

Diagnosis of dermatophytoses still problematic for general practitioners — 10 case studies and review of literature

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ABSTRACT

Dermatophytoses, also referred to as tinea or ringworm, is a fungal infection of keratinized tissues (skin, hair, nails) caused by Trichophyton, Microsporum and Epidermophyton dermatophytes. It presents clinically as an erythematous, scaly, pruritic rash with a well-defined border. Diagnostic errors are not uncommon with this condition. It can have a close resemblance to lesions of another etiology (e.g. psoriasis, discoid eczema) or present atypically due to the prior use of topical steroid preparations (e.g. tinea incognito). A cohort of 10 cases with varying initial misdiagnoses of dermatophyte infection were analysed based on their cutaneous presentations, clinical course, and treatments in order to give guidance for general practitioners.

Forum Derm. 2017; 3, 4: 157–165

Key words: tinea, dermatophyte, corticosteroids, antifungal treatment, tinea incognito

INTRODUCTION

Dermatophytoses, also referred to as tinea or ringworm, is a fungal infection of keratinized tissues (skin, hair, nails) caused by Trichophyton, Microsporum and Epidermophyton dermatophytes. It presents clinically as an erythematous, scaly, pruritic rash with a well-defined border [1, 2]. Diagnostic errors are not uncommon with this condition. It can have a close resemblance to lesions of another etiology (e.g. psoriasis, discoid eczema) or present atypically due to the prior use of topical steroid preparations (e.g. tinea incognito). It is now well known that potent corticosteroids increase the number of fungal hyphae on the cutaneous surface due to a suppressed immune response, all whilst giving the impression that the patient's lesions are improving [3–6]. A cohort of 10 cases with varying initial misdiagnoses of dermatophyte infections were analysed based on their cutaneous presentations, clinical course, and treatments in order to give guidance for general practitioners.

Epidemiology

On a global scale, about 20–25% of the population is affected by cutaneous fungal infections and they generate 4 million outpatient visits in the United States [7, 8]. Epidemiological studies from Japan show dermatophytes were

the most common fungi responsible and accounted for as much as 89.1% of fungal infections [9, 10]. This was followed by Candida (8.4%) and Malassezia (2.4%) infections. Among dermatophytoses, tinea pedis is the most frequent, then in decreasing order, tinea unguium, tinea corporis, tinea cruris, tinea manuum, and tinea capitis including kerion. The most common dermatophyte is Trichophyton rubrum, which accounts for 80–90% of dermatophyte infections [11, 12]. Interestingly, when compared to yeasts and other mycoses, dermatophytes may cause a higher level of tissue damage and inflammatory reaction [11], making it important to investigate the species of fungi when managing patients.

Mechanism of infection

Dermatophytes can be acquired mainly from three sources: from an infected person (via fomites rather than skin-skin contact), from pets or from soil. Environmental factors such as sweating, occlusion, occupational exposure and high humidity also play a role. The clinical course of infection also depends on the fungus "species-specific" ability to elicit a host reaction, host factors and the topographical site of infection [13]. An example of a species-specific factor would be that Trichophyton rubrum initially could start with a mild inflammatory response and chronic course while Microspo-

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Figure 1A. Tinea incognito on the neck and chest



Figure 1B. Tinea incognito on the posterior neck and upper back

rum canis usually causes an acute infection and inflammation with spontaneous resolution [1, 2, 14]. Host factors that predispose to acquiring a dermatophyte infection are: compromised cell-mediated immunity, atopy, ichthyosis, collagen vascular disease and the use of topical or systemic glucocorticosteroids [1]. In the case of systemic immunosuppression, patients can develop a more stubborn and deeper fungal invasion. Dermatophytes infect and grow only in non-viable keratinized structures such as the stratum corneum (tinea corporis), the nail apparatus (tinea unguium) and hair (dermatophytic folliculitis or tinea capitis). Tinea corporis is an infection of the trunk, legs, arms or neck and *Trichophyton rubrum* is its most common causative fungal species [13]. Infection of the face has its own unique term: tinea faciei and infection of the groin is called tinea cruris. When the infection is located on the scalp it is termed tinea capitis and most often presents with pruritic, scaly areas with alopecia. *Microsporum* species is the major cause of tinea capitis, it is mainly transmitted from pets and most frequently occurs in children [7]. Failure to deliver prompt treatment of tinea capitis can result in progression of the infection. It can turn into a deep-seated folliculitis and develop into a kerion or Majocchi's granuloma. Kerion celsi is an inflammatory form of tinea capitis resulting from a T-cell-mediated hypersensitivity reaction to a dermatophyte infection. It is important to know that early diagnosis may prevent unnecessary consequences such as surgical intervention [8]. There is also special type of tinea reserved for fungal infection that is concealed to the eye of the clinician due to the use of topical steroids, this is called tinea incognito. It appears as an ill-defined lesion that is slowly spreading peripherally and characteristically lacks the typical raised and scaly border [15, 16]. Tinea incognito and the problem with inappropriately prescribing steroids dates back to 1968 when it was first described by Ivey and Marks [17]. It can account for up to 39% of all tinea

cases observed making it one of the hardest skin conditions to correctly treat [18].

CASES

Patient 1: Tinea incognito treated as contact dermatitis

A 58-year-old male, generally fit and well, presented with erythematous, scaly lesions with a well-defined border on the chest, neck and upper back (Fig. 1A, 1B) and complained of intense pruritus.

- *Duration of skin lesions:* ~12 weeks;
- *Allergies:* Animal fur, wood resins, no known drug allergies (NKDA);
- *Family history:* No similar skin lesions appeared in his household members;
- *Initial treatment of skin lesions:*
 - Topical: Methylprednisolone, mometasone, clobetasol propionate, bethamethasone dipropionate in combination with gentamicin cream;
 - Systemic: Azithromycin and fexofenadine.

A skin biopsy was taken for direct microscopy with potassium hydroxide (KOH) and culturing. These tests revealed *Trichophyton rubrum*. The diagnosis made at this stage was tinea incognito. Steroid treatment was stopped immediately and oral terbinafine treatment for 6 weeks was prescribed. Topical treatment with ciclopirox cream and shampoo were used for the face and head for 8 weeks. It took about 2 months for lesions to fully resolve (Fig. 2A, 2B).

Patient 2 and 3: Tinea corporis treated as phototoxicity

A 73-year-old female presented with erythematous, exfoliative, oozing eruptions on her face, neck, chest and right subscapular area (Fig. 3A). The oral mucosa was unaffected.

- *Duration of skin lesions:* ~2 months;
- *Allergies:* Nil;



Figure 2A. Resolution of tinea incognita on the upper back after 8 weeks of treatment with oral terbinafine and topical ciclopirox cream and shampoo



Figure 2B. Resolution of tinea incognita on the neck and chest after proper treatment



Figure 3A. Severe tinea corporis on the face, neck and chest



Figure 3B. After 3 weeks of treatment with topical ciclopirox there is a full resolution of fungal lesions on the face and neck and substantial improvement of the lesions on the chest

- *Initial treatment of skin lesions:*
 - Topical: Physiogel, Cutibase;
 - Systemic: Prednisone 30 mg once daily (OD), azathioprine 100 mg OD;
- *Family history:* Admitted on a second survey that her husband developed similar lesions recently. Mycological tests showed *Trichophyton rubrum*. Tinea corporis was established. She was treated with topical ciclo-

pirox and her skin lesions subsided in about 3 weeks (Fig 3. B). Her husband's lesions (Fig. 4A) had the same aetiology upon testing and his lesions improved on the same treatment (Fig. 4B).

Patient 4: Tinea faciei treated as allergy to cat

A 10-year-old girl was admitted due to erythematous lesions on her cheeks (Fig. 5A) and erythematous, inflam-



Figure 4A. Tinea corporis on the neck and chest



Figure 4B. Tinea corporis on the neck and chest after 3 weeks of treatment with ciclopirox



Figure 5A. Tinea faciei on the chin



Figure 5B. Tinea corporis on the dorsal aspect of the hand

matory, pustular skin lesions on the chin and hands (Fig. 5B) that were painful and pruritic.

- *Duration of skin lesions:* Onset ~10 days after contact with a homeless cat and they have lasted for ~8 weeks since then;
- *Allergies:* Nil;
- *Family history:* No family members had similar lesions;
- *Initial treatment of skin lesions:*
 - Topical: betamethasone with gentamicin, mometasone, antibiotic (type unknown), Tormentiol, ichtiol and a vitamin cream.

Mycological tests showed *Trichophyton mentagrophytes granulorum*. Tinea faciei was diagnosed and an antifungal regimen was introduced consisting of: terbinafine (125 mg OD), topical ciclopirox cream (BD) and bilastine (1 tablet OD) for pruritus. After 6 weeks of treatment the skin lesions disappeared.

Patient 5: Tinea faciei treated as herpes zoster infection

A 57-year-old woman presented with erythematous, crusty lesions on the right cheek (Fig. 6).

- *Duration of skin lesions:* ~4 weeks;
- *Initial treatment:*
 - Topical: Acyclovir, betamethasone in combination with gentamicin, clindamycin for ~3 weeks;
- *Allergies:* Nil;
- *Family history:* Nil;

Mycological tests showed *Trichophyton mentagrophytes granulorum*. Tinea faciei was diagnosed. Full remission was observed after 6 weeks of treatment with oral terbinafine (250 mg OD) and topical terbinafine cream.

Patient 6: Extensive tinea genitalis

A 43-year-old female presented with erythematous, well-circumscribed, itchy and burning lesions in the genital region (Fig. 7).



Figure 6. Tinea faciei on the right cheek



Figure 7. Tinea genitalis

- Duration of skin lesions: ~3 weeks;
- Initial treatment: Nil.

Mycology results revealed *Trichophyton tonsurans*. Terbinafine (250 mg OD) and topical ciclopirox (BD) were prescribed for 4 weeks.

Patient 7: Tinea capitis treated as diffuse lupus erythematosus (DLE)

A 65-year-old woman presented with diffuse, inflammatory lesions on the head with patches of alopecia (Fig. 8).

- Duration of skin lesions: ~6 months;
- Allergies: Nil;
- Family history: Nil;
- Initial treatment: Topical corticosteroids and oral prednisone.



Figure 8. Tinea capitis

Due to the lack of improvement of the skin lesions there was suspicion of DLE. Mycological results were performed and yielded *Microsporum canis* and therefore the correct diagnosis of tinea capitis was established.

Patient 8: Tinea capitis treated as DLE

A 7-year-old girl presented with patches of alopecia and pustular folliculitis on the scalp (Fig. 9A).

- Duration of skin lesions: ~8 weeks;
- Initial treatment:

- Topical: Mometasone cream topically and ciclopirox shampoo, clotrimazole cream with fusicidic acid;
- Systemic: Cefaclor, desloratadine.

Due to the lack of improvement (Fig. 9B), the initial diagnosis was DLE. It was later investigated that there is a cat, dog and rabbit in the house. Mycological results yielded *Microsporum canis* and tinea capitis was established. She was then treated with griseofulvin (10 mg/kg/day) and the lesions regressed slowly and about 4 months later there was a recovery (Fig. 9C).

Patient 9: Tinea capitis treated as psoriasis

A 7-year-old girl presented with inflammatory skin changes affecting her scalp (Fig. 10A).

- Duration of skin lesions: ~3 months;
- Allergies: Nil;
- Family history: Nil;
- Initial treatment: Topical corticosteroids.

The lesions enlarged and worsened over time. Mycological tests were performed and resulted in *Microsporum canis* and tinea capitis profunda was established as a diagnosis. Terbinafine (125 mg daily) was started. Ten weeks later the patient's condition improved (Fig. 10B). After 4 months the lesions disappeared.



Figure 9A. Tinea capitis with scarring and alopecia



Figure 9B. Tinea capitis



Figure 9C. Tinea capitis resolution with hair re-growth after 16 weeks of treatment with griseofulvin



Figure 10A. Tinea capitis with extensive alopecia



Figure 10B. Tinea capitis partially treated

Patient 10: Tinea capitis in its inflammatory form — kerion celsi

A 7-year-old boy presented with a nodular mass on the head with alopecia and pustular folliculitis (Fig. 11A).

- Duration of skin lesions: ~4 months;
- Allergies: Nil;
- Family history: Sister and mother both affected with similar lesions.

The Wood's lamp test was positive and revealed a green immunofluorescence, and a mycological test revealed *Microsporum canis*. Kerion celsi was diagnosed. Treatment with terbinafine (125 mg daily for 3 weeks) was initiated. The possible source of infection was the household guinea pig or other children at the boy's school. After 10 weeks

of griseofulvin treatment the nodules regressed and hair began to grow back (Fig. 11B).

RESULTS

Out of the 10 patients included in this case series, 4 cases resulted in the diagnosis of tinea capitis, 2 of tinea corporis, 2 cases of tinea faciei, 1 case of tinea genitalis and 1 case of tinea incognito. Initially the fungal infections were mistaken for other skin conditions.

In the group studied the mean age was 39; (youngest = 7, oldest = 75 years old). The average duration of symptoms (erythema, pruritus, inflammation) was about 7.2 weeks, (shortest = 3 weeks, longest course = over 6 months). The shortest time to proper diagnoses was for tinea genitalis



Figure 11A. Kerion celsi, inflammatory form of tinea capitis



Figure 11B. Kerion celsi after 10 weeks of griseofulvin treatment

(Case 6). This was probably due to the unbearable location and clearer cutaneous signs due to no previous treatment. The longest time for diagnosis was for Case 1 and 9. Case 1 was initially treated with topical corticosteroids for a presumed contact dermatitis; the steroid probably masked the cutaneous signs. In Case 9 psoriasis was the presumed diagnosis and the clinician was probably willing to be more patient to expect effects of treatment. It is to be highlighted that 60% of patients were inappropriately and unnecessarily treated with one or more topical corticosteroid creams.

For initial treatment of presenting skin lesions the most popular were potent topical steroids Mometasone and Bethamethasone in combination with Gentamicin. In the same percentage antibiotics were prescribed. Unfortunately, oral prednisone was used in 40% of cases and this led to immunosuppression during infection. Only 20% of patients had an antifungal in their primary treatment regimen and 80% were treated with some other additional therapies (anti-histamines, vitamins etc.).

DISCUSSION

Diagnostic methods

Many physicians usually do not actually perform extra methods to support their clinical diagnosis when tinea is obviously recognizable. However the problem arises when it is mistaken for another disease. These include skin diseases with similar erythematous, scaly patches such as: rosacea [19], seborrheic dermatitis [19, 20], contact dermatitis [21], eczema [21, 22], discoid lupus erythematosus [23], erythema migrans [24], psoriasis [13, 25] and folliculitis. Other conditions without such traits have also been misdiagnosed such as: scleroderma [26], pemphigus foliaceus [27], impetigo [17] and scabies [12]. Mycological tests used in the diagnosis of dermatophyte infections include microscopy (KOH test), Wood's Lamp Illumination testing, biopsy for histopathological

examination and culturing. Skin scrapings, hair specimens and nail clippings can be examined under microscopy using potassium hydroxide (KOH). This method should reveal the characteristic dermatophytic hyphae or in the hairshaft-uniform spores. In cases where there are dystrophic nails or where dermatophyte infection is still suspected despite negative KOH test biopsy and histopathology can be of good value. Wood's lamp illumination test is used in cases of suspected *Microsporum canis* because under the black light emitted it is seen as a characteristic blue-green fluorescence. However, Wood's lamp test is used to diagnose many other skin lesions that fluoresce such as pityriasis versicolor and erthrasma. Another limiting factor of Wood's Lamp is that it is only specific for *Microsporum canis* and not for *Trichophyton tonsurans*, which is the leading cause of Tinea capitis in North America [28]. Mycological cultures are more accurate however declaring positive results can take 7-14 days [28]. This relatively long duration can also be a contributing factor to why physicians do not perform mycological testing before starting treatment. Generally, we are still limited to basic fungal tests however newer methods are developing that are trying to be more efficient and specific for example nested-PCR which identifies *CHS1* gene in dermatophytes [29]. Taking a thorough history (Patients 4&5 and 10 are good examples of why to inquire about household members) and full body skin examination are also extremely helpful.

Misuse of corticosteroids

Misdiagnoses or uncertainty of skin disease often leads to prescribing unnecessary treatments that can deteriorate the patients' condition further. Prescribing steroids has become too relaxed and its place in infective skin disease is unfortunately not infrequent. Steroid-induced dermatoses are increasing [30]. Steroids suppress inflammation and diminish the appearance of erythematous skin lesions. Ho-

however, this trend in steroid use is not only due to health care professionals but this quick amelioration of symptoms tempts patients to self-prescribe and purchase over the counter corticosteroid-containing creams or borrow them from household members. Their low cost and broad availability makes topical steroids one of the most over prescribed treatments in dermatology [6, 31]. The usual agent is a fluorinated steroid such as Betamethasone dipropionate and Clobetasol propionate, but also milder steroids, such as 1% hydrocortisone cream can suppress tinea so well they may result in tinea incognita [3, 12, 32]. Immunomodulators such as tacrolimus or pimecrolimus can also suppress the appearance of tinea and help it spread [21, 22, 33]. Physicians have to become aware of this and take it into account when examining 'treated skin'.

Treatment

Proper treatment of dermatophyte infection includes: removing the offending immunosuppressive agent (if applicable), reducing the risk of secondary infection to other areas of the body or to other people, initiation of anti-fungal therapy immediately to prevent a deeper invasion, and lastly, alleviation of associated symptoms (e.g. pruritus). Generally, the diagnosis and treatment of fungal infection depends of the type of fungus; therefore, mycological results play a crucial role.

Topical treatments

Superficial dermatophyte infections can be treated topically. Topical terbinafine has been associated with a higher cure rate and more rapid response [34]. Every local treatment course should be later confirmed with negative laboratory results. Cure rates for tinea corporis are high, with infections resolving within 2–4 weeks of topical therapy. Safety of therapy is less of a concern for topical medications than oral medications, as serum absorption tends to be minimal [34].

Systemic treatments

Tinea that is extensive or fails to resolve with topical therapy can be treated with oral antifungals. For refractive and very extensive cutaneous infections extending to the dermis oral griseofulvin should be considered. However, it is not without risk as oral antifungal agents are extensively metabolized in the liver. Oral ketoconazole use for example is no longer approved as being safe for treating fungal infections due to its hepatotoxicity [35]. Oral terbinafine and oral itraconazole should be completely avoided in patients with hepatic impairment. Oral griseofulvin and oral fluconazole can still be used but with caution and national prescribing and drug monitoring policies should be checked before commencing this treatment for pregnant patients and ones with hepatic impairment.

Supportive treatment

It is important to consider prescribing anti-pruritic lotion as supportive treatment. Topical anti-pruritic treatment should not contain medium or high potency corticosteroids, only a low dose can be used if itching is severe. Some physicians prescribe combinations of steroids and antifungals, such as betamethasone and clotrimazole, but it is well known now that betamethasone has a dominant effect over the antifungal agent and an exacerbation of the infection may occur [13].

CONCLUSION

Education for diagnosis and management of dermatophyte infections is needed in the general medical field. The use of topical steroids should be avoided in unclear cases of skin lesions as they can disrupt the clinical picture and result in the spread of an underlying dermatophyte infection. Taking a clear history is always an extremely important factor in the diagnosis of dermatological disease. In cases of persisting and refractive infection, after exhausting the treatment described above, an underlying immune disorder should always be taken into consideration.

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