

Review

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Review

# Tinea Incognito: Challenges in Diagnosis and Management

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**Abstract:** Tinea incognito is a kind of skin infection that is caused by a fungus which has been changed by the use of topical or systemic steroids or other immunosuppressive medications. This alteration in the typical clinical presentation of the infection makes it difficult to diagnose for general practitioners and dermatologists. Delayed diagnosis, spread of the infection to critical body surfaces, resistance to antifungal drugs, and increased costs due to prolonged hospitalization and multiple treatment regimens often complicate tinea incognito. This condition can affect individuals of all ages and genders, but it is more common in children. Tinea incognito is a type of fungal skin infection that can present with various and non-specific clinical features, such as sudden onset of itching in an area that was previously unaffected, making it challenging to differentiate from other diseases such as eczema, seborrheic dermatitis, lupus erythematosus, psoriasis, or other non-fungal skin conditions. The treatment of tinea incognito usually involves stopping the use of topical steroids or other immunosuppressive medications. Preventive measures and management of the underlying fungal infection are necessary and can be achieved with antifungal drugs. Patients should wear loose cotton clothes, use boiling water for laundry, and iron their clothing before wearing them. Additionally, they should avoid sharing bed linens, towels, clothes, and shoes. This review aims to raise awareness of tinea incognito among health practitioners, provide tips for suspecting the disorder, include it in the differentials, and evaluate the available diagnostic procedures. The therapy utilized in published studies is also reviewed.

**Keywords:** tinea incognito; fungal infection; dermatophyte infection; corticosteroid; tacrolimus; pimecrolimus

## 1. Introduction

Tinea incognito (TI) is a type of fungal infection caused by dermatophytes, which can be treated with a topical immunosuppressive agent or systemic steroids. The primary cause of TI is the use of a topical corticosteroid, which is prescribed by a medical professional for a pre-existing disorder or suggested for the treatment of a missed mycoses diagnosis. [1,2]. Topical calcineurin inhibitors like tacrolimus ointment and pimecrolimus cream have also been known to cause TI due to their immunomodulating properties in the area of application. This type of fungal infection can slowly extend and mimic other cutaneous disorders, leading to a misdiagnosis and delayed or unsuccessful treatment. TI is also known as steroid-modified tinea, which has become an increasingly concerning pandemic. The term "incognita" refers to the masking of the clinical signs of a fungal infection [3].

Tinea Incognito (TI) was first reported in 1968 by two UK dermatologists, Dr. Adrian Ive and Dr. Ronnie Marks. They observed a group of patients with unusual skin lesions, resembling seborrheic dermatitis, eczema, lichen ruber planus, scleroderma, folliculitis, rosacea, and psoriasis, that responded to usual treatments. One-third of the cases presented with typical ringworm characteristics. Upon further examination, they diagnosed an underlying fungal infection that was masked by the use of topical steroids or other immunosuppressive medications, justifying the term "tinea incognita." Subsequent publications also employed the term "tinea incognita." [1]. Clinical variations in dermatophytosis can be attributed to the fungi's invasive capacity, infection location,

and humidity. The term "tinea atypica" is suggested for dermatophytosis without typical clinical features. [4]

## 2. Epidemiology

The epidemiology of TI, which represents approximately 40% of dermatophytoses, is not well-established due to underreporting and misdiagnosis, despite being common and having a global distribution. [1,5-7] TI was first described in 1968 and there have been more cases reported in recent years. [1,8] It is more prevalent in tropical and subtropical regions due to favorable conditions for fungal growth, such as high humidity and temperature. [9,10] TI is a condition that can affect individuals of all ages and genders, ranging from 2 to 81 years old. However, it is more commonly found in children, as evidenced by a retrospective review of cases [11]. Another study of 818 cases of TI diagnosed in children at a referral hospital between 1977 and 2006, also supports the higher pediatric incidence. Additionally, young adults are also commonly susceptible to dermatophytosis. [12,13] Based on previous reports, it was found that males had a higher infection rate of dermatophytic fungi. [14] Immunocompromised patients and those with diabetes mellitus (DM) are at a higher risk of developing TI, especially tinea pedis and onychomycosis [10, 14-16].

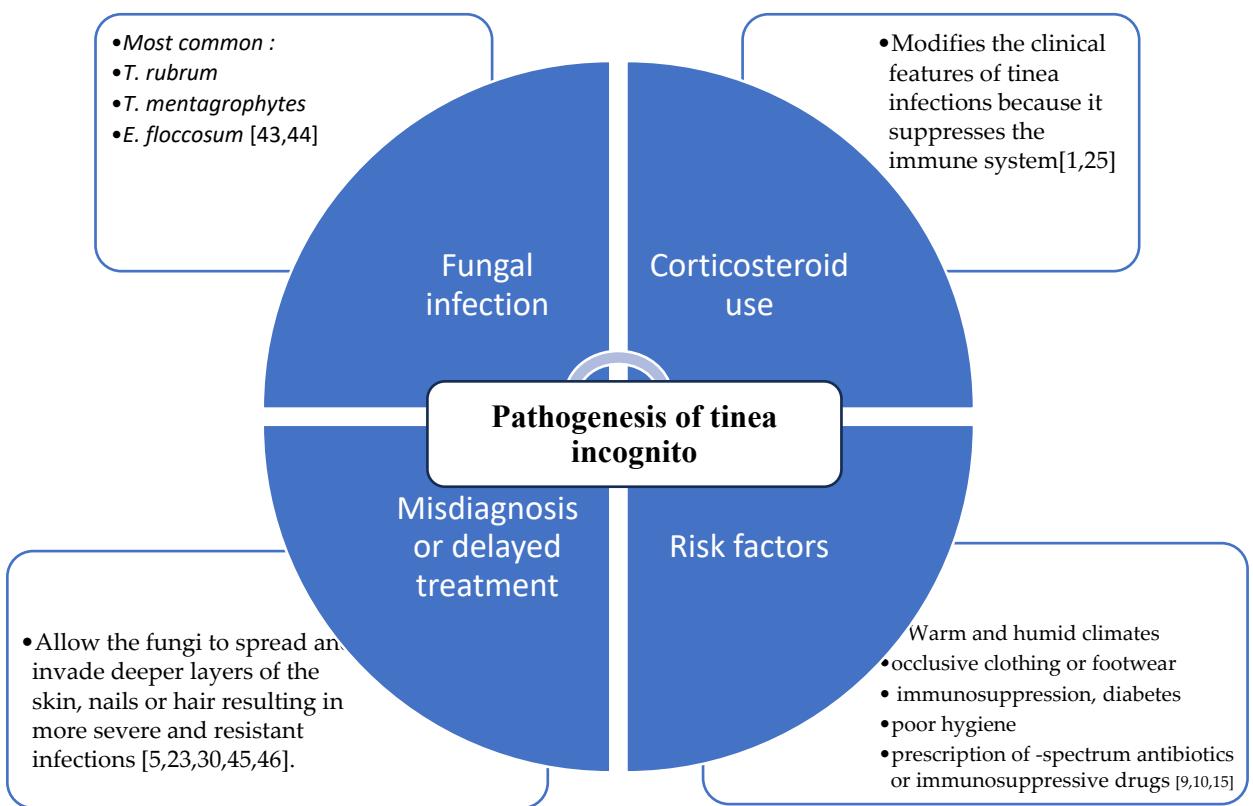
As topical corticosteroids are used to treat an increasing number of dermatologic diseases, the prevalence of TI has also risen. [6,9,17]. Topical calcineurin inhibitors may be less of a risk factor for TI due to their limited use caused by high cost. [9] Tinea infections are caused by a type of fungi that thrive in warm and moist environments. Various factors such as sweating, abrasion, and maceration can contribute to the development of this disorder. Certain populations, such as people living in crowded or unsanitary conditions or those who participate in activities involving prolonged contact with water, like swimming or water sports, may have a higher prevalence of tinea infections. [18-21].

Initially reported, *T. rubrum* and *E. floccosum* were the most commonly isolated dermatophytes. [1]. *T. rubrum* is the most commonly isolated dermatophyte in TI, followed by *T. mentagrophytes*, *E. floccosum*, *M. canis*, *M. gypseum*, *T. violaceum*, *T. tonsurans*, *T. verrucosum*, *T. schonleinii* and *T. erinacei* [21-31]. It is worth noting that *T. rubrum* and *T. interdigitale* are the most common fungi causing dermatophytosis in some areas. [4,32]. *Currently, the prevalent strain in India is T. interdigitale*. [33]. A few studies found *T. mentagrophytes* (44.4%) and *T. verrucosum* (33%) to be also frequently isolated species [31,34]. Trichophyton species are usually the most isolated fungi in studies, except for one where *M. canis* was the most common. [4].

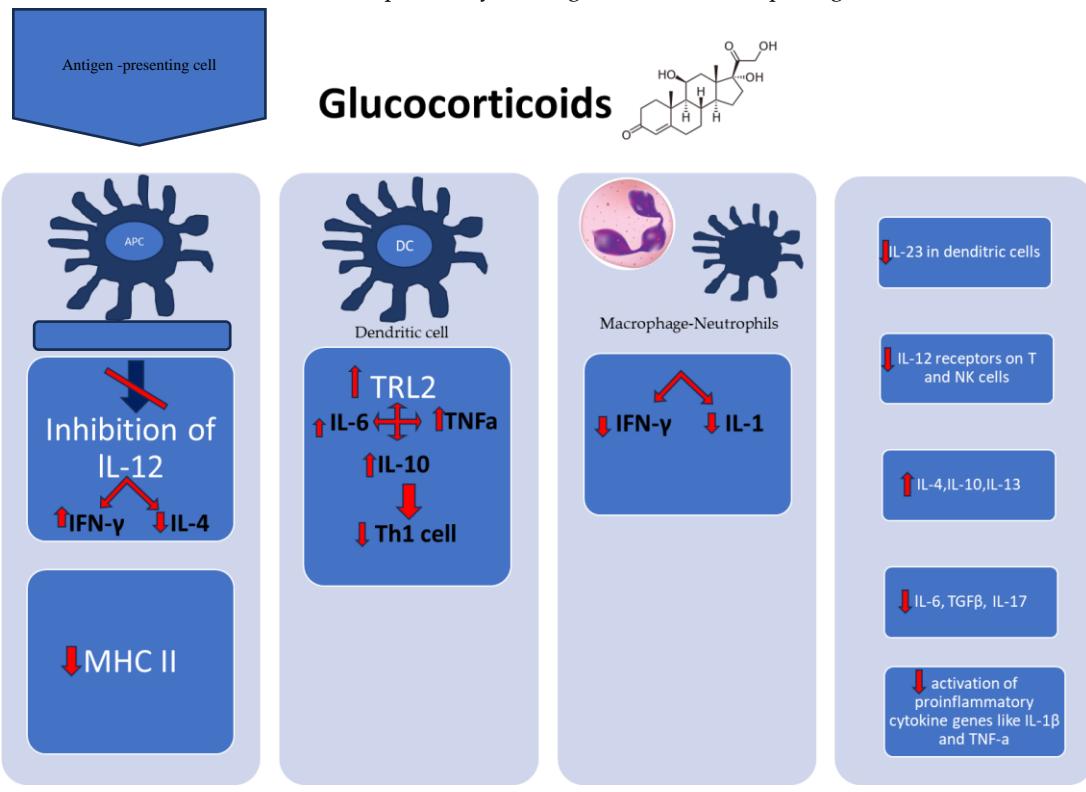
The exact prevalence of TI among immunocompromised patients, particularly those with HIV, is not currently known. It may even be underestimated, especially in developed countries where antiretroviral treatment is initiated early and antifungal medication is prescribed more frequently for other mycotic disorders. [35] *M. gypseum* is a type of fungus that has been found in HIV patients, particularly in areas with limited resources. [36-39]. Exposure to humid soil and dust may also increase the likelihood of developing a *M. gypseum* infection. [40].

## 3. Pathogenesis

The development of TI results from the interaction of fungal infection, corticosteroid use, misdiagnosis and/or inappropriate treatment, as well as individual and environmental factors that affect the sensitivity and severity of the infection. (Table 1). The risk factors that increase the likelihood of developing dermatophytosis include a weakened immune system and damaged skin. [41,42]. The diminished nutrient availability with concomitant reduced oxygenation, and antimicrobial peptides dysfunction (AMP) at the infection site might also contribute to the risk factors for the initial infection.

**Table 1.** The pathogenesis of tinea incognita.

TI and uncomplicated tinea infections are typically caused by the same pathogens, such as dermatophytes *T. rubrum*, *T. mentagrophytes*, and *E. floccosum*. [47]. The pathogenesis of TI is distinct from traditional tinea infections because it is altered by the use of topical steroid creams, systemic corticosteroids, or other immunosuppressive medications. [48]. These medications, particularly when used frequently or in high doses, can weaken the normal immune responses of the skin and reduce the inflammation that fights against fungal infections. This can result in a lowered resistance of the skin at the site of application, which may contribute to the growth and spread of fungal infections. (Table 2) [4,5,14]. Furthermore, dermatophytes metabolize keratin, leading to an inflammatory response. A lack of keratin limits the spread of the fungal infection. Immunosuppressants prevent the inflammatory response, allowing the fungus to thrive. [4,14]. Topical steroids can create a warm and moist environment on the skin, which may lead to the growth of fungi. [1,26]. They can also mask the symptoms of a fungal infection, such as redness, itching, and scaling, making the infection appear less severe. [9]. If left undiagnosed or misdiagnosed as another skin condition, a fungal infection may not be treated in a timely manner. This can cause the fungus to spread and result in a more severe and recurrent infection that is harder to treat. In such cases, oral antifungal medications may be required for a more aggressive treatment approach. [25].

**Table 2.** The effect of topical or systemic glucocorticoids on pathogenesis of tinea [49].

Corticosteroids act on APCs suppressing the inhibition of IL-12 and decreasing MHC II expression. Furthermore, exposure of dendritic cell to corticosteroids induces the expression of IL-6, IL-10, and TNFα through TLR2 activation, that leads to inhibition of Th1 cell activation. Corticosteroid application, shifts the immune response towards a Th2 response. This is mediated through downregulation of IL-12 receptors on T and NK cells and via inhibition of IL-12 production. Th17 differentiation and function may be affected by corticosteroids. Corticosteroids decrease the expression of IL-23 in dendritic cells and IL-6, TGFβ, IL-17 in the joints of arthritic mice. Additionally, corticosteroids upregulate production of IL-4, IL-10, IL-13 and directly or indirectly suppress the activation of proinflammatory cytokine genes like IL-1β and TNF-α. Finally, Corticosteroids reduce macrophage and neutrophils recruitment and IL-1 and IFN-γ release by macrophages [46].

APC-Artigen presenting cell; IFN-γ-Interferon gamma; IL-Interleukin; MHC-Major histocompatibility complex; NK cell-Natural killer cell; TGFβ-Transforming growth factor beta; Th-T helper; TLR-Toll like receptor; TNF-Tumor necrosis factor

#### 4. Clinical Features

A typical scenario begins with a misdiagnosis of TI as eczema, (Figure 1), leading to the use of topical steroids that reduce inflammation and make the condition less irritable. [5,9,46]. The more topical steroids are applied, the more extensive and unrecognizable the fungal infection becomes. Furthermore, long-term use of topical steroids can cause skin atrophy, which complicates the diagnosis of TI. [9]. Thinning of the skin caused by atrophy may resemble the chronic phase of other skin conditions that are treated with steroids for a prolonged period, such as eczema, seborrheic dermatitis, lupus erythematosus, psoriasis, or other non-fungal skin conditions. [23,30,45]. As topical

steroid application continues, it becomes increasingly challenging to differentiate between these conditions and Tinea Incognito (TI). Delayed treatment may allow the fungi to spread and invade deeper layers of the skin, nails, or hair, resulting in more severe and resistant infections.



**Figure 1.** Tinea incognito treated as nummular eczema with topical steroids.

A variation of the typical scenario involves the use of a combination of topical steroid and antifungal cream. In the case of open skin areas, the combination cream could provide an adequate response for treating the fungal infection. However, in areas of occlusion, such as the buttocks, groin, and armpit, the impaired immunity caused by the topical steroid might be sufficient to cause TI, despite the application of a topical antifungal.

TI is a type of fungal skin infection that can present with various and non-specific clinical features, such as a sudden onset of pruritus in a previously unaffected area. The symptoms depend on the type of the underlying fungus and the location of the infected body site. Lesions might appear larger, diffuse, poorly defined, less red and scaly, and more pinkish or flesh-colored. [21,23,31] Table 3 provides a summary of various clinical conditions that need to be distinguished from TI. (Figures 2 and 3).

**Table 3.** Differential diagnosis of tinea incognito [21,31].

TABLE 3: Differential diagnosis of tinea incognito [21,31]	
<b>Granuloma annulare</b>	<b>Rosacea</b>
<b>Discoid Lupus Erythematosus</b>	<b>Morphea</b>
<b>Pityriasis rosea</b>	<b>Lichen Planus</b>
<b>Perioral Dermatitis</b>	<b>Folliculitis</b>
<b>Seborrheic Dermatitis</b>	<b>Impetigo</b>
<b>Erythema Annulare Centrifugum</b>	<b>Scaly Achromatic Plaques</b>
<b>Irritant Contact Dermatitis</b>	<b>Scattered/Extensive Erythematous Plaques</b>
<b>Nummular Eczema</b>	<b>Erythematous Plaques of Concentric Circles</b>
<b>Purpura</b>	<b>Orificial Granulomatous Dermatitis</b>
<b>Psoriasis Vulgaris</b>	<b>Atypical Annular Lesions</b>
<b>Pustular psoriasis</b>	<b>Pustular, Inflammatory Lesions</b>
<b>Pyoderma</b>	<b>Ear- Face Erythema</b>



**Figure 2.** Tinea incognito treated with topical steroids for three months resulting in scar formation.



**Figure 3.** Dermatophyte infection treated with topical combination cream of topical steroid plus topical fusidic acid.

A typical dermatophytosis usually begins as a red circular plaque with a slightly raised scaly border. As the plaque expands, it takes a ring shape with scales at the periphery. [8,15]. In time, multiple itchy lesions can develop and coalesce into patches. These patches may exhibit a poorly defined border, florid growth, and loss of characteristic features due to inflammation suppression. [26].

TI often results in a red, scaly rash that can be itchy, uncomfortable or painful. The rash may appear in patches or spread over a larger area on any part of the body, but it is usually seen in areas where skin folds, such as the groin, the axillae and the inframammary region. Unlike an untreated fungal infection, TI's margin is less elevated, less scaly, and more commonly pustular, and the typical

"ring-like" shape may be absent. Next to the initial plaque, secondary lesions may emerge. Sometimes, a secondary bacterial infection may occur, showing concurrent pustules and impetigo. In some cases, the rash may also be accompanied by blisters or pustules that are filled with fluid and can break, open and ooze. [8,15]. The affected skin may appear lighter or darker than the surrounding skin due to erythematous or hyperpigmented lesions. [25]. In chronic infections, the skin may thicken or develop a leathery texture. It can also cause hair loss due to persistent dermatophytic infection and possible invasion of deeper tissues and hair follicles [52].

A useful clinical tip to differentiate between bacterial infections and TI complicated by a secondary bacterial infection is that in the latter case, hairs can be pulled out of the follicle without the patient feeling any pain.

In several tinea infection cases, the borders of the scaly plaques might not be well-established, and new satellite lesions might appear beyond the blurred border of the initial lesion, due to suppression of the inflammatory reaction at the periphery. More often, especially in the initial stages of the disease, the central remission of the lesion and the presence of a faintly marked ellipsoidal or semi-annular border peripherally is observed. This should raise the suspicion of underlying dermatophytes.

Tinea infections might also develop inflammatory nodules. [26] Concentric circles sometimes appear during fungal infections, which can indicate repeated episodes of fungal expansion. Alongside clinical variations caused by topical agents, systemic immunosuppression can make the condition worse, leading to deep subcutaneous abscesses. This can cause the formation of a secondary granuloma, which is known as "Majocchi's granuloma". Majocchi granuloma is a skin infection caused by fungi that typically affect hair follicles. The most common causative agents are Trichophyton rubrum and Microsporum canis. The infection occurs when the fungi spread into the surrounding tissues after an injury to a hair follicle. This condition is rare. It results in the formation of nodular granulomatous perifolliculitis, which is characterized by an intense inflammatory response. Clinically, lesions appear as granulomata, cellulitis, or plaques. They are often seen on the anterior aspect of the legs, but inguinal, scrotal, vulval, and facial involvement has also been reported. In some cases, patients may have an underlying immunodeficiency state, particularly due to corticosteroid therapy, which mainly affects delayed hypersensitivity reactions. Disseminated skin lesions may rarely occur in immunosuppressed individuals [50,51].

In some cases, the fungal infection can persist for months or years, become systemic, and even lead to onychomycosis. Lesions on the hands may progress towards the nails, causing nail dystrophy and the chronic form of TI [52]. In cases of extensive dermatophyte infection, the patient's sexual partners should also be checked for the infection. Although extremely rare, an id-reaction may occur in TI.

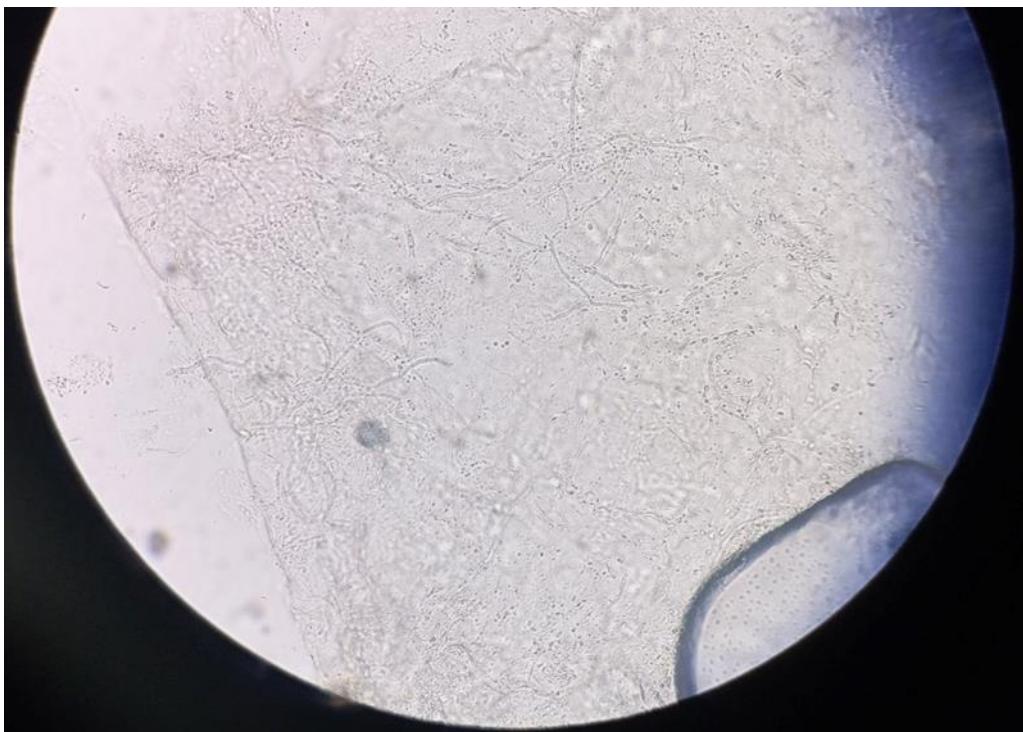
## 5. Diagnosis

Diagnosing tinea incognito (TI) can be quite challenging due to the atypical appearance of the lesions. Physical examination of the affected skin is usually necessary to detect signs of a fungal infection, such as scaling and redness.

Dermoscopy, a non-invasive technique might assist in the diagnosis of TI. The most commonly observed dermoscopic findings in TI cases include dotted vessels, superficial white scales with peripheral distribution, and the presence of a 'moth-eaten' scale with an outward-peeling direction, which appears to be the most specific feature [53]. Tinea capitis is a fungal infection which can be diagnosed through trichoscopy. The most common trichoscopic findings include comma hairs, corkscrew hairs, zigzag hairs, and Morse code hairs. In addition, black dots and broken hairs are frequently seen. Successful treatment of tinea capitis is characterized by a reduction in short broken hairs, corkscrew and zigzag hairs, and later, comma hairs and black dots [54]. Distal lateral subungual onychomycosis can be identified through specific features such as a jagged proximal edge with spikes of the onycholytic area, longitudinal streaks, and subungual hyperkeratosis. [55].

Confirmation of TI diagnosis requires mycological examination after discontinuing topical steroids. To prevent bacterial growth, skin scales should be collected from the periphery of the plaque

and kept dry. [56,57,58] Direct microscopic examination is a quick, simple, and affordable method for screening superficial mycosis. A 10%-40% potassium hydroxide solution (KOH-10% for skin scrapings and 40% for nail clippings) is applied. Positive scrapings are indicated by the presence of hyphal filaments, with or without arthroconidiospores. Hyphae can be seen between epithelial cells. [54,55] The accuracy of the diagnosis depends on the quality of the sample and the skill of the physician [56]. Adding either 36% dimethyl sulfoxide (DMSO) or Chicago Sky Blue stain to the KOH solution will stain the fungal hyphae blue against a pink background, providing better visualization of the fungal elements [59]. Fluorescent staining using diaminostilbene is the most sensitive microscopic method for specimen visualization. [60]. Fluorescent microscopy combined with Calcofluor or Blankophor improves identification speed, ease, and safety [61]. During fluorescent microscopy, fungal filaments and spores appear as blue-white when exposed to ultraviolet light. Chorazol black E staining increases specificity to about 98% [62].



**Figure 4.** KOH preparation of fungal elements.

Fungal culture is the most reliable method to diagnose TI, but it may not always be accessible. [57] Utilization is important, especially in stubborn or severe cases. [63] To identify the fungus responsible for an infection, experts rely on the appearance of the colonies.

In cases where a person is suspected to have tinea, a skin or nail biopsy may be necessary to confirm the diagnosis. The biopsy will be examined under a microscope using stains such as periodic acid-Schiff (PAS) or Gomori - Grocott methenamine silver, which will highlight any fungal elements present. Although the biopsy appearance may vary, certain elements support the diagnosis of tinea. The histopathology of tinea incognito is similar to other fungal infections, with visible fungal hyphae between cornified cells in the stratum corneum (also known as the 'sandwich sign'), compact orthokeratosis, and neutrophils in the stratum corneum. [64,65].

Polymerase chain reaction (PCR) and nucleic acid sequence-based amplification are two of the more recent diagnostic tools [45]. Uniplex PCR can detect and identify fungi with overlapping characteristics of culture, sensitivity, and specificity [66]. Multiplex PCR can detect 21 dermatomycotic pathogens with DNA using agarose gel electrophoresis. Other techniques include gene-specific PCR, sequencing of rRNA gene, chitin synthase encoding gene, PCR fingerprinting, and DNA hybridization.. [66]

The matrix-assisted laser desorption ionization-time of flight mass spectrometry (MALDI-TOF-MS) is a diagnostic technique that detects proteolytic degradation products resulting from fungal activity [56]. Reflectance confocal microscopy is another method used for diagnosing tinea. It offers in-vivo imaging of the epidermis and superficial dermis at the cellular level to detect fungal infections. [67,68]

## 6. Treatment

The treatment of TI typically involves stopping the use of topical steroids or other immunosuppressive medications. Preventive measures and management of the underlying fungal infection are necessary and can be achieved with antifungal medications. Patients should wear loose cotton clothes, use boiling water for laundry, and iron their clothing before wearing them. Additionally, they should avoid sharing bed linens, towels, clothes, and shoes. Obese patients or those with hyperhidrosis should be encouraged to lose weight and apply topical aluminium salts or anticholinergics to reduce sweating.

The ideal pharmacological treatment should be highly effective and provide long-term prophylaxis against relapses. It should also have potent anti-inflammatory action, minimal adverse events, low cost, and be safe to use in pregnancy and lactation, renal and hepatic failure. Treatment modalities include oral or topical antifungals or a combination of both, depending on the severity and extent of infection, as well as the type of the culprit fungi. [3] Combinations of various antifungal groups should be used to prevent resistance from emerging.

Topical antifungal medications are typically the first line of treatment for mild cases of tinea infections and for localized and primary dermatophytic infections. (Table 5). Antifungal creams, gels, or lotions, which contain agents like terbinafine, clotrimazole, miconazole, or econazole, can be used to treat fungal infections. The medication is applied to the affected area(s) twice daily for several weeks until the infection disappears. Treatment typically lasts for several weeks. Additionally, bland antipruritic lotions can also be applied. [44].

**Table 5.** Topical antifungal medications [44].

**Table 5: Topical antifungal medications [44]**

Agent		Preparations	Duration of use
IMIDAZOLES	Clotrimazole	Cream, lotion	4-6 weeks
	Econazole	Cream	4-6 weeks
	Miconazole	Cream, lotion	4-6 weeks
	Oxiconazole	Cream, lotion	4 weeks
	Sertaconazole	Cream	4 weeks
	Luliconazole	Cream, lotion	2 weeks
	Eberconazole	Cream	2-4 weeks
	Efinaconazole	Solution	Up to 52 weeks in co-existing tinea
ALLYLAMINES	Terbinafine	Cream, lotion, gel	2 weeks
MORPHOLINES	Amorolphine	Cream, lotion, lacquer	2-3 weeks (up to 6 weeks for foot mycoses)
Intravaginal Agents			
	Clotrimazole	Cream 1%	5g daily for 7-14 days
		Cream 2%	5g daily for 3 days
	Miconazole	Cream 2%	5g daily for 7 days
		Cream 4%	5g daily for 3 days
		<u>100mg vaginal suppository</u>	<u>One suppository daily for 7 days</u>
		<u>200mg vaginal suppository</u>	<u>One suppository daily for 3 days</u>

<b>IMIDAZOLES</b>		<u>1,200mg vaginal suppository</u>	<u>One suppository daily for 1 day</u>
	Tioconazole	<u>Ointment 6.5%</u>	<u>5g intravaginally in a single application</u>
	Butoconazole	<u>Cream 2%</u>	<u>5g intravaginally in a single application</u>
		<u>Cream 0.4%</u>	<u>5g intravaginally daily for 7 days</u>
	Terconazole	<u>Cream 0.8%</u>	<u>5g intravaginally daily for 3 days</u>
		<u>80 mg vaginal suppository</u>	<u>One suppository daily for 3 days</u>

In cases where the infection is severe or widespread, or involves hair-bearing areas, oral antifungal medications may be prescribed. (Table 6). Terbinafine, itraconazole, and fluconazole are medications that have been shown to be more effective than griseofulvin. Treatment duration varies depending on infection severity and patient response, but usually lasts for several weeks to several months. [44].

**Table 6.** Systemic antifungal agents [44].

<b>Table 6: Systemic antifungal agents [44]</b>				
<b>Class</b>	<b>Representative Drugs</b>	<b>Pediatric dosing Per kg Body Weight Dose</b>	<b>Adult Dose</b>	<b>Mechanism of action of systemic antifungal agents</b>
<b>Heterocyclic benzofuran</b>	Griseofulvin	15–20 mg/kg/day (microsize suspension) 10–15 mg/kg/day (ultramicrosize suspension)	500 mg/day	Inhibition of fungal mitosis
<b>Imidazoles</b>	systemic ketoconazole			Inhibition of ergosterol synthesis
<b>Triazoles</b>	Fluconazole, itraconazole, voriconazole, posaconazole, isavuconazole	6 mg/kg/week 3–5 mg/kg/day	150–450 mg/week 200 mg/day 150 mg orally in a single dose *	Inhibition of ergosterol synthesis
<b>Allylamines</b>	Terbinafine		250 mg/day	Inhibition of lanosterol and ergosterol synthesis
<b>Echinocandins</b>	Caspofungin, micafungin			Fungal cell wall synthesis inhibition
<b>Antimetabolite</b>	Flucytosine			Inhibition of nucleic acid synthesis
<b>Others</b>	Tetrandrine			
* For vulvovaginal infection				

Systemic antifungal agents can have adverse effects. Itraconazole may lead to elevated liver enzymes and gastrointestinal issues. [29] Terbinafine has been linked to a condition called neutropenia or agranulocytosis, which is a reduction in the number of white blood cells. [28]. Laboratory evaluation should be considered before starting treatment, and monitoring should be performed regularly. It is important to review the patient's medical history because systemic antifungals can interact with other medications prescribed for concomitant disorders.

The emergence of resistance to antifungal drugs is becoming a problem [69]. Information on dermatophyte resistance is limited compared to systemic infections. [69,70,71,72]. There is a need for novel therapeutic agents that can target the mechanisms by which fungi limit the immune response and promote the re-establishment of cell-mediated immunity. [49].

## 7. Recurrence

Patients may purchase combination formulations (steroids with antifungal and antibacterial agents) or over-the-counter medications to prevent recurrence [9,21]. Literature suggests that most TI patients self-treat or rely on advice from friends and family for treatment [21,31]. Using these modalities can actually increase the risk of a recurrence instead of providing preventative measures.

Physicians should emphasize the importance of completing the prescribed treatment in order to completely eradicate the infection, even if the symptoms improve earlier. Additionally, patients should visit their healthcare provider to ensure that the treatment was effective and to prevent the possibility of relapse. It has been reported that patients with impaired epidermal barrier functionality, such as those with atopic dermatitis or those with immunosuppression due to malignancies like leukemia or lymphoma or due to the use of immunosuppressive agents for autoimmune disorders such as lupus erythematosus or rheumatoid arthritis, are at higher risk of recurrent infections. [73-75].

## 8. Financial Considerations

It has been observed that TI leads to an increase in the number of consultations and laboratory investigations. It's possible that treatment for dermatophytosis may take longer and require more expensive antifungal medication. In India, a recent analysis of treatment costs showed that patients who use topical steroids spend 40% more on treatment than those who don't use steroids. This was true for both patients who had never used steroids before and those who had used them in the past [3]. The epidemiology of dermatophytosis in India indicates that the high temperatures and humidity prevalent in the region promote the acquisition and perpetuation of fungal infections. Therefore, these results may not be applicable to other regions with different climatic conditions. However, the additional costs associated with the treatment of these infections should raise concerns regarding the increased morbidity and economic burden in all geographical settings.

## 9. Conclusion

The clinical presentation of Tinea Incognito (TI) can be unusual and highly variable. The lesions may not appear as expected for a typical fungal infection, making it difficult to diagnose and treat. Furthermore, the use of topical steroids or other immunosuppressive medications may make the affected skin thinner and more fragile, increasing the risk of injury or secondary bacterial infections. Misdiagnosis of TI can result in delays in treating the underlying fungal infection correctly. Therefore, diagnosing TI requires a high index of suspicion, and additional tests such as skin scrapings, cultures or other diagnostic tests may be necessary to confirm the presence of a fungal infection. Early and accurate diagnosis is crucial to prevent the spread of infection and ensure that appropriate antifungal treatment is administered.

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**Conflicts of Interest:** The authors declare no conflict of interest.

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