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The current epidemic of dermatophytosis in India – lessons to learn for the world

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Key words: Dermatophytosis, *Trichophyton mentagrophytes*, tinea, terbinafine resistance, India, potent topical corticosteroids.

Dermatophytosis (commonly called 'tinea') constitutes a significant proportion of infective dermatoses worldwide especially in developing countries. However, in the past 5-6 years, there has been an alarming rise in its incidence in certain areas of the world, beginning in south east Asia.[1] The rise in numbers has been accompanied by a profusion of atypical presentations of dermatophytoses, adding to the complexities of this dire situation. Although the initial reports of these atypical patterns, combined with reduced responsiveness to standard antifungal therapy, came from India and other countries in south east Asia, gradually reports are emerging from west Asia, notably the United Arab Emirates, and Europe. A major cause of this rapidly unfolding epidemic appears to be the rampant use of irrational combinations of potent topical corticosteroids, antifungals and antibacterial agents.

Steroid-antifungal creams available over the counter and other drivers of the ongoing epidemic of tinea

The unregulated sale and erratic, unsupervised use of topical steroids and fixed-dose combination creams (FDC) containing steroids are considered prime culprits in the unprecedented epidemic of recalcitrant dermatophytosis on the Indian subcontinent as well as in Middle Eastern countries. A large proportion of combination creams contain a steroid, antifungal and an anti-bacterial agent. The most common steroid in these combination creams is the highly potent clobetasol propionate. While short term, supervised use of a mild steroid with an antifungal and antibacterial agent, prescribed in Europe and USA, can mitigate the associated inflammation and itch, it is a double-edged sword when the mild steroid is replaced with a more potent steroid molecule. In resource-poor areas they are most often bought over the counter and used erratically for periods ranging from many weeks to several months. This phenomenon, fuelled by pharmacists, unqualified medical practitioners and inadequately trained general practitioners leads to local immune suppression as well as an altered skin microbiome. Other important factors that cannot be ignored include the use of suboptimal strength and duration of oral antifungal agents and the questionable quality of antifungal drugs.

The following are commonly observed phenomena in countries where there has been a notable increase in the incidence and prevalence of tinea.

1. It is now uncommon for the dermatologist to see patients with pristine tinea as described in Western textbooks. Many of the forms described in the accompanying review are becoming increasingly common, even in developed countries. Multiple members of the family are often infected.
2. A rather quick epidemiological shift from predominance of *Trichophyton*

Continued overleaf…

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rubrum to *T. mentagrophytes* has occurred in India and presumably in the rest of South Asia where identical lesions and easy availability of topical steroid-containing FDCs are reported. In addition there are increasing reports of terbinafine-resistant *T. mentagrophytes*, owing to mutations in the squalene epoxidase (*SQLE*) gene. Notably this change is evident chiefly in only one genotype of *T. mentagrophytes*, ITS genotype VIII, the so called Indian genotype, which has been labelled as a new species, *Trichophyton. indotinae*.[2] Its gradual global march has been documented in several other countries like Iraq, Iran, Germany, Estonia, Switzerland, Finland, Poland, Cambodia[3,4] While thought to be occurring more frequently in migrant populations it is being increasingly reported in individuals with no history of travel to endemic countries.

3. A distinctly reduced responsiveness or even non-responsiveness to the standard dose and duration of antifungal treatment is often seen. There is sometimes partial or complete subsidence of lesions followed by a quick recurrence of lesions upon stopping medications. This 'difficult to treat' tinea has led to experience-based treatment regimens in India and other countries.[5] As investigative tools are not always available in resource-poor settings, infection with this new genotype should be suspected in patients with extensive disease, unresponsive to conventional medications.

Curbing the menace of topical steroid abuse – the way forward

Regular awareness campaigns are essential, targeting health-care workers as well as the general public. The harmful side-effects of topical steroids need to be regularly highlighted. The initial temporary benefit provided by their anti-inflammatory effect is far outweighed by the risk of recalcitrant dermatophytosis and other cutaneous side-effects. The policy makers need to be convinced to allow manufacturing of only rational combination creams after vetting safety and efficacy data.

The importance of frequent and sustained representations to the relevant government authorities and policy makers cannot be overemphasized. Strong support and representations by local, national dermatology and physicians' professional bodies, the International League of Dermatological Societies and the World Health Organization have a vital role in approaching relevant government authorities to ensure strict implementation of 'prescription only' sales of creams containing potent topical steroids. The drug regulators of the countries need to be made aware to ensure a ban on all irrational combinations.

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QUIZ

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Miss C, a 22-year-old from Kandy, Sri Lanka, presented with progressive pigmentation of the hands for a year. The pigmentation particularly affected the finger tips and knuckles on the dorsal aspect as well as the palms (Fig. 1). There was no mucosal or generalized pigmentation.

Working at a local vegetarian restaurant, she felt well and had no past health issues.

Physical examination revealed mild pallor. Her liver chemistry, electrolytes, renal and thyroid profiles were normal. Blood count showed normal white cell count with haemoglobin 9.9 g/dl

What would you expect to see in her blood picture?

a) Macrocytic red cells
b) Microcytic red cells
c) Parasitized macrophages
d) Fragmented red cells
e) Hypo-segmented neutrophils

Answers on page 15.

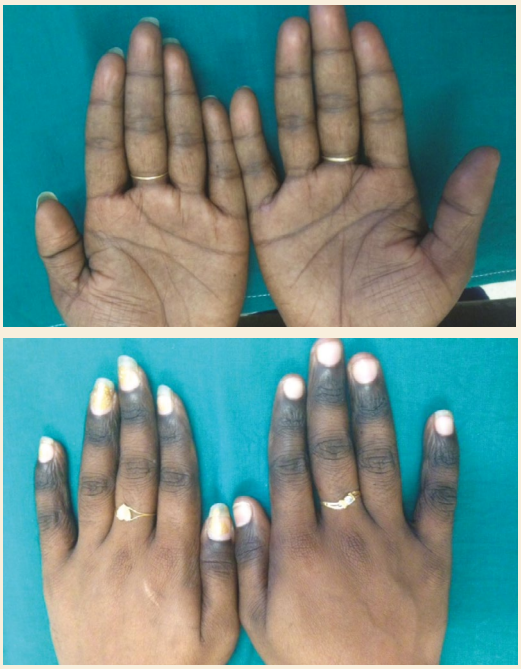

Fig 1. Acral and palmar pigmentation.

Management of dermatophytosis – an illustrated review

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Conflict of interests: None.

Key words: Dermatophytosis, tinea, antifungal, corticosteroids, India, *Trichophyton mentagrophytes.*

Introduction

The clinical presentations of fungal skin infection have recently changed in India and the Middle East, and there are signs that this altered pattern may increase in other parts of the world. Classical textbook presentations are less commonly seen. Inappropriate use of potent topical corticosteroids is a major cause of this epidemic.

Causative organisms

Fungi causing superficial skin infection are classified as **anthropophilic** (adapted to live entirely on a human host), **zoophilic** (living on both animals and humans) or **geophilic** (living in soil).

Three genera, *Microsporum, Trichophyton* and *Epidermophyton* commonly cause superficial infection of skin, hair and nails.

Clinical patterns of dermatophytosis

The most frequently seen morphological patterns in the current scenario in developing countries are discussed in detail. Misuse of topical steroids and a possible role of the predominantly seen *Trichophyton. mentagrophytes* are blamed for the change in morphology. There is no evidence that this variant is implicated in tinea capitis or onychomycosis.

Tinea corporis is dermatophytosis of the glabrous skin, thus seen on the trunk and limbs. Historically, the most common causative organism reported has been *Trichophyton. rubrum* followed by *T. mentagrophytes*. In the present epidemic, however, *T. mentagrophytes* has gradually achieved predominance.

Tinea corporis is characterized by pruritic annular/polycyclic, scaly lesions which gradually spread centrifugally with or without central clearing that may be complete or incomplete (Fig. 1). The level of clinically appreciable inflammation ranges from low to very high, when there is scaling and vesiculation of the edges. Inflammation is more prominent in infections caused by *T. mentagrophytes* and other zoophilic dermatophytes such as *Microsporum* species. It has become common to see multiple, often coalescing and at times bizarre shaped, lesions (Figs 2 and 3).

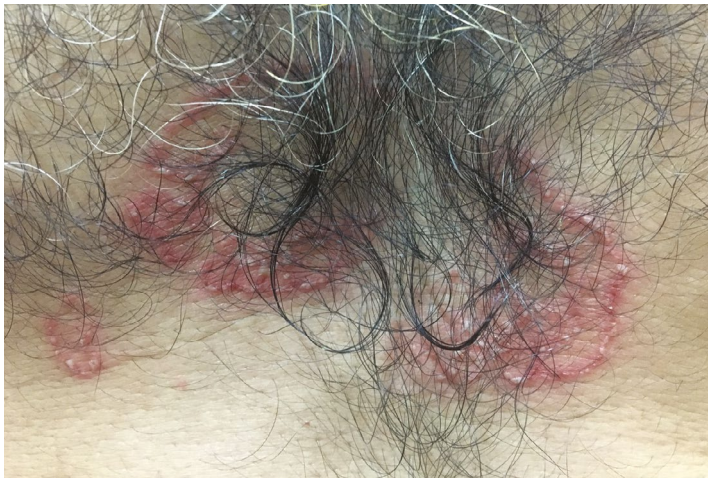

Fig 1. Typical lesion of tinea corporis: Annular plaques with papulovesicles on border and central clearing.

Differential diagnosis:

Psoriasis vulgaris commonly presents as erythematous plaques covered with silvery white scaling. Generally there is no central clearing as in tinea corporis. It is preferentially located on extensor aspects of extremities like elbows, knees and the lower back. Involvement of scalp, especially its margins and that of nails in the form of nail pitting, yellow discoloration, 'oil drop' appearance and onycholysis (variable destruction of the nail) are additional clues.

Nummular (discoid) eczema: The plaques of nummular eczema are often symmetrically placed on the limbs (especially lower limbs) and lack the central clearing seen in tinea.

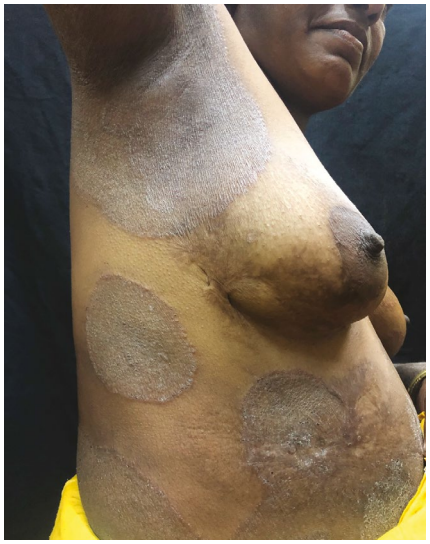

Fig 2. Extensive tinea corporis with large, geographic, scaly, annular plaques.

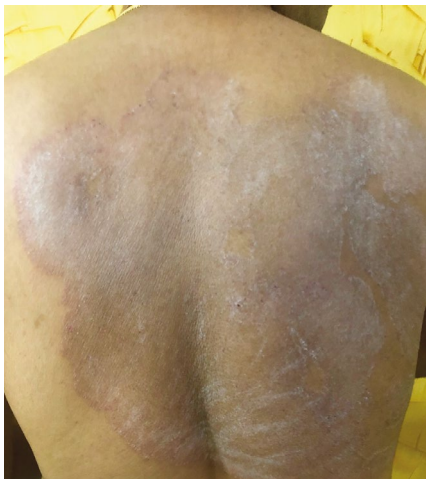

Fig 3. Large single polycyclic plaque of tinea corporis with diffuse scaling.

Impetigo: The circinate lesions of impetigo in children can mimic tinea. The honey coloured crusts in impetigo are, however, characteristic.

Pityriasis rosea : The annular plaques with collarette of scales are characteristically symmetrically distributed in a Christmas tree pattern on the trunk in a typical case.

Tinea cruris, dermatophytosis involving the groin area, is commonly seen in males. However, recently an increasing number of women also present with the disease. Lesions are clinically similar to those of tinea corporis. Extension to adjacent sites i.e. buttocks, the lower back, lower abdomen and pubic area is commonly seen in the present epidemic with

Continued overleaf…

occasional extensive lesions that may involve large areas of the trunk (Figs 4a and 4b). It is common to see tell-tale signs of steroid misuse/abuse in the form of variable linear, broad striae with or without hypopigmentation (Fig. 5).

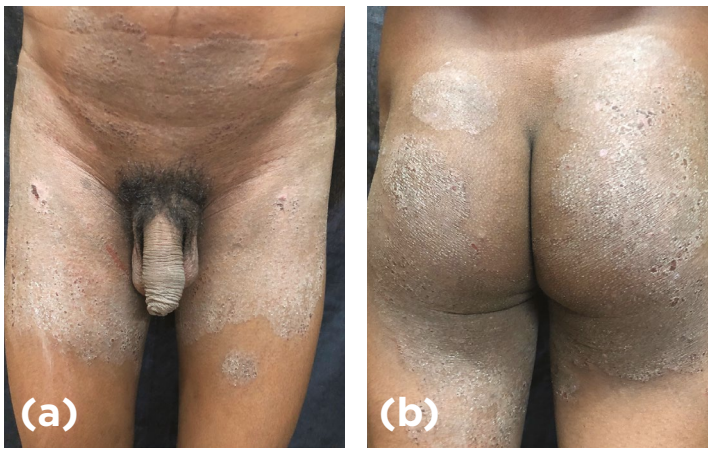

Fig 4. Extensive tinea cruris extending to mid-thigh, lower abdomen, scrotum and distal penis (a) and buttocks (b).

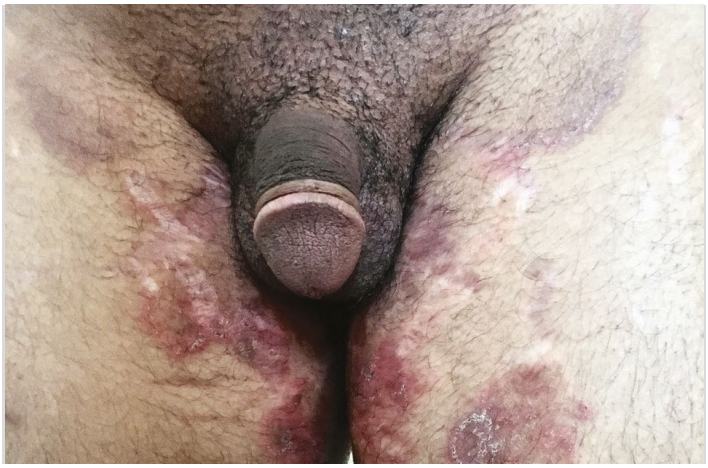

Fig 5. Steroid modified tinea cruris extending to the thigh. Note the hypopigmentation and striae formation secondary to repeated topical steroid application.

Tinea faciei is a dermatophytosis of the glabrous skin of the face excluding the moustache and beard areas in an adult male for which the term ***'tinea barbae'*** is used. Although scaly, annular plaques are classic, patients often present with ill-defined scaly plaques with accompanying acne-like lesions leading to confusion in diagnosis (Figs 6 and 7). Ears may be involved (Fig. 8). Pruritus, burning and worsening upon exposure to sun often lead to a mistaken diagnosis of photodermatosis. Sometimes there is extensive involvement of the face that may be confused with seborrhoeic dermatitis. Widespread involvement can descend to the neck or ascend to invade the hair margin (Fig. 9). The non-inflammatory type of tinea barbae presents as scaly plaques of alopecia with lustreless broken hair stubs. The inflammatory type is characterized by a boggy swelling studded with pustules. It can lead to scarring and cicatricial alopecia (scarring hair loss) of the beard area. In conclusion, tinea faciei is increasingly seen in the current scenario of tinea.

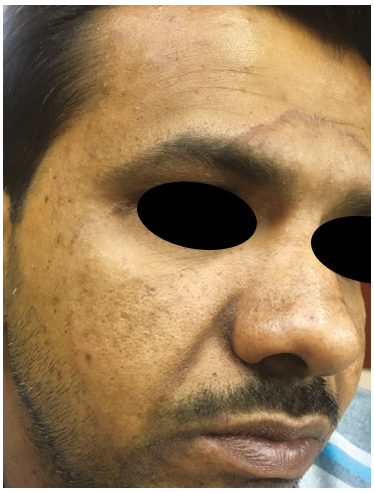

Fig 6. Tinea faciei: An annular plaque extending from forehead to both cheeks and chin. Note the barely perceptible scaling and ill-defined margins on the cheek.

Differential diagnosis:: Tinea faciei needs to be differentiated from seborrhoeic dermatitis, photodermatoses and facial lesions of atopic eczema. Inflammatory lesions of tinea barbae can be confused with bacterial folliculitis and pseudofolliculitis. Non-inflammatory lesions of tinea barbae need to be distinguished from seborrhoeic dermatitis and alopecia areata.

Tinea capitis is an infection of scalp hair caused

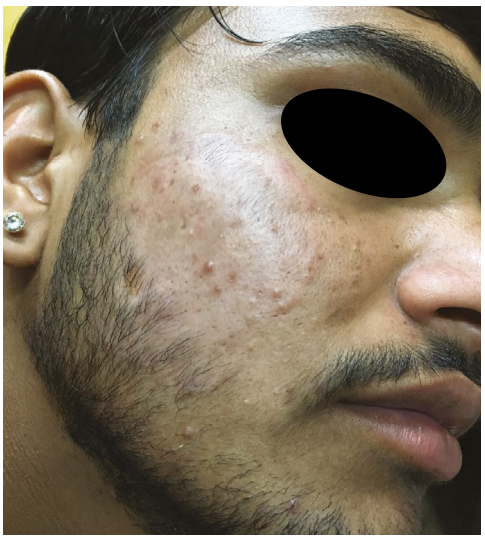

Fig 7. Tinea faciei: An ill-defined annular plaque on right cheek with acne-like lesions.

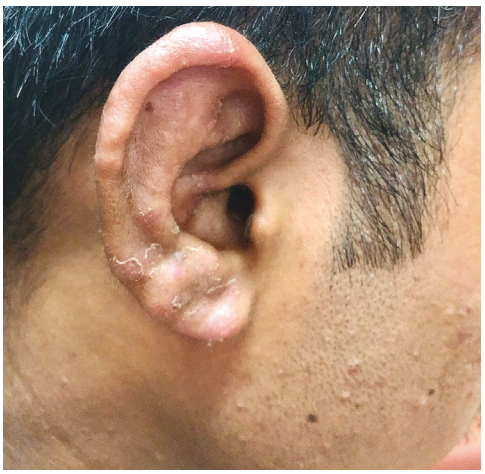

Fig 8. Tinea faciei with involvement of ear.

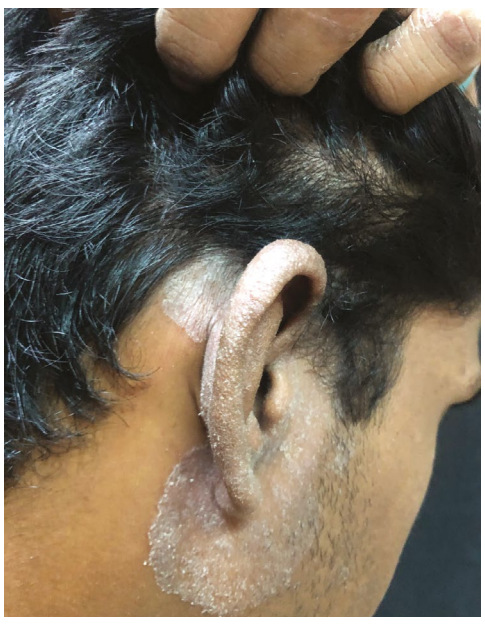

Fig 9. Tinea faciei extending to ear pinna and scalp.

by dermatophytes. It is most commonly seen in children. The characteristic presentation of tinea capitis is patches of alopecia. These can be non- inflammatory (dry scaly patches with broken hair stubs) or inflammatory (boggy swellings and pustules). The inflammatory type of tinea capitis can result in cicatricial alopecia and permanent hair loss.

Differential diagnosis: The non inflammatory types of tinea capitis need to be distinguished from alopecia areata and trichotillomania (habitual plucking of hair).

Inflammatory types need to be differentiated from bacterial folliculitis.

Tinea manuum is a dermatophytosis of the palms. It can be non-inflammatory (scaly type) or inflammatory (vesicular type) and is often associated with fungal nail infection (**tinea unguium**). Tinea manuum is most often unilateral. Accentuation of palmar fissures is considered a characteristic feature. Extension to the dorsal surface of the hand can give rise to annular plaques (Figs 10 and 12).

Both tinea manuum and tinea pedis are often associated with hyperhidrosis.

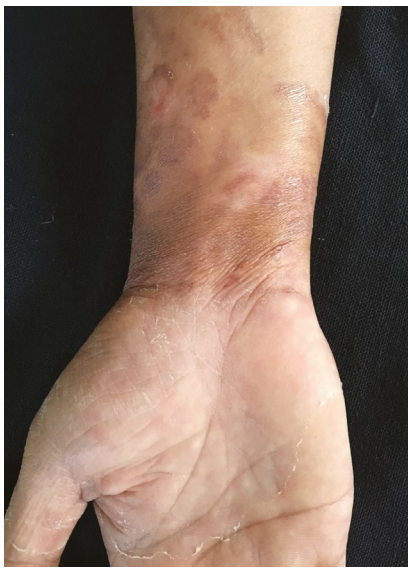

Fig 10. Tinea manuum: A large plaque with scaly border extending from palm and thumb area to volar aspect of forearm. Note the accentuation of scaling in the palmar creases.

Differential diagnosis: Tinea manuum is to be distinguished from contact dermatitis and palmar psoriasis.

Tinea pedis (Athlete's foot) The non-inflammatory type of tinea pedis presents as maceration with fissuring between the toes or as scaling and thickening of skin of the soles. (Fig. 11) The inflammatory types present as vesicles/bullae and may also result in ulceration. Coexisting tinea unguium is often seen.

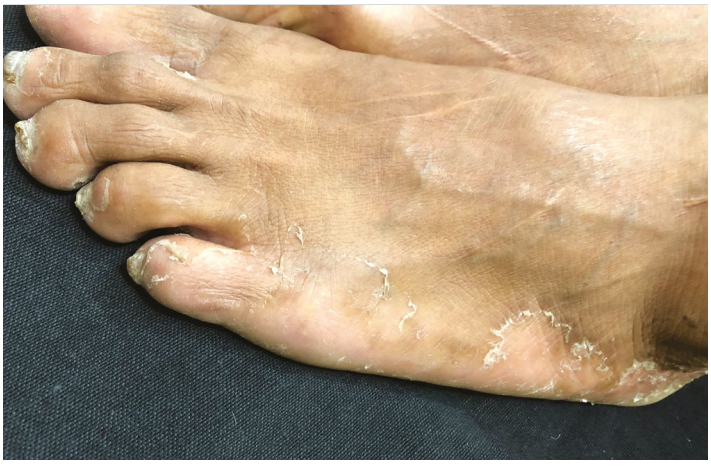

Fig 11. Tinea pedis: Scaling on lateral border of sole extending to dorsum of foot. Note the scaling in the intertriginous spaces between the toes and thickening, discoloration of nails (tinea unguium).

Early detection and treatment of tinea pedis, (especially intertriginous and vesicobullous type) is important as it can serve as a nidus for entry of bacteria resulting in cellulitis. This is particularly important in diabetic patients.

Differential diagnosis: Tinea pedis needs to be distinguished from plantar psoriasis and foot eczema.

Tinea unguium is caused by invasion of nail plate by the fungus. It tends to be chronic and indolent due to slow growth of nails, especially of toenails. The nail plate becomes thickened, discoloured and is lifted up due to hyperkeratosis beneath the nail (Figs. 11 and 12).

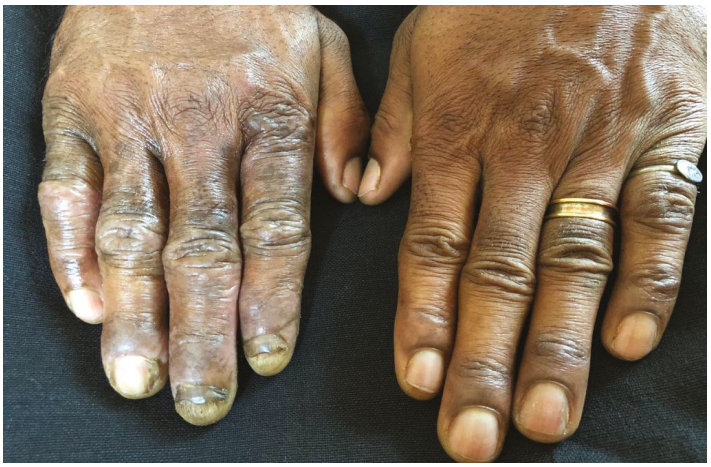

Fig 12. Tinea manuum with tinea unguium: Scaling on dorsum of hand and intertriginous spaces with thickening, discoloration of nails.

Later the nail may undergo partial or total destruction.

Differential diagnosis: Tinea unguium needs to be distinguished from nail psoriasis.

Atypical variants and unusual locations of dermatophytosis:

- *Psoriasiform Tinea*: Use of immunosuppressive drugs can reduce the local inflammation resulting in a lesions that resemble psoriasis.
- *Tinea incognito*: Usage of topical steroids, and sometimes oral steroids, results in reduction of inflammation with loss of scaling and less prominent margins (Fig. 13).

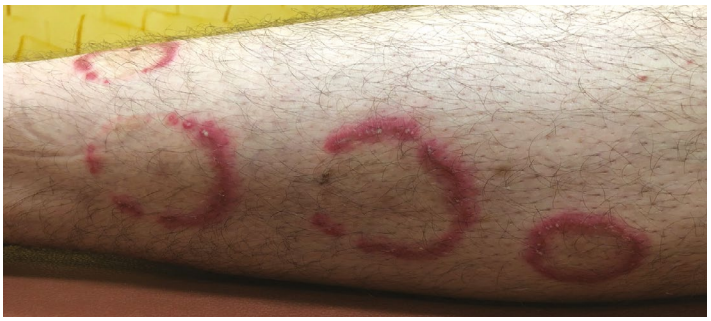

Fig 13. Tinea incognito: Annular plaques, erythematous border with minimal scaling and very few pustules.

- *Tinea pseudoimbricata*: This is predominantly seen in individuals who misuse topical steroid-containing creams. It is characterized by two or more concentric circles within a plaque. This can be explained by subsidence of inflammation and apparent clearing of lesions due to topical steroid application eventually followed by reappearance and often a

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flare up of the lesions due to reactivation of the immune system leading to a subsequent inflammatory response. Erratic application of topical steroids often gives rise to subsequent rings (Fig. 14).

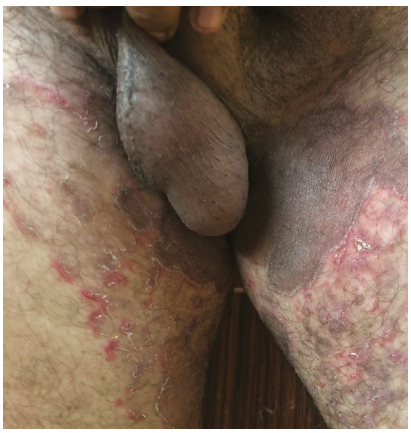

Fig 14. Tinea pseudoimbricata with multiple concentric plaques.

- *Deep dermatophytosis*: Large scaly, nodular lesions without a background of tinea corporis are seen in this type, commonly in immunocompromised patients.
- *Majocchi's granuloma* (a form of deep dermatophytosis): These are pruritic papules and nodules that appear over pre-existing lesions of dermatophytosis and are due to involvement of hair follicles. Local immunosuppression resulting from application of topical corticosteroids is responsible for this (Fig. 15).

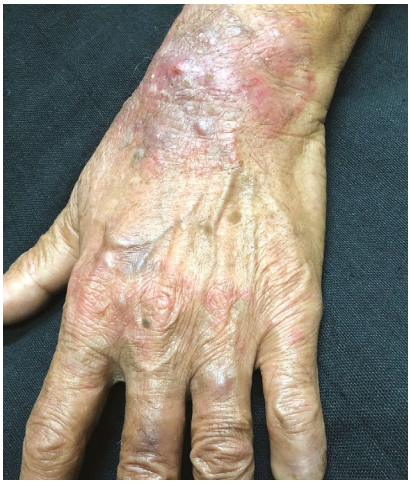

Fig 15. Majocchi's granuloma: An ill-defined erythematous plaque of tinea corporis on forearm and hand with erythematous nodular lesions.

- *Bullous Tinea*: This type occurs due to marked inflammation and is typically seen with zoophilic dermatophytes. It particularly affects the feet.
- *Tinea recidivans*: This term denotes appearance of new lesions, often in the periphery of recently healed plaques of tinea (Fig. 16).
- *Genital dermatophytosis*: Male genital dermatophytosis is also becoming more common with involvement of penis, uncommonly the preputial skin and scrotum (Fig. 17). It is seen less commonly in females, with involvement of mons pubis and labia majora (Fig. 18). Lesions over genitalia may be subtle in which case a potassium hydroxide (KOH) mount is helpful in confirming tinea.

Diagnosis of dermatophytosis

Wood's lamp: Wood's lamp if available is a useful aid to diagnosis; bluish green fluorescence is seen in tinea capitis due to *Microsporum* spp. It is also helpful in diagnosis of early infection in contacts and for mass screening, especially in community settings like hostels and dormitories.

Dermoscopy in trained hands can be helpful in diagnosis of tinea capitis, tinea corporis and tinea unguium.

Microscopic examination: OH mount is a simple bedside investigation to detect dermatophyte fungi. 10% KOH is added to scales or infected hair/nail clippings obtained from patients. It dissolves the keratin and enables visualization of the long, septate, branched hyphae. Addition of special stains such as Parker Ink or Chicago sky blue can improve the visibility of the fungus.

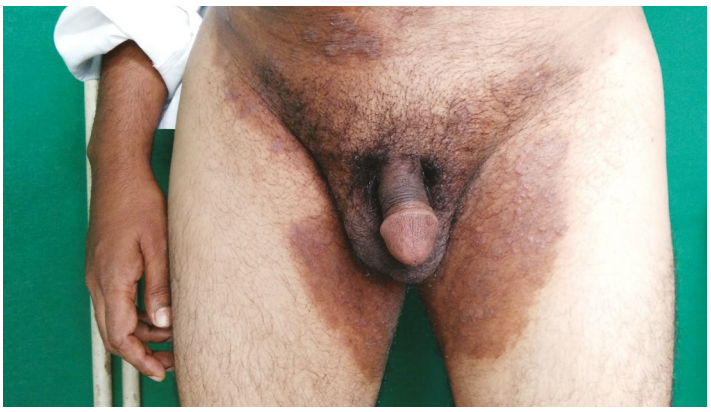

Fig 16. Tinea recidivans.

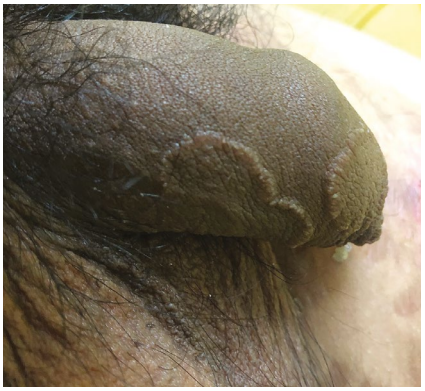

Fig 17. Genital dermatophytosis: well defined annular plaques with erythematous papules/pustules on the border.

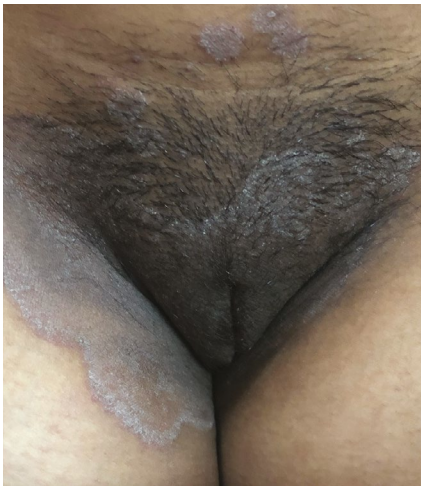

Fig 18. Tinea cruris, genital dermatophytosis: Scaly, annular plaques involving groins and mons pubis.

Culture: Whenever facilities are available, culture can be done on Sabouraud's dextrose agar to determine the causative dermatophyte.

Molecular studies: Polymerase chain reaction (PCR)-based diagnosis and DNA sequencing studies are expensive and not readily available, especially in resource-poor settings.

Antifungal susceptibility tests: Can be done in recalcitrant cases to determine minimum inhibitory concentration (MIC) values for various antifungal agents. However, there can be in-vivo in-vitro dissociation (clinico-microbiologic mismatch) with patients not responding to specific antifungals even when the MIC values are within conventionally normal limits.

Management of dermatophytosis

General measures for prevention of dermatophyte infections include the following advice;

- Wear loose fitting cotton clothes, avoid tight fitting clothes made with synthetic fabric.

- Wash clothes in hot water and/or dry clothes in sunlight.
- Avoid sharing clothes, combs and shoes. Clothes of infected individual to be washed separately when possible.
- Avoid closed occlusive footwear. Dust talcum powder between toes to keep feet dry.
- Look for itchy areas or patches of hair loss in pets at home, and get it treated by a veterinarian. Avoid contact with stray animals.
- Avoid walking barefoot in public areas such as swimming pools and gymnasiums.

Treatment of dermatophytosis

The mainstays of treatment are oral and topical antifungal agents along with oral antihistaminic drugs for control of pruritus.

Commonly used antifungals belong to the following groups;

- **Azoles** including imidazoles and triazoles; e.g. fluconazole, itraconazole
- **Allylamines**: Terbinafine, naftifine
- **Morpholines**: Amorolfine
- **Hydroxypyridones**: Ciclopirox olamine
- **Griseofulvin**

Topical antifungal agents

Topical therapy can be used as monotherapy or with oral antifungal agents, e.g. to reduce spread of fungus in tinea capitis.

Indications for topical therapy

- Limited number of lesions
- Pregnancy, lactation
- Paediatric age group
- Patients on multiple other medications with potential drug interactions
- Presence of comorbidities; diabetes mellitus, hypertension

Oral antifungals: Dosage and duration of antifungal therapy are enumerated in Table 1.

Table 1

Dosages of oral antifungal agents in dermatophytosis

Type of Tinea	Antifungal	Dosage and duration	
		Standard duration and dosage	Suggested modified dosage and duration*
Tinea corporis/ cruris	Itraconazole	100-200mg/day for 2-4weeks (child below 11y 3.5mg/kg/day)	200mg/day for 4-6 weeks Occasional extension up to 8 weeks
	Griseofulvin	500g-1gm/day for 4 weeks (child below 11y 10mg/kg/day)	Duration: 4-6 weeks. Occasional extension up to 8 weeks
	Terbinafine	250mg OD for 2-4 weeks (child , not licensed, 10-19kg 62.5mg OD 20-39kg 125mg OD, 40mg + 250mg OD)	250mg BD for 4-6 weeks
	Fluconazole	150-300 mg/week for 2-4 weeks (child , not licensed, 3mg/kg/day)	150mg thrice a week for 4-6 weeks
Tinea pedis	Itraconazole	200mg/day for 2-4 weeks (child 3.5mg/kg)	200mg/day for 4-6 weeks Occasional extension up to 8 weeks
	Griseofulvin	250mg TDS for 4-8 weeks (child 10mg/kg/day)	Up to 1gm
	Terbinafine	250 mg OD for 2-6 weeks (child as T corporis)	250mg BD for 4-6weeks
	Fluconazole	150mg/week for 2-6 weeks	150mg thrice a week for 4-6 weeks
Tinea barbae	Itraconazole	200mg/day for 4-6 weeks	Occasional extension up to 8 weeks
	Griseofulvin	250mg TDS for 4-8 weeks	Occasional extension beyond 8 weeks
	Terbinafine	250 mg OD for 4-6 weeks	250mg BD Occasional extension beyond 6 weeks up to 8 weeks
	Fluconazole	150mg/week for 4-6 weeks	150mg thrice a week for 4-6 weeks
Tinea faciei	Itraconazole	200mg/day for 3-4 weeks	200mg/day for 4-6 weeks Occasional extension up to 8 weeks
	Griseofulvin	250mg TDS for 4-6 weeks	Dosage may be increased to 1gm/day Occasional extension up to 8 weeks
	Terbinafine	250 mg OD for 3-4 weeks	250mg BD for 4-6weeks
	Fluconazole	150mg/week for 3-4 weeks	150mg thrice a week for 4-6 weeks
Tinea unguium	Itraconazole	200g/day for 12 weeks for toenails Pulse therapy: 400mg/day for 1 week per month Fingernails: monthly for 2-3 months. Toenails: monthly for 3-4 months	
	Griseofulvin	1gm/day. Fingernail: 4-8months. Toenail: 9-12 months	
	Terbinafine	250mg/day. Fingernail: 6 weeks. Toenail: 12 weeks	
	Fluconazole	150-300mg once a week. Fingernail: 3-6 months. Toenail: 9-12 months	
Tinea capitis	Terbinafine	250mg/day for 4-6 weeks (child, as T. corporis for 4-6 weeks)	
	Fluconazole	150mg once a week for 4-7 weeks or 50mg daily for 4-8 weeks	
	Itraconazole	100-200mg daily for 4 weeks (child 3.5mg/kg/day for 2-6 weeks)	

*Modified dosage is based on clinical unresponsiveness to standard dose and duration of antifungals. Increased dosage and duration calls for regular monitoring of patients including monitoring of liver function tests. Decision to increase duration must be based on clinical response to therapy as the increased duration is not yet standardized.
BD, twice a day; OD, daily; TDS, three times a day.

CASE REPORT
Erythrodontia in congenital erythropoietic porphyria

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Acknowledgements: *Dr. Matthew Verheyden has undertaken a clinical clerkship at the Preah Kossamak Hospital, Phnom Penh, Cambodia.*

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Key words: Congenital porphyria, erythrodontia, Cambodia, photodamage, uroporphyrinogen III synthase.

Brief description

A 6-year-old male from a remote Cambodian community, presented with photodistributed bullae, erosions and scarring. His parents reported fragile skin, and blisters beginning from 2 years of age, which continued throughout childhood, resulting in scarring of the light-exposed areas. Further history revealed episodes of reddish-coloured urine in infancy.

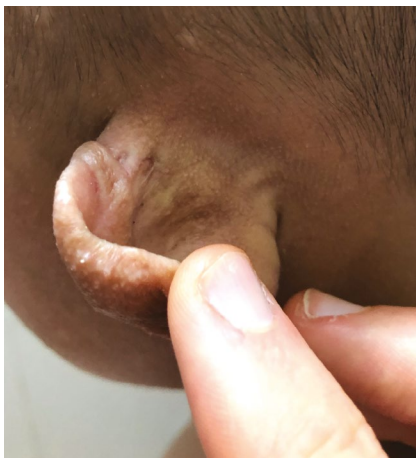

Fig 1. Photomutilation of the ear.

Physical examination revealed hypertrichosis, conjunctival pallor, scarring and photomutilation, particularly of the ears (Fig. 1). Numerous milia were distributed over the face, and nape of neck, and were accompanied by localized areas of scale. Examination of the oral cavity revealed deformities and reddish-brown discolouration of the teeth, which fluoresced with Wood's lamp (Fig. 2). Palpation of the abdomen revealed splenomegaly.

Fig 2. Fluorescence of teeth under Wood's light.

A clinical diagnosis of congenital erythropoietic porphyria (CEP) was made. It was not feasible in this remote resource limited setting to test for porphyrins in the urine, faeces, red blood cells, or to undertake genetic testing. Ophthalmology review was organized to assess for ocular complications, and he was found to have bilateral cataracts, and mild ectropion associated with excess tearing.

Management included emphasis on sun-protective measures such as avoidance of daylight where feasible, sun-protective clothing including densely woven long-sleeved shirts and long trousers, gloves, broad brimmed hats, sunglasses and sunscreens containing physical blockers (zinc oxide and titanium dioxide). Our treatment regimen incorporated low-dose hydroxychloroquine (50 mg daily), isotretinoin (10 mg nocte) for facial, neck and ear papules, alongside sedating antihistamines to assist with pain and sleep. Emollients were introduced alongside topical steroids, with limited effect. He was reviewed at 3-weekly intervals. At the 3-month follow-up, the pre-existing lesions had begun to resolve, and no new blisters had appeared. Facial papules had notably decreased in size and number. Perifollicular erythema and scaling had improved. Hydroxychloroquine has been continued in an attempt to limit progression. Cataracts were surgically extracted successfully with no ongoing sequelae.

Porphyrias arise from aberrations in haem synthesis, resulting in accumulation and increased excretion of porphyrins or porphyrin precursors, due to mutations in uroporphyrinogen III synthase (UROS). CEP is an exceedingly rare autosomal recessive disorder with 130 cases reported in the literature.[1] The enzymatic activity of uroporphyrinogen III synthase is deficient, resulting in accumulation of uroporphyrin 1 and coproporphyrin 1 in red bloods cells, bone marrow and other tissues, and are incorporated into the teeth and bones.[2] When these individuals are exposed to visible light within the Soret band, these precursor molecules release energy, which leads to tissue damage through the generation of reactive oxygen species.[2] Typical features are severe photosensitivity, resulting in blisters and hypertrichosis on sun-exposed sites, with eventual scarring and photomutilation.[3] Eye (inflamed corneas with scarring) and teeth (red-brown teeth which fluoresce with ultraviolet [UV] A) involvement are characteristic. Haemolytic anaemia results in splenomegaly and bone fragility.

Rigorous photoprotection from sunlight and other UV light is essential for all patients,[4] and low-dose chloroquine may assist with the excretion of porphyrins. Multidisciplinary team involvement should be implemented in order to achieve optimal treatment outcomes.

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Effective training for health

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Key words: Training, adult learning, community health workers, chunking, Myanmar, assessment, Burma skin initiative.

Introduction

This article explores the processes involved in delivering effective training. The word "training" is used to describe a learning experience, but for it to be effective it is rather more involved than showing someone how to do a procedure. The old adage "see one, do one, teach one" should be relegated to history!

Training can be described simply as a process which enables someone to learn the skills required to do a job. Therefore, by its nature, the focus of training is to develop specific skills which will have a practical application. By contrast, education tends to have a broader and more theoretical basis. Training is a critical tool within health care to enable the development of new skills that will have an impact on the well-being of patients. Whilst the sessions themselves focus on the acquisition of practical abilities rather than theoretical constructs, the planning of the content of sessions must be evidencebased. For training to be effective there are several critical components that must be included, these will be covered in this article.

Adult learning

When training colleagues or other health workers the starting point has to be an awareness of the principles of adult learning. When adults come to a training session, they do so with a wealth of knowledge, experience and opinions, they are not blank canvases and will want to share their own thoughts. Space and time should be planned into the session so that individuals can learn from each other. Participants are likely to be goal-driven and it will need to be clear what they will get from attending the training and how it will contribute to their job. Carrying out a training-needs analysis prior to working with a particular cohort, will help you to make sure that the training being offered is relevant for that particular group of health-care professionals.

Learning objectives

Once the training-needs analysis has identified the relevant topic, clear objectives need to be set. Learning objectives are the end points that participants must reach by the end of the training session. They can relate to knowledge (what should participants know), skills (what should they be able to do) and/or attitudes (how should they approach a particular undertaking).

Consider this example of training to teach community health-care workers how to treat scabies. By the end of the training session the student will be able to:

1. Correctly diagnose scabies through patient examination and microscopic observation (knowledge and skill);
2. Demonstrate to an individual how to apply antiparasitic lotion (skill and attitude);
3. Engage with families and close contacts to ensure that relevant behaviours are followed after the diagnosis of scabies (knowledge and attitude).

Clear objectives then help in the development of the training plan with all of the content being relevant to achieving those objectives. It is important not to get side-tracked with irrelevant material even if it feels like it might be interesting! The objectives should also guide the amount of time set aside for the training and will also help to determine what sort of resources need to be available to make the training effective. When delivering the training plan, participants should be made aware of the objectives and how each objective relates to developing new knowledge, a new skill and/or a new attitude.

Developing a training plan

The needs of the participants should be at the core of the training plan. Being aware of the background and current skill level of those to be trained, will help in pitching the training at the right level and can also make it easier to draw on their experience to facilitate learning. There should be a clear journey through the content to be delivered with information being given in "chunks" that then build to form a whole picture. Think of creating blocks of learning that build in a logical manner. As the training journey progresses using different methods of learning, sometimes known as blended learning, this is most likely to appeal to the different learning styles of the participants. This could include some information giving, discussions, research using digital devices, group work to solve a problem and practising the hands-on skills that need to be developed. Creating simulated scenarios that replicate real life will allow people to practice their new skills in a safe environment. The energy in training

Continued overleaf…

sessions should be high with plenty of interaction between participants.

Training resources

A training plan can only be successfully delivered with the appropriate training resources. In the example above this training could not be completed without access to microscopes. Visual aids of all sorts should be included and although a Powerpoint presentation may be used, it should not form the central part of the training as it might in a lecture. The only limit to training resources is the imagination and availability; here are a few ideas:

- Using flip charts to capture participant ideas;
- Encouraging students to use their mobile phones to search for answers to particular questions;
- Referring to a handout or learning aid that has been developed specifically for learning a new skill;
- Asking participants to work in small teams to build a structure out of recyclable materials to develop leadership and team-work skills.

Spaced learning

An effective training session will find ways of incorporating review and repetition, this is sometimes referred to as spaced learning. It refers to time gaps between presenting new information and then repeating that information. The time spaces may be quite brief (minutes) or they may be a few weeks. This section will explore this idea in further depth.

Earlier in this article "chunking" was mentioned, referring to the technique of presenting blocks of information which build to form the whole picture. In the example of the scabies training an early objective is for the participant to be able to examine a patient and clinically diagnose scabies. This key point can be referred back to throughout the rest of the training so, for example, clinical slides could be used in the second half of the day to ask participants to identify cases of scabies. When discussing the third objective around behaviour change in those who have been diagnosed with scabies, the key requirements for diagnosis could be discussed again. This is not about repeating information in the same way over and over, but rather finding different ways to bring up key learning points to review whether participants have understood and whether they can remember what they have been taught. If time allows, revisiting training with a review day 4-6 weeks after the initial training is a very useful way of establishing what has been learnt and again re-emphasizing the key messages. This also gives participants the opportunity to practice their new skills and then return to the safety of a learning environment to discuss any issues or problems they may have encountered.

Assessing learning

It may be appropriate to conclude a training session once a review of new knowledge, skills and attitudes has taken place. However, in some instances it will be necessary to undertake some sort of more formal assessment. Knowledge may be assessed using a test. This will usually be written and could be in the form of multiple choice or short answers. But more informal verbal questioning may also be deemed sufficient to test knowledge. Skills must be demonstrated through observation, this is usually a simulated situation but may be conducted in a "live" clinical environment. It is useful to have a competence framework to measure against, the framework giving a baseline for what is deemed safe and proficient. Developing new attitudes can be most effectively tested by observation in practice, but if this is impractical then creating a role-play situation where the participant is required to demonstrate their new skills is acceptable.

Student feedback

Participants like to have a certificate to say they have attended the training session, but before they get this they should be asked to provide feedback for the trainer. There are several approaches that might be used – for example asking participants to use post-it notes to write the best thing and the least good thing about the session, then one element that they will take back to practice and sticking these to one of 3 labelled A3 sheets. A questionnaire is another approach in which specific questions can be asked. As the trainer it is important to then use this feedback to make changes to improve the training session for the next time.

Conclusion

This article came about following a training event held in Myanmar as part of the Burma Skin Initiative conference. A group of 90 Myanmar nurses had attended a series of lectures in the morning followed by some training sessions on fundamental dermatological skills in the afternoon. What was observed by all those present was the energy and enthusiasm present in the room. Whilst the morning lectures were politely listened to, the delivery of information was one way from lecturer to listener. Using adult learning principles, the afternoon training sessions allowed for discussion, hands-on learning, demonstrations and skill acquisition. The content of the workshops was developed following discussions with local clinicians and whilst this was not an in-depth training-needs analysis it did allow us to focus the sessions on topics that were relevant. Informal feedback demonstrated that those taking part enjoyed the sessions, however, there was not the opportunity to test knowledge nor to gauge the impact that practical training workshops had on practice. These learning points will be taken forward for any future opportunities of this type.

Training is not an easy option. The trainer must be confident in their chosen area and ready to get involved with discussion and sometimes disagreement. Meticulous preparation of learning resources and clarity about how these will be used are essential, with the key focus of achieving set objectives which are clinically relevant for those participating in the training.

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Atypical variants of leprosy (Hansen's disease) – a case series

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Conflict of interests: None.

Key words: Atypical leprosy, histoid leprosy, Lucio phenomenon, Lazarine leprosy, erythema nodosum leprosum, skin ulceration, *Mycobacterium leprae, Mycobacterium lepromatosis*, multidrug therapy, India.

Introduction

The saying, "diseases don't read textbooks" is certainly applicable to leprosy. In an era when it is commonly thought that leprosy has been eliminated, we still encounter numerous new cases and more so atypical ones. Although not uncommon, these varied presentations are met with diagnostic challenges which delay multidrug therapy (MDT) and increase the risk of impending complications. This necessitates a high clinical index of suspicion, especially in the tropics and subtropics where leprosy is endemic. In this article we highlight some atypical forms of leprosy which should alert primary care-givers and dermatologists in training.

Description

Leprosy is a chronic infectious disease of antiquity, caused by *Mycobacterium leprae* or *M. lepromatosis*. There seems to be a resurgence of new cases of leprosy in spite of it being declared eradicated in India one-and-a-half decades ago. India now has the highest number of new leprosy cases in the world, followed by Brazil and Indonesia.[1] Infected patients exhibit a wide spectrum of presentations ranging from the typical tuberculoid (TT) to lepromatous pole (LL), with immunologically unstable borderline (BT-borderline tuberculoid, BB-mid borderline and BL-borderline lepromatous) forms in between, depending upon the immune status of the patient.

The cardinal signs of leprosy, which include hypopigmented or erythematous lesions which are hypo – or anaesthetic, thickened peripheral nerves and demonstration of acid-fast bacilli on histopathology, still hold good even today. However, as leprosy is a great imitator, unusual presentations still occur in almost all the types of leprosy.[2]

Histoid leprosy (HL), first described by Wade in 1963, is an unusual type which presents with soft to firm skin-coloured painless dome-shaped papules or nodules that appear on normal looking skin[3] (Fig. 1). Some authors have reported lesions resembling neurofibromatosis and a molluscoid appearance as well.[4] Common sites of involvement include face, abdomen, back, buttocks and extremities. We came across a patient who presented with multiple keloidal lesions of bizarre morphology over the body, who on further examination showed ear lobe infiltration and had a high bacillary load on slit skin smear (Fig. 2). Many authors have also described a giant nodular variety of HL as well, shown in Fig. 3. HL is so called because the microscopic appearance of the nodule shows spindle-shaped cells in a storiform arrangement resembling those seen in dermatofibroma. These cases were believed to be an outcome of resistance to dapsone monotherapy/multibacillary MDT; however, in the modern era most cases appear *de novo*.

Differential diagnosis HL may be misdiagnosed as molluscum contagiosum, xanthoma and skin tumours.[5]

Most cases of HL respond well to MDT, but in addition some cases may benefit from ofloxacin due to the high bacillary load. Ofloxacin is required in cases of rifampicin resistance.[6]

Lazarine leprosy, first described in 1852 by Raphael Lucio and Ignacio Alvarado, denotes a severe and widespread ulcerative phenomenon in patients with type 1 reaction.[7] Usually in type 1

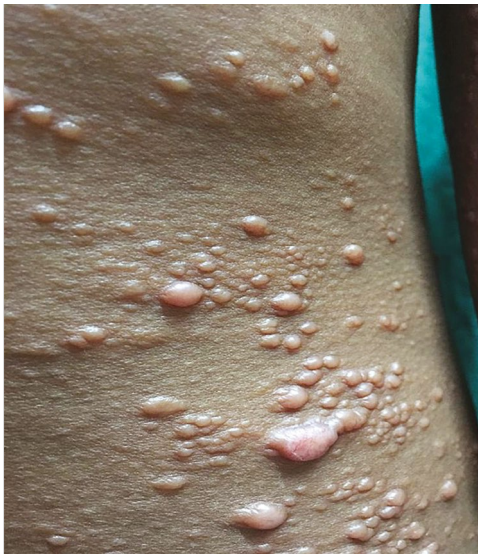

Fig 1. Skin-coloured papules and nodules in histoid leprosy.

Fig 2. Bizarre keloid-like plaque in histoid leprosy.

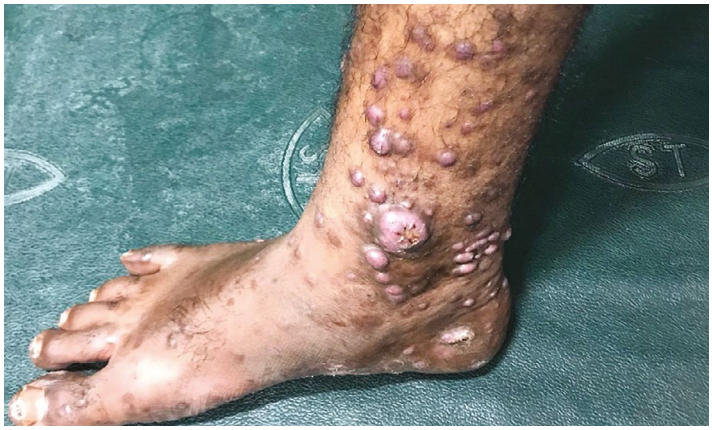

Fig 3. Giant nodular lesion overlying medial ankle in histoid leprosy.

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reaction the pre-existing lesions become erythematous and oedematous but rarely ulcerate.

It is reported that these ulcerations occur more commonly in the BT pole but sometimes even in BL pole.[8] Although its exact pathogenesis is unknown, it is attributed to breakdown of local immunity, protein-energy malnutrition, severe tissue reaction and a high bacteriological load in the BL spectrum. Histopathology shows necrosis due to epidermal attack by *M. leprae*.[9] This form of leprosy shows no localized skin infiltration. The lesions progress in an upward direction, frequently involving the extensor aspects of the extremities and rarely the face and torso. A few patients also report tingling of hands and feet, hypo- or anhidrosis, alopecia of eyelashes and eyebrows, and smooth and rosy skin.[7] The ulcerated areas heal leaving hypochromic, atrophic scars with a thin hyperpigmented border.[9] Systemic corticosteroids are necessary in addition to antileprotic drugs and if the disease remains undiagnosed and untreated patients subsequently manifest with typical skin lesions and nerve involvement.[10]

Lucio phenomenon (LP) is another rare variant of leprosy. The term describes an acute necrotizing pan-vasculitis and is observed in diffuse non-nodular LL, rarely in BL and mainly in a special variant referred to as Lucio-Latapí leprosy or Lepra Bonita (beautiful leprosy), due to the shiny wrinkle-free appearance of the skin. Later, puffiness below the eyes may mimic myxoedema. Lucio leprosy is typically found in Mexico and Central America, and appears to occur in areas where there is a sudden drop in temperature.[7] Inadequately or untreated forms of diffuse leprosy are associated with LP. Clinically LP presents as irregular, bizarre jagged purpuric lesions with superficial ulceration which involve the extremities and, rarely, trunk and face[11] (Fig. 4). It is usually not associated with fever or constitutional symptoms.[12] Histopathology shows patterns of leucocytoclastic vasculitis as the underlying aetiology in few cases, while others show colonization of the endothelial cells by acid-fast bacilli, endothelial proliferation, with or without thrombosis of the small vessels of the superficial dermis and ischaemic necrosis.[13] Treatment of LP requires multibacillary-MDT with additional high-dose systemic steroids in severe cases with additional necrotic erythema nodosum leprosum.

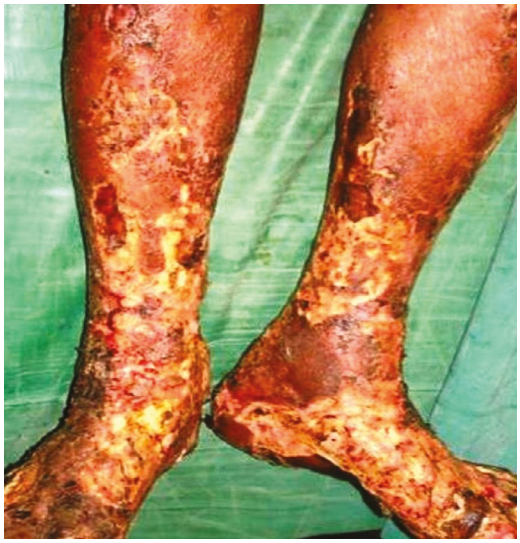

Fig 4. Multiple areas of superficial ulceration.

Differential diagnosis includes vasculo-necrotic erythema nodosum, other causes of systemic vasculitis, disseminated intravascular coagulation, cutaneous tuberculosis, deep mycoses and tertiary syphilis.

Resolution is seen within 1-2 weeks of treatment and ulcers heal with superficial hypochromic scars.[14]

Necrotic erythema nodosum leprosum (ENL) usually occurs in multibacillary LL and BL cases before or after starting MDT. Severe ENL can become vesicular or bullous and breakdown, when it is termed erythema necroticans (Fig. 5). The patients develop severe, deep painful ulcers over the extremities and trunk, which may be associated with constitutional symptoms, visceral involvement and neuritis.[15] The ulcers heal with fibrotic scars. Histopathology shows necrotizing vasculitis of small-and medium-sized dermal vessels with predominant neutrophilic infiltrate involving dermis and hypodermis. This helps to differentiate it from LP.[12]

Erythema necroticans responds well to thalidomide and high-dose steroids along with MDT.

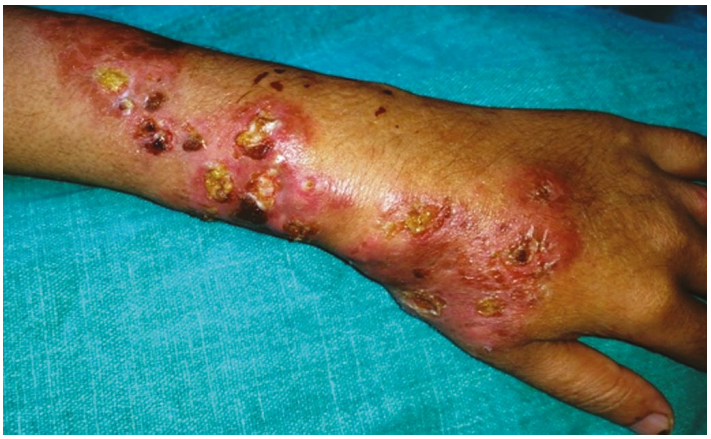

Fig 5. Erythema necroticans.

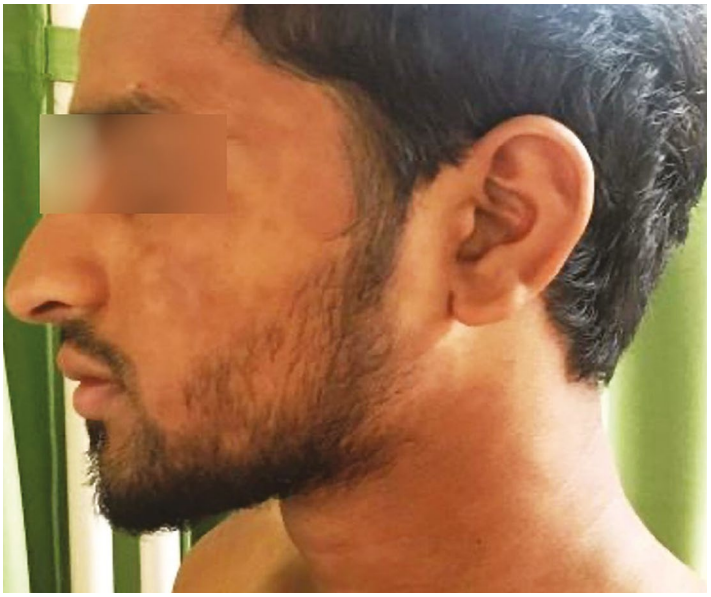

Fig 6. Post inflammatory hyperpigmentation resembling lichen planus pigmentosus.

In our practice we came across a few other atypical presentations of leprosy which we would like to highlight. Of special interest is a case who presented with dark-brown

macules over the face and trunk with minimal pruritus (Fig. 6) which was misdiagnosed as lichen planus pigmentosus(LPP). In LPP the lesions predominate in exposed areas and flexural folds and its course is characterized by exacerbations and remissions, occasionally accompanied by pruritus. A skin biopsy revealed acid fast bacilli and numerous granulomas.[16]

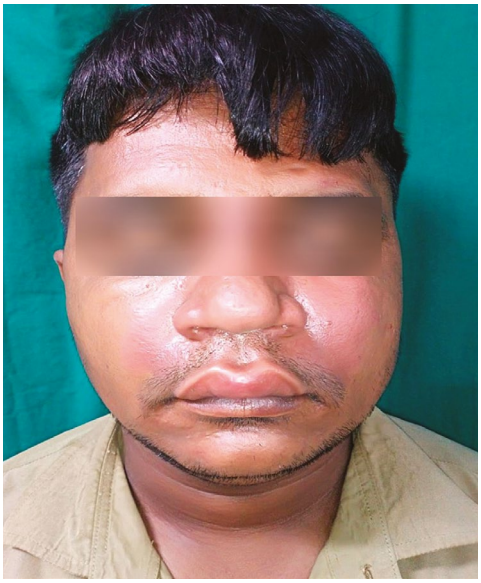

Fig 7. Facial swelling which could be mistaken for angioedema.

Questioning failed to elicit a history of clofazimine ingestion as a cause of hyperpigmentation.

Angioedema is another common disease presenting to dermatologists which can mimic leprosy, especially when patients first present with a reaction (Fig. 7). This case was treated for angioedema but persistence of the oedema and erythema prompted a biopsy which showed type 1 reaction changes. Poor response to antihistamines and persistent nature should raise a suspicion of leprosy. Verma *et al.* reported a case of leprosy mimicking acute urticaria.[17]

Unusual presentations of leprosy continue to be reported from time to time. For example, a few cases of atypical manifestations of leprosy manifesting as angioedema, lupus vulgaris and cutaneous T cell lymphoma have been reported by Raval *et al.*[18] Das *et al.* reported cases resembling granuloma annulare and erythema multiforme.[19] Pruthi *et al.* reported a case of leprosy masquerading as relapsing polychondritis.[20]

Proper history taking and examination, with strong suspicion, is required to diagnose atypical presentations of leprosy. Histological examination is required to confirm the diagnosis when dermatological features are not typical. If the clinical diagnosis is uncertain, skin biopsy is the gold standard to confirm the diagnosis.

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Introducing the GLODERM Trainee Committee

The newly formed GLODERM Trainee Committee aims to promote knowledge equity by improving access to education for dermatology trainees across the world. Our aim is to provide free educational events and opportunities to build networks and collaborate. By improving educational opportunities for trainees we hope to empower future generations of leaders in dermatology from around the world.

Currently, the GLODERM Trainee Committee are running free monthly online seminars delivered by expert speakers and trainees from around the world on a range of different dermatology topics. Looking ahead to the next year, our team hope to deliver educational events focusing on research, leadership and management skills.

To attend and keep up with future events please sign up to the GLODERM mailing list at https://gloderm.org/

The GLODERM Trainee Committee are now looking for speakers for September 2021 and beyond. If you are interested please get in touch by emailing glodermalliance@gmail.com.

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Men who have sex with men and sexually transmitted infections in Jomvu-Mombasa, Kenya

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Conflict of interests: None.

Key words: Men who have sex with men, homosexuality, sexually transmitted infections, Kenya.

Abstract
A small cross-sectional, clinic-based pilot study was conducted during the course of study for the Advanced Diploma of Dermatovenerology (ADDV). It investigated the prevalence and possible contributary factors of sexually transmitted infections (STIs) in men who have sex with men (MSM) in Jumvu-Mombasa, Kenya. One hundred and forty-seven self-identified MSM were enrolled using consecutive sampling. The majority (123; 84%) of participants were aged between 21 and 30 years old. Eighty-nine (61%) were unemployed and just over half (75; 51%) of them earned less than 10,000 KSHS (88 Euro) per month. Overall, 64 (44%) participants had an STI (non-specific urethritis [including chlamydia] 30 (20%); gonorrhoea 22 (15%); HIV 18 (12%); anal warts 12 (8%); anal ulcers 8 (5%) and syphilis 2 (1%). Fourteen patients (10%) had more than one infection. Of the four possible contributory factors investigated only the regular use of condoms was suggestive of a degree of protection but this could have been due to chance (*P*=0.25).

Introduction
According to the World Health Organization, there are over 30 viral, bacterial and parasitic pathogens causing sexually transmitted infections (STIs). Of these, four are easily curable; the others treatable. Gonorrhoea, chlamydia, syphilis and trichomoniasis were responsible for 498.9 million new patients with STIs in 2008.[1] Globally, only less than one out of 20 men who have sex with men (MSM) have access to STI prevention, treatment and care. HIV infection is 13 times higher in MSM than in the general population. MSM suffer from stigmatization, discrimination and even criminalization of their homosexual behaviour. This is certainly the case in Africa where the existence of homosexuality is publicly denied at all levels, including heads of state.[2]

In Nairobi, the HIV prevalence among MSM is estimated at 18.2%.[3] There is very little known about other STIs, hence this small pilot study was conducted on the prevalence and contributing factors to STIs among MSM in Jomvu-Mombasa.

Methods
This was a hospital-based, cross-sectional, descriptive study on the prevalence and the contributing factors of STIs among MSM in a dedicated clinic specifically for MSM in Mombasa County, Kenya (Fig. 1). Consecutive patients attending that clinic were invited to participate in the study.

The study was approved by the Ethical Committee of Kilimanjaro Christian Medical Centre (NO: 1151) and was conducted from 5 September – 30 October 2018.

Results
A total of 147 self-identified MSM patients attending the clinic were enrolled. The social demographics of the participants showed that the majority (123; 84%) were between 21 and 30 years old (Table 1). Seventy-four (50%) of them had received secondary education and 24 (16%) higher education. The majority (89; 61%) were unemployed, 36 (24%) were married, 61 (41%) were separated from their wives and 50 (34%) were single. Seventy-five (51%) earned an income below 10,000 KSHS per month (equivalent to 88.5 Euro).

Fig 1. Mombasa County in Kenya.

Table 1
Social demographic characteristics of the study participants (N=147)

Variables	N (%)
Age group (years)	
21-30	123 (84%)
31-40	24 (16%)
Education level	
Primary	49 (33%)
Secondary	74 (50%)
Tertiary	24 (16%)
Employment status	
Employed	58 (39%)
Unemployed	89 (61%)
Marital status	
Single	50 (34%)
Married	36 (24%)
Separated	61 (41%)
Income per month (KSHS)	
<10,000	75 (51%)
10,000-20,000	72 (49%)

Overall, 64 (44%) participants had an STI (non-specific urethritis [including chlamydia] 30 (20%); gonorrhoea 22 (15%); HIV 18 (12%); anal warts 12 (8%); anal ulcers 8 (5%) and syphilis 2 (1%) (Fig. 2). Fourteen patients (10%) had more than one infection.

The presence of an STI diagnosis was then cross tabulated with potential explanatory variables (Table 2). Of the four potential

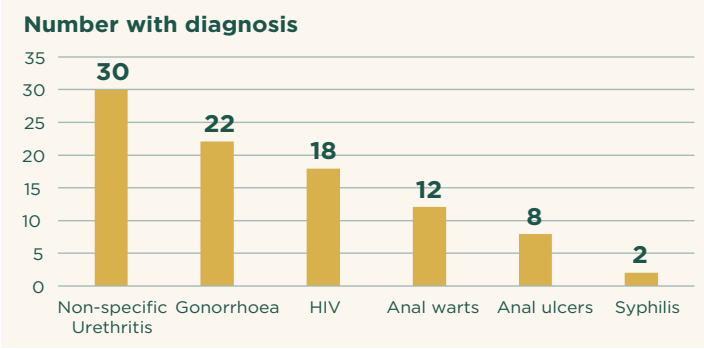

Fig 2. The frequency of STIs observed in participants.

explanatory variables investigated only the regular use of condoms was suggestive of a degree of protection but this could have been due to chance (*P*=0.25).

Table 2

Cross-tabulation of potential explanatory variables with presence of STI amongst MSM patients attending a clinic

Variable	Total	Number with STIs (%)	Chi²	*P*-Value
Age group (years)				
21-30	123	55 (45%)	0.4	0.51
31-40	24	9 (38%)		
Alcohol/drug or substance abuse				
Yes	114	50 (44%)	0.02	0.88
No	33	14 (42%)		
Condom use				
Occasional	61	30 (49%)	1.35	0.25
Regular	86	34 (40%)		
Multiple sexual partners				
Yes	112	50 (45%)	0.23	0.63
No	35	14 (40%)		

Discussion

Amongst MSM patients attending the STI clinic a diagnosis of STI was made in 64 (44%) individuals. A study performed in Canada similarly highlighted that STIs were a common finding amongst MSM.[4] In our study the most frequent diagnosis was non-specific urethritis and, when specific tests could be done, *Chlamydia trachomatis* was the most frequent pathogen. The next most common STI was gonorrhoea, followed by HIV, whilst the least common was syphilis.

Drug abuse[5,6] and lack of condom use[6] have been highlighted as risk factors for STIs in MSM. Within our own study the only possible contributory factor for the presence of STIs was lack of regular use of a condom but this was not significant (49% vs 40%, *P*=0.25).

To the best of our knowledge this is the first report on STIs in MSM in East Africa. The research was approved by the official authorities which represents a key milestone and another strength of the study was the cooperation of the MSM patients. Nevertheless, there are considerable limitations of the study since the sample was only amongst patients attending the special clinic and fear and shame may still have resulted in inaccurate responses. The findings, however, point to the importance of health education and awareness of the risk of STIs in MSM. The medical and general public should accept that MSM are present in Africa too.

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QUIZ ANSWER a) Macrocytic red cells

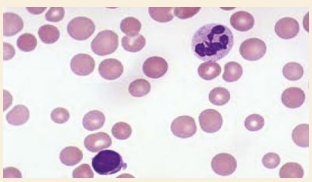

Fig 2. Macrocytic red cells and hyper-segmented neutrophils in peripheral blood film.

Miss C's peripheral blood film demonstrated macrocytic red cells with hyper-segmented neutrophils indicating megaloblastic anaemia (Fig. 2). Her red cell indices supported the diagnosis. Vitamin B12 deficiency, the commonest cause of megaloblastic anaemia, was suspected in her case as the purely vegetarian diet she received long term from her work place was likely to be deficient in the vitamin.

Although her serum vitamin B12 levels could not be assessed (cost not affordable), her skin pigmentation serves as valuable supportive evidence.

Reversible acral pigmentation is a recognized early feature of vitamin B12 deficiency. Pigmentation of the knuckles, palms and palmar creases are well described.[1] In severe cases, the pigmentation may generalize in association with weakness and neuropathy. A similar pattern of hyperpigmentation, particularly affecting the palmar creases, is seen in Addison's disease. The differential diagnosis also includes folic acid deficiency and argyria (silver deposition in the skin), which also favours the palmar creases. Ochronosis (pigmentary changes in connective tissue in patients with alkaptonuria), is also seen in patients taking some antimalarial drugs over a long period. It typically causes darkening of the nailbeds, ear and nasal cartilages, sclerae and conjunctiva but rarely may also cause pigmentation of palmar skin.

Miss C was treated with intramuscular and oral vitamin B12 supplements resulting in marked improvement by the third week of treatment.

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Journal Club

Indian Association of Dermatologists, Venereologists and Leprologists (IADVL) Task Force against Recalcitrant Tinea (ITART) Consensus on the Management of Glabrous Tinea (INTACT)
Rengasamy M, Shenoy MM, Dorgra S *et al. Indian Dermatol Online J* 2020; **11**;502-19.

Superficial fungal infections (dermatophytosis) have always been common in India. However, in the past 6-7 years, more cases have been observed affecting glabrous skin (tinea corporis, tinea cruris and tinea faciei). The condition can become recurrent or chronic, presenting a major public health problem and a significant economic burden. Multidrug resistant forms of fungal species are emerging. Lack of compliance with treatment and inappropriate use of topical preparations containing potent corticosteroids are key factors.

Seventeen dermatologists, with expertise in management of dermatophytosis in India, took part in a Delphi consensus method. Each participated in multiple rounds of anonymous questionnaires, responding to statements prepared by lead experts in the field. The results were finally analysed and reviewed at a face to face meeting and subsequent drafts were circulated.

There was overall consensus that microscopy of potassium hydroxide (KOH) mounts of skin scrapings was recommended when there was clinical doubt. Patients should be counselled to comply with treatment, which in most cases should comprise both topical and systemic antifungals. Topical treatment should be continued for 2 weeks after clinical remission. Terbinafine was recommended as the first-line systemic agent, or itraconazole if terbinafine is contra-indicated or previously ineffective. Topical corticosteroids may have been obtained over the counter, or prescribed for an incorrect diagnosis, and their use should be strongly discouraged.

There is a need for education of health professionals and the public, together with high-quality epidemiological, laboratory and clinical studies in this emerging public health problem, which has relevance to the rest of the world.

CR Lovell

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