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REVIEW

Tinea Incognito

Amal Atallah Kokandi

Department of Dermatology, Faculty of Medicine, King Abdulaziz University Hospital, King Abdulaziz University, Jeddah, Saudi Arabia

Correspondence: Amal Atallah Kokandi, Tel +966 126400000 Ext:22009, Email akokandi@kau.edu.sa

Abstract: Tinea incognito (TI), or incognita, are superficial fungal dermatophyte infections that are changed in shape, most frequently because of topical, systemic steroids use and other immunosuppressants. There are different clinical presentations depending on host factors and the species of dermatophyte causing the disorder. Diagnosis can be challenging and might need combined methods. Once diagnosed the treatment is the usual antifungals, either topical or systemic or combined depending on the site, extent, and host factors.
Keywords: Tinea incognito, antifungals, topical corticosteroids, tinea, misdiagnosis

Introduction

Superficial dermatophyte infections are usually diagnosed based on their clinical picture of scaly erythematous patches in an annular arrangement, with active peripheral enlargement and central clearing.[1] Tinea incognito (TI), or incognita, are superficial fungal infections that are changed in shape because of topical or systemic steroids use. Recently other immunosuppressants such as calcineurin inhibitors,[2] tumor necrosis factor inhibitors and other drugs such as fumaric acid esters as anti-psoriatic agents[3] are reported to cause this phenomenon.

This phenomenon has been long recognized that the clinical pictures that resulted from the application of topical corticosteroids (TCSs) to fungal infections were bizarre and difficult to recognize, or atypical in appearance though of unusual extent.[4] This descriptive review tries to comprehensively summarize the causes, different clinical presentations, diagnosis, and management of tinea incognito.

Causes

The most common causes are TCSs. Usually potent steroids are the culprits, but even mild TCSs can precipitate TI.[5] Systemic steroids or other immunosuppressants, topical calcineurin inhibitors[6] were reported to cause the disorder as well. The use of TCSs can be recommended by physicians including dermatologists, family physicians and others such as friends, relatives, and pharmacists.[3] The availability of TCSs without prescription facilitates its use by self-administration and advice from others. In Saudi TCSs can be purchased without prescription. Unfortunately, these can be advised to be used for cosmetic reasons such as skin whitening or combined with other ingredients. Moreover, some creams with unknown ingredients are sold as whitening or glowing creams and they usually contain some TCSs.[7] Hairdressers, herbal salesmen, and saloon beauticians advise about the use of TCSs after hair waxing or other beauty procedures. With infrequent or occasional use, it prevents inflammation associated with these procedures, thus preventing post-inflammatory hyperpigmentation commonly encountered in our area because of skin type. The benefit of improving inflammation and itching leads to the use of steroids for any inflamed or red lesion. If the initial lesion was a fungal infection, it will relieve the symptoms initially and lessens the signs for a while. Then, the lesions will reappear and enlarge in size. More frequent cycles of topical steroid use can lead to large areas of eczema-like lesions. If a detailed history of previous use of TCS is not obtained it can be easily misdiagnosed as eczema leading to more usage of steroids. The frequent use of combination creams of antifungal with potent topical steroids (such as clotrimazole/betamethasone) to treat undiagnosed dermatitis can lead to TI.[8,9] Iraq studies found that the topical steroid abuse by a patient or doctor can provide strong possible explanations for the epidemic of resistant dermatophytosis.[10]

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Some other factors may play a role in chronicity of superficial fungal infections and lead to the development of TI. The dermatophyte invasion ability and pathogen virulence can determine the presentation of the infection. More aggressive strains may induce pustules, vesicles, and central relapse in contrast to less invasive dermatophytes that cause minimal but persistent dermatitis with milder erythema, scaling, and colonization of hair follicles. Other factors include the glandular structures, presence of vellus hairs, proximity to orifice and natural anatomical folds affected by TI. Additional external factors such as excessive washing, tight-fitting clothes, sun exposure and warm wet climate can modify the fungal growth rate and/or inflammatory response.[1] The fungal infection might start in atypical presentation which leads to the use of immunosuppressants such as topical or systemic steroids leading to TI later. The initial type of immune response might be different in some individuals.[11]

The emergence of antifungal resistance[12,13] might be a factor as well. For cases where fungal infections were suspected and not responding to topical or systemic antifungals, with the addition of low yield of fungal cultures, in some institutions this can lead to misdiagnosing and mismanagement of tineas with topical or systemic immunosuppressants with subsequent TI.

Diverse species were reported to be causing TI including the three subspecies: zoophilic (*Trichophyton verrucosum, Microsporum canis*, and *Trichophyton mentagrophytes*), geophilic (*Microsporum gypseum, Microsporum langeronii*, and *Microsporum* ferrugineum) and anthropophilic (*Trichophyton interdigitale, Epidermophyton floccosum, Trichophyton violaceum*, and *Trichophyton rubrum*).[1,3,14–16]

Clinical Presentations

The site of infection is reported to depend on climatic variation and occupation. Tinea corporis and tinea faciei are the most common presentations followed by tinea cruris. Females are mostly affected in the face and males are mainly affected by truncal lesions.[3]

Dermatophytosis is considered an imitator for several common skin problems.[17] The most common presentation of TI is the eczema-like patches and plaques.[18,19] The scales are usually absent or scant. However, a thorough clinical examination might reveal an active edge with some scales. Proper examination might reveal asymmetrical lesions ending abruptly with adjacent normal skin, which is not typical of eczema. The absence of dryness associated with eczema should raise a suspicion of alternative diagnosis.

In immunocompromised individuals a deep plaque or nodular lesions or superficial perifollicular form can develop which can be confused with bacterial infection or prurigo. It occurs mainly in legs of healthy women and is named Majocchi granuloma.[20,21]

In a retrospective study (from 1981–2000) from Poland, including 814 patients diagnosed with tinea, 318 (39.1%) were misdiagnosed initially. The most common misdiagnoses were eczema, pyoderma and allergic dermatitis.[22]

In another retrospective study of 200 cases from Italy of TI cases, the clinical appearances of the infections reported were eczema-like, pyoderma-like, lupus erythematosus discoid-like, rosacea-like, Psoriasiform, purpura-like, finely scaly achromic patches, seborrheic dermatitis-like, lichen planus-like and scleroderma-like.[14]

Additional retrospective study of TI from Spain including 54 children reported that most cases were misdiagnosed as eczema. They reported high prevalence of tinea faciei and tinea corporis clinical forms.[23]

Another survey of 154 cases of atypical dermatophytosis in Italy, the most observed clinical forms were those mimicking impetigo, eczematous dermatitis, lupus erythematosus, polymorphous light eruption, psoriasis, and rosacea. They suggested the use of the term of tinea atypica instead of tinea incognito to include all atypical clinical presentations of fungal infections.[1]

In a more recent prospective study from India of 100 cases of suspected TI, eczema-like conditions were the most common clinical manifestation, followed by inflammatory, autoimmune, and infective conditions. Tinea faciei was the most clinical form.[15]

Another recent prospective study from Iraq for 3 years reported 90 patients with TI. The presentations were of acute eczema-like lesions commonly observed on the trunk and limbs, tinea imbricata-like lesions, mainly on the trunk, papulo-nodular and bruise-like lesions with dusky red erythema, malar area of the face of infants (eczema-like), discoid lupus

erythematosus-like on the face, psoriasiform lesions, chronic hand eczema-like lesions and napkin dermatitis-like lesions. The authors suggested that TI is being seen more in recent years.[24]

Tinea faciei, although rare, can be challenging and misdiagnosed as contact dermatitis, lupus erythematosus and rosacea. If mismanaged and treated with steroids it can be a real challenge.[25] It can present with ill-defined erythema mimicking rosacea, SLE, seborrheic dermatitis, polymorphous light eruption, granuloma faciale, contact dermatitis, psoriasis of the face,[26,27] sarcoidosis, or tuberculosis granuloma annulare.[28]

With increasing use of facial masks, a recently described entity of "Mask Tinea" with the SARS-CoV-2 pandemic which is tinea faciei could also present with TI.[29] Usually, the rash will be misdiagnosed as contact dermatitis and will be treated with topical steroids. In the presence of fungal infection this will usually resolve partially to reappear later and might spread to other sites.[30]

Several other clinical presentations are reported in the literature. Erythroderma has been reported as a presentation for TI as a sole cause or in association with other cause for erythroderma.[31] It can be extensive and disseminated.[15]

It has been reported to mimic treatment-resistant pemphigus foliaceus in a 68-year-old woman.[32] TI was reported to present with bullous eruption,[33] inverse psoriasis,[6] and subcorneal pustular dermatosis.[34]

It can present as gyrate erythema with faint or no scales. This type was reported with treatment of tinea corporis with topical antifungal with potent TCSs[8] and can be mistaken for psoriatic lesions in patients suffering from psoriasis initially due to the lack of scaling with psoriasis treatment.[35,36] TI was reported to mimic ulcerated plantar plaque, mimicking a bacterial infection in children.[37]

A rather rarer type of presentation is folliculitis-like lesions.[18] Tinea incognito can present as a vesicular rash mimicking herpes simplex or herpes zoster infection,[38] in a child as well[39] and was reported to mimic pustular psoriasis.[40] Another reported presentation was polycyclic annular lesions resembling lupus erythematosus.[41]

A case report of invasive dermatophytosis resembling vasculitis was reported in a patient on immunosuppressive therapy (teriflunomide) for multiple sclerosis for two years and TCS.[42]

A reaction has been reported with TI in which periocular inflammation has accompanied TI in the dorsum of a hand.[5]

When it affects scalp it can mimic central centrifugal cicatricial alopecia and alopecia areata.[43]

Another clinical form is called tinea pseudoimbricata or tinea indecisiva because of its clinical resemblance to tinea imbricata caused by *Trichophyton concentricum*. In this form successive waves of concentric rings of scales and erythema are formed with dermatophyte infections (most commonly *Trichophyton mentagrophytes* and *Trichophyton rubrum*) in which they were treated with TCSs or have immunosuppression.[44,45]

Diagnosis

The diagnosis of TI can be challenging and can be delayed. Careful history can raise the suspicion of TI in individuals with the use of topical corticosteroids, calcineurin inhibitors or systemic immunosuppressants and flares following stopping them. Likely careful examination to look for the active scaly erythematous edges of skin lesions can aid in diagnosis.

With increasing use of dermoscopy, it has proved to be a useful diagnostic tool. Several signs were reported to be helpful such as hair changes like Morse-code hairs, deformable hairs, translucent hairs, comma and corkscrew hairs, and perifollicular scaling may be seen despite steroid use.[46,47] Additionally, concentric erythematous areas separated by scales, follicular micro pustules, and black spots surrounded by a white-yellowish halo can be detected.[48]

In vivo confocal laser scanning microscopy is a modern non-invasive method for examining the skin which allows real-time visualization of individual cells and subcellular structures with the highest resolution imaging comparable to the routine histopathology. It has the potential as a non-invasive tool for the diagnosis of TI.[49]

A classically and more frequently used diagnostic tool is the KOH mount of skin scraping and fungal culture in Sabouraud agar with and without antibiotics. The culture usually takes a long time (up to 4 weeks) and has a low yield at times.[2,5] Culture is still the gold standard for the diagnosis of dermatophytosis.[3]

Because of the modified clinical picture of TI, sometimes skin biopsy is performed prior to the mycological studies and periodic acid-Schiff (PAS) stains usually reveal the correct diagnosis.

More recently, other innovative methods are being studied and used such as polymerase chain reaction (PCR) which is more sensitive than cultures and matrix assisted laser desorption/ionization time-of-flight spectroscopy (MALDI-ToF).[2,3]

Treatment

Once the diagnosis is made it is relatively easy to manage. Topical antifungals for small lesions, and systemic antifungals are usually needed for large and deeper ones.[1,2] It is of importance to explain to the affected patient the process to prevent the misuse of TCSs again without medical advice. Topical or systemic terbinafine and azole antifungals (fluconazole, itraconazole, and less commonly voriconazole) are being used usually.

There are increasing reports of emerging antifungal resistance among dermatophytes which might contribute to chronicity and spreading infections. These reports came from India, some parts of Europe, Iran, China, Japan, and USA.[50] It is being increasingly noticed clinically (unreported observations) failure to respond to one antifungal and switching to another to achieve clearance and sometimes prolonging the duration of therapy. This practice is done because of lack of testing for antifungal susceptibility and resistance in routine practice.

Conclusion

Tinea incognito results from topical or systemic immunosuppression leading to different clinical pictures from the usual dermatophyte infection. The diagnosis is usually challenging which might be evident by skin biopsy. Once diagnosed the therapy is the usual antifungal treatment. It should be emphasized to the patient if topical steroids were the cause then stopping them is crucial. Of note, is that antifungal resistance among dermatophytes might contribute to the spreading and chronicity of infections.

Abbreviations

TI, tinea incognito; TCS, topical corticosteroids.

Disclosure

The author reports no conflicts of interest in this work.

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