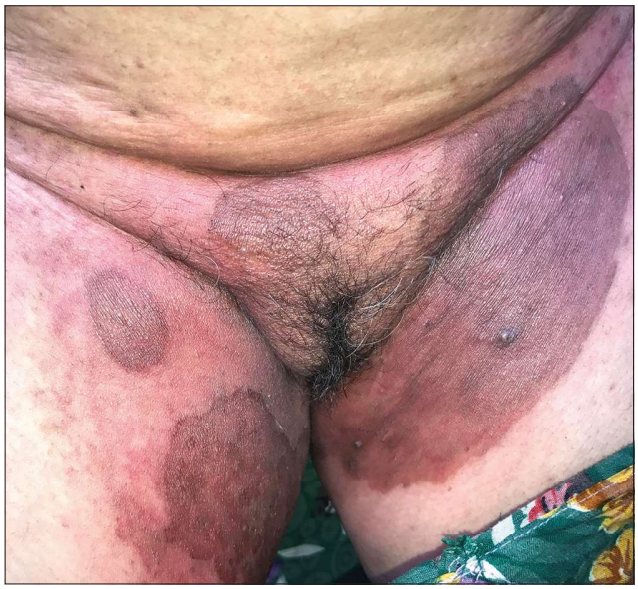

Figure 28a: Irritant contact dermatitis presenting as erythema, edema and brownish pigmentation over the areas of superficial dermatophytosis due to the application of a dithranol-containing cream

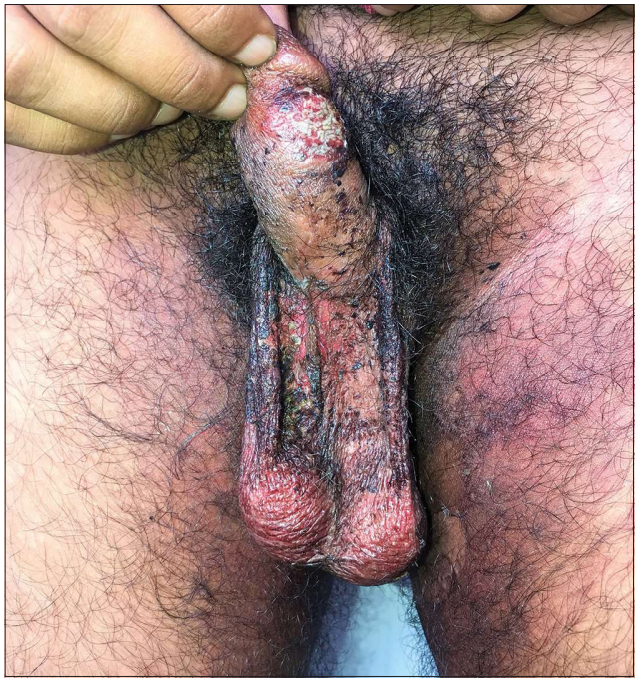

Figure 28b: Irritant dermatitis presenting as erythema, edema, erosions and brownish pigmentation over the areas of superficial dermatophytosis due to application of dithranol-containing cream

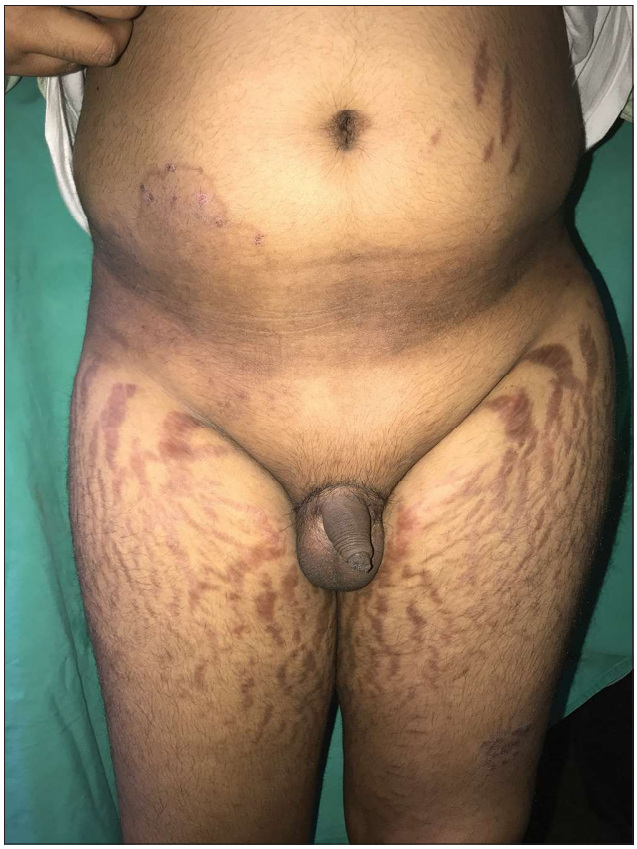

Figure 29: Exogenous Cushing's syndrome presenting with central obesity and broad striae, due to the application of topical steroids in a patient with tinea corporis

Conclusion

The epidemiology and clinical presentations of superficial dermatophytosis in India have undergone a sea change. This importantly includes the rather abrupt change from the *Trichophyton rubrum* to *Trichophyton mentagrophytes* as the predominant species in less than a decade. The disease now occurs irrespective of climatic changes, age, sex and educational or socioeconomic status, and is notable for its unprecedented high rate of transmission amongst family members and close contacts. There is a change in the morphology of individual lesions with varying degrees of inflammation, and a significant number of cases present with steroid-modified dermatophytosis. Despite many years of discussion and documentation of the adverse role of topical steroid containing fixed-dose combination creams, these irrational and often hazardous creams continue to be freely available in the market and are being abused by patients.

There is a stark increase in chronic, recurrent and relapsing dermatophytosis that impairs the quality of life due to severe

pruritus. The disease also has financial implications for families with multiple affected members.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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