REVIEW

Tinea corporis: an updated review

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Abstract

Background: Tinea corporis is a common fungal infection that mimics many other annular lesions. Physicians must familiarize themselves with this condition and its treatment.

Objective: This article aimed to provide a narrative updated review on the evaluation, diagnosis, and treatment of tinea corporis.

Methods: A PubMed search was performed with Clinical Queries using the key term 'tinea corporis.' The search strategy included clinical trials, meta-analyses, randomized controlled trials, observational studies, and reviews. The search was restricted to the English language. The information retrieved from the mentioned search was used in the compilation of the present article.

Results: Tinea corporis typically presents as a well-demarcated, sharply circumscribed, oval or circular, mildly erythematous, scaly patch or plaque with a raised leading edge. Mild pruritus is common. The diagnosis is often clinical but can be difficult with prior use of medications, such as calcineurin inhibitors or corticosteroids. Dermoscopy is a useful and non-invasive diagnostic tool. If necessary, the diagnosis can be confirmed by microscopic examination of potassium hydroxide wet-mount preparations of skin scrapings from the active border

of the lesion. Fungal culture is the gold standard to diagnose dermatophytosis especially if the diagnosis is in doubt and results of other tests are inconclusive or the infection is widespread, severe, or resistant to treatment. The standard treatment of tinea corporis is with topical antifungals. Systemic antifungal treatment is indicated if the lesion is multiple, extensive, deep, recurrent, chronic, or unresponsive to topical antifungal treatment, or if the patient is immunodeficient.

Conclusion: The diagnosis of tinea corporis is usually clinical and should pose no problem to the physician provided the lesion is typical. However, many clinical variants of tinea corporis exist, rendering the diagnosis difficult especially with prior use of medications, such as calcineurin inhibitors or corticosteroids. As such, physicians must be familiar with this condition so that an accurate diagnosis can be made and appropriate treatment initiated.

Keywords: butenafine, dermatophytosis, fluconazole, itraconazole, naftifine, ringworm, terbinafine.

Introduction

Tinea corporis, also known as 'ringworm,' is a superficial dermatophyte infection of the skin, other than on the hands (tinea manuum), feet (tinea pedis), scalp (tinea capitis), bearded areas (tinea barbae), face (tinea faciei), groin (tinea cruris), and nails (onychomycosis or tinea unguium).[1] Tinea corporis is most commonly caused by dermatophytes belonging to one of the three genera, namely, *Trichophyton* (which causes infections

on skin, hair, and nails), *Microsporum* (which causes infections on skin and hair), and *Epidermophyton* (which causes infections on skin and nails).[1–3] Dermatophytes are grouped as either anthropophilic, zoophilic, or geophilic, depending on whether their primary source is human, animal, or soil, respectively.[4,5] Because tinea corporis is common and many other annular lesions can mimic this fungal infection, physicians must familiarize themselves with its etiology and its treatment.

The purpose of this article was to provide a narrative updated review on the evaluation, diagnosis, and treatment of tinea corporis. A PubMed search was performed with Clinical Queries using the key term 'tinea corporis.' The search strategy included clinical trials, meta-analyses, randomized controlled trials, observational studies, and reviews. The search was restricted to the English language. The information retrieved from the above search was used in the compilation of the present article.

Etiology

Tinea corporis is most often caused by *Trichophyton rubrum*, *T. tonsurans*, and *Microsporum canis*.[6–12] *T. rubrum* is by far the most common cause of dermatophytosis worldwide and is the most common cause of tinea corporis in North America.[13–15] Tinea corporis secondary to tinea capitis is often caused by *T. tonsurans*.[16] On the other hand, tinea corporis resulting from close contact with dogs or cats is often caused by *M. canis*.[8,17–19] Other causative organisms include *T. interdigitale* (previously known as *T. mentagrophytes*), *T. verrucosum*, *T. violaceum*, *T. concentricum*, *Epidermophyton floccosum*, *M. audouinii*, and *M. gypseum*.[6,20–29] In recent years, *T. interdigitale* has replaced *T. rubrum* as the most common cause of tinea corporis in Southeast Asia. Rare causative organisms include *T. erinaceid*, *T. equinum*, *T. simii*, *T. schoenleinii*, *Nannizzia gypsea*, *N. nana*, and *M. gallinae* and *M. fulvum*.[30–40]

Epidemiology

Tinea corporis is the most common dermatophytosis.[41] While tinea corporis occurs worldwide, it is most commonly observed in tropical regions.[42] The lifetime risk of acquiring tinea corporis is estimated to be 10–20%.[6] Tinea corporis occurs most frequently in post-pubertal children and young adults.[18,43,44] Rare cases have been reported in the newborn period.[45] There is no sex predominance.[1] Humans may become infected through close contact with an infected individual, an infected animal (in particular, domestic dog or cat), contaminated fomites, or contaminated soil.[46–48] Infection may be acquired as a result of spread from another site of dermatophyte infection (e.g. tinea capitis, tinea pedis, onychomycosis).[49,50] Transmission among household family members is by far the most common route; children often become infected by spores shed by an infected household family member.[15,48,51] Autoinfection by dermatophytes elsewhere in the body may also occur.[52] Transmission of the fungus is facilitated by a moist, warm environment, sharing of towels and clothing, and wearing of occlusive clothing.[1,52] Predisposing factors include personal history of dermatophytosis (e.g. tinea capitis, tinea pedis, tinea cruris, and tinea unguium), concurrent affected family members, pets in the home, crowding in home, recreational exposure (e.g. wrestling and marital arts), hyperhidrosis, low β-defensin 4 levels, immunodeficiency, diabetes mellitus,

genetic predisposition (in particular, tinea imbricata), xerosis, and ichthyosis.[2,18,48,53–55]

Pathogenesis

Mannans in the cell walls of some dermatophytes, such as *T. rubrum*, have immune-inhibitory properties.[2] This allows the fungus to stay on the skin without being sloughed off prior to invasion of the skin. The causative fungus can produce proteases (enzymes that digest keratin), serine-subtilisins (enzymes that digest protein by initiating the nucleophilic attack on the peptide bond through a serine residue at the active site), and keratinases (enzymes that penetrate keratinized tissue), which allow the fungus to invade the horny layer of the skin and spread outward.[3,56] Infection is usually cutaneous and confined to the outer, non-living, cornified layers of the skin. The fungus is unable to penetrate the deeper tissues in healthy immunocompetent hosts because of host defense mechanisms, such as activation of serum inhibitory factor, polymorphonuclear leukocytes, and complements.[3] Scaling of the active border results from increased epidermal cell proliferation in response to the fungal infection.[56]

Clinical manifestations

The incubation period is 1–3 weeks.[57] Tinea corporis typically presents as a well-demarcated, sharply circumscribed, oval or circular, mildly erythematous, scaly patch or plaque with a raised leading edge (Figure 1).[44,52,57] The lesion starts off as a flat scaly spot that spreads centrifugally and clears centrally to form a characteristic annular lesion giving rise to the term 'ringworm.'[52,58,59] The central area becomes hypopigmented or brown and less scaly as the active border progresses outward.[4,58] The border is usually annular and irregular.[58] Occasionally, the border can be papular, vesicular, or pustular.[4,15] Lesions may assume other shapes such as circinate and arcuate. Mild pruritus is common.[52,60] In general, lesions caused by anthropophilic species (e.g. *T. rubrum*, *T. tonsurans*, *T. interdigitale*, *T. schoenleinii*, *T. soundanense*, *T. violaceum*, *M. audouinii*, and *E. floccosum*) are often less inflammatory/ erythematous than those caused by zoophilic species (e.g. *M. canis*, *M. nanum*, *M. ferrugineum*, *M. distortum*, *M. nanum*, *T. equinum*, and *T. verrucosum*) or geophilic species (e.g. *M. gypseum*).[44] The lesions tend to be asymmetrically distributed. When multiple lesions are present, they may coalesce into polycyclic patterns.[15] In adults, tinea corporis most commonly occurs on exposed skin. In children and adolescents, the site of predilection is the trunk.[61]

In tinea gladiatorum, the lesion presents as well-demarcated, erythematous, annular, scaling plaques on areas of skin-to-skin contact, such as the head, neck, and arms.[6] Tinea gladiatorum is most often caused by *T. tonsurans*.[62–66] The condition is most common among those who engage in contact sports such as wrestling and judo.[62–66] In a 2020 systematic review

Figure 1. An annular, erythematous, scaly plaque with a raised leading edge on the left arm characteristic of tinea corporis.

Figure 2. Tinea incognito resulting from topical corticosteroid treatment of tinea corporis on the medial aspect of the right thigh.

Figure 3. Tinea imbricata. Note the generalized, concentric, annular, lamellar, scaly plaques on the anterior trunk and upper limbs. The undulating lines were composed of overlapping scales.

and meta-analysis of 13 studies involving 4818 wrestlers, the prevalence of tinea gladiatorum varied from 2.4 to 96.62%, with an overall prevalence of 34.29% (95% confidence interval: 20.33–48.25).[67]

Tinea incognito refers to a cutaneous fungal infection that has lost its classical morphological features because of the use of calcineurin inhibitors or corticosteroids.[68,69] The clinical manifestations of tinea incognito are highly variable. Generally, compared with the lesion of tinea corporis, the lesion seen in tinea incognito is less erythematous and scaly, with a less defined border and is typically more widespread (Figure 2).[24] Pruritus is usually mild or absent.[69] Th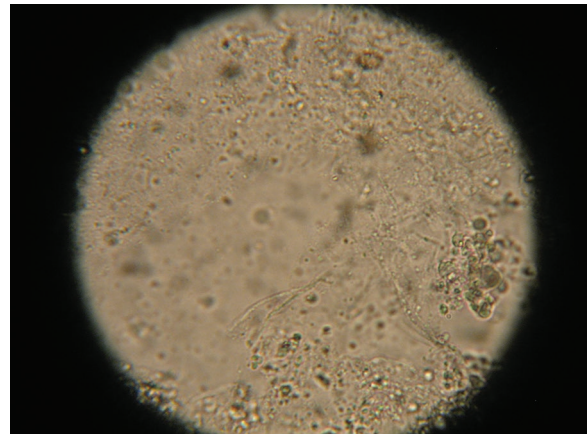e rash can be eczema-like, rosacea-like, or discoid lupus erythematosus-like, especially on the face, and eczema-like or impetigo-like on the trunk and limbs.[69]

Many clinical variants of tinea corporis exist. Tinea imbricata, caused mainly by a strictly anthropophilic dermatophyte, *T. concentricum*, typically presents as multiple, scaly, annular, concentric, erythematous rings that can extend to form polycyclic plaques (Figure 3).[53,60,70] With time, multiple overlapping lesions develop and the plaques become lamellar with abundant thick scales adhering to the interior of the plaque, giving rise to the appearance of overlapping roof tiles or lace, fish scales.[53,70] The trunk is the site of predilection. Tinea imbricata has a high tendency to generalize and large areas of the body may be affected. Pruritus is common. Tinea imbricata is endemic in Central and South America, Southwest Pacific, and Southeast Asia.[53]

Figure 4. Erythematous papules located on the glabella in a 5-year-old girl with a Majocchi granuloma.

Figure 5. Microscopic examination of skin scrapings in 10% KOH showed a large amount of branching fungal hyphae.

Majocchi granuloma, also known as nodular granulomatous perifolliculitis, results from penetration of the fungus along the hair follicle to the dermal or subcutaneous tissue, leading to a suppurative folliculitis.[71] The condition may be precipitated by occlusion of hair follicles or trauma to the skin.[24] Majocchi granuloma is most commonly seen in immunocompromised individuals or those treated with topical corticosteroids.[72] *T. rubrum* is the most common causative organism, followed by *T. interdigitale*, *T. violaceum*, and *T. tonsurans*.[72,73] Majocchi granuloma typically presents as inflammatory perifollicular papules or pustules, mainly on the face or limbs (Figure 4).[24,73,74] Nodular lesion and subcutaneous abscess are more commonly seen in immunocompromised individuals.[24] The trichophytin skin test is usually positive.[74]

Bullous tinea corporis, a rare clinical variant of tinea corporis, is characterized by vesicles or bullae, usually limited to the borders of an erythematous scaly plaque.[75–77] Rupture of the vesicles or bullae may leave behind erosions and crusts over an erythematous background.

In immunocompromised individuals, tinea corporis may present as a disseminated skin infection or subcutaneous/deep abscess.[78] Rarely, tinea corporis may present as purpuric macules, known as tinea corporis purpurica.[79]

Diagnosis

The diagnosis of tinea corporis is most often clinical, especially if the lesion is typical.[6] A well-demarcated, sharply circumscribed, erythematous, annular, scaly plaque with a raised leading edge, and scaling and central clearing on the body is characteristic. At times, the diagnosis can be difficult due to the prior use of medications, such as calcineurin inhibitors or corticosteroids. Dermoscopy is a useful and non-invasive diagnostic tool. Dermoscopic findings in cases of tinea corporis include diffuse erythema, dotted vessels with peripheral to patchy distribution, white scales with peripheral distribution, 'moth-eaten' scale, peeling in an outward direction, brown spots surrounded by a white-yellow halo, follicular micropustules, wavy hair, and broken hair.[80,81] These changes may be seen despite the use of topical corticosteroids or calcineurin inhibitors.[82] Reflectance confocal microscopy is another useful diagnostic tool.[2,83] With reflectance confocal microscopy, branching fungal hyphae can be detected over an erythematous annular scaly patch in individuals with tinea corporis.[2,83] Wood lamp examination of the affected area is not useful as the lesion of tinea corporis usually does not fluoresce with a Wood lamp.

If necessary, the diagnosis can be confirmed by microscopic examination of potassium hydroxide (KOH) wet-mount preparations of skin scrapings from the active border of the lesion.[46] The skin scrapings should be transported in a presterilized black chart paper so as to keep the specimen dry and prevent overgrowth of bacteria that may be contaminants.[2] To perform the test, a drop of 10–20% KOH is added to the scrapings on a microscopic slide.[1] The specimen is gently heated to accelerate the destruction of the squamous cells.[1,59] The KOH dissolves the epithelial tissue, leaving behind easily visualized septate hyphae with or without arthroconidiospores (Figure 5).[1,59] The addition of dimethyl sulfoxide to KOH may permit more rapid examination without heating.

Fungal culture is the gold standard to diagnose dermatophytosis, especially if the diagnosis is in doubt and results of other tests are inconclusive, or the infection is widespread, severe, or unresponsive to treatment.[46,56] Fungal culture can help to differentiate fungal species.[84] However, fungal culture is expensive and it usually takes 7–14 days for results.[2,15] For certain species, it may take up to 4 weeks for results.[16] The most common culture medium is Sabouraud peptone–glucose agar (4% peptone, 1% glucose).[59] However,

Sabouraud peptone–glucose agar does not contain antibiotics and thus may allow overgrowth of bacterial contaminants. On the other hand, mycosel agar and dermatophyte test medium both contain antibiotics. The antibiotics help to suppress the growth of bacterial species, which may contaminate the culture. If the results of the investigations are inconclusive, a polymerase chain reaction (PCR) assay for fungal DNA or a PCR-restriction fragment length polymorphism method based on a ribosomal DNA internal transcribed spacer may be considered for fungal identification in academic settings for research purposes.[85–89]

Differential diagnosis

Diseases that present with annular lesions may mimic tinea corporis. The differential diagnosis is broad and includes pityriasis rosea (non-itchy herald patch, generalized, bilateral, symmetrical eruption 4–14 days later, characteristic 'Christmas tree' appearance on the back and a V-shaped pattern on the upper chest); tinea versicolor (multiple, well-demarcated, finely scaly, brownish macules/patches in fair-skinned individuals and hypopigmented macules/patches in dark-skinned individuals, minimal or absent erythema, absent collarette of scales in individual lesions, typically asymptomatic); nummular eczema (well-demarcated, pruritic, coin-shaped, symmetrical, eczematous, scaly lesions, involvement of the extremities rather than the trunk, serous exudate in acute lesions, no central clearing, rapid response to topical steroids); plaque psoriasis (well-demarcated, sharply circumscribed, annular, erythematous, round or oval, pruritic plaques with loosely adherent silvery-white micaceous scales, positive Auspitz sign, Koebner phenomenon, nail pitting, arthritis, uveitis, geographic tongue, positive family history); atopic dermatitis (flexural involvement in older children and adolescents, highly pruritic, excoriations, lichenification in chronic lesions, chronically relapsing); contact dermatitis (well-demarcated, erythematous lesion localized to the area of contact, immediate skin reaction with burning, stinging, or discomfort if caused by an irritant, delayed response associated with pruritus caused by an allergen); seborrheic dermatitis (salmon-colored or erythematous, sharply demarcated patches with yellow-white, greasy scales); localized granuloma annulare (asymptomatic, firm, erythematous, violaceous, flesh-colored or brown, non-scaly plaques with central involution, annular configuration, usually involve the extensor surfaces of distal extremities); fixed drug eruption (history of medication use, well-demarcated, round-to-oval, erythematous or violaceous macules/plaques, absent systemic symptoms, sites of predilection include hands, feet, lips and perianal area, usually subsides within 14 days after the offending medication has been discontinued, recurs in the same location with repeat exposure to the medication); subacute cutaneous lupus erythematosus (annular, erythematous, scaly plaques often in sun-exposed areas); discoid lupus erythematosus (well-demarcated, erythematous, hyperkeratotic, indurated, coin-shaped plaques covered by partially adherent, scales in sun-exposed areas); urticaria (pruritic, erythematous, and edematous wheals of the superficial layers of the skin, individual lesions wax and wane rapidly); urticaria pigmentosa (pruritic, yellow-tan to reddish-brown macules/papules on the trunk and proximal extremities, positive Darier sign); pityriasis lichenoides chronica (polymorphic pink to reddish brown papular rash with overlying mica-like scales, chronic relapsing course without herald patch, residual hypo- or hyperpigmentation); lichen planus (characterized by 6 Ps: *p*lanar (flat-topped), *p*urple (violaceous), *p*olygonal, *p*ruritic, *p*apules/*p*laques, lesions may be covered with white, lacy, reticular lines [Wickham striae]); erythema migrans (flat, erythematous, rapidly expanding [days], asymptomatic, annular lesion at the site of a tick bite, central clearing as the lesion expands, 'bull's eye' appearance); erythema multiforme (acrally distributed, distinct targetoid lesions with central erythema); erythema dyschromicum perstans (slowly progressive, symmetrical, ashy gray-colored macules/patches, truncal distribution, slightly raised, erythematous border in the early stage); erythema marginatum (migratory, rapid expanding [hours], evanescent, non-pruritic, arciform/polycyclic/annular, erythematous plaque, lesion extends centrifugally with central clearing, border is irregular, serpiginous, and sharp on the outer edge but diffuse on the inner edge, a major manifestation of acute rheumatic fever); superficial erythema annulare centrifugum (annular or arcuate, erythematous patch/plaque that enlarge centrifugally with central clearing, 'trailing scale' along the inner portion of the advancing edge, associated with drugs, systemic infection, malignancies, and autoimmune disease); impetigo contagiosum (characteristic yellowish-brown or honey-colored 'stuck-on' crust over the superficial erosion, satellite lesions in the vicinity, most common on the face); erythema gyratum repens (paraneoplastic eruption, erythematous concentric rings with trailing scale at their edges, characteristic 'wood grain' appearance); and secondary syphilis (asymptomatic, diffuse, symmetrical, round-to-oval, pink-to-reddish-brown monomorphous macules or patches on the trunk and extremities including the palms and soles, absence of herald patch, 'moth-eaten' alopecia, lymphadenopathy, history of venereal exposure, and/or chancre).[15,49,52,54,56,60,90–96]

Complications

Tinea corporis is contagious and therefore may have significant psychological, social, and occupational health effects.[84] Secondary bacterial superinfection may occur as a result of scratching and abrasion of the skin. Post-inflammatory hypopigmentation and hyperpigmentation may occur.[59] Dermatophytid (id) reaction, also known as id reaction, auto-eczematization, or disseminated eczema is a secondary dermatitic eruption that may occur in association with a fungal infection especially just after commencement of systemic antifungal treatment.[5] Affected patients often develop widespread, intensely pruritic, erythematous, scaly papules, maculopapules, papulovesicles, or pustules. Presumably,

the dermatitic eruption is an immunologic reaction to the fungal antigen like a delayed-type (type IV) hypersensitivity response.[24] Rarely, psoriatic flares precipitated by tinea corporis have been described.[97]

Treatment

Non-pharmacologic measures

As fungi thrive best in moist and warm environments, patients should be advised to wear light and loose-fitting clothing.[59] The skin should be kept clean and dry.

Pharmacotherapy

The standard treatment of tinea corporis is with topical antifungals and there is evidence of the superiority of topical antifungals over the use of placebo.[84,98] Localized or superficial tinea corporis usually responds to topical antifungal therapy applied to the lesion and at least 2 cm beyond the lesion once or twice daily for 2–4 weeks.[24] Commonly used topical antifungal agents include azoles (e.g. econazole, ketoconazole, miconazole, clotrimazole, miconazole, oxiconazole, sulconazole, sertaconazole, eberconazole, and luliconazole), allylamines (e.g. naftifine, terbinafine), benzylamine (butenafine), ciclopirox, and tolnaftate.[18,24,99–110] In this regard, nystatin, which is an effective treatment for Candida infections, is not effective for tinea corporis.[24] In a 2013 meta-analysis of 65 trials (trials with a common comparator and head-to-head trials) involving 14 topical antifungals, there was no significant difference among the antifungals regarding the outcome of mycologic cure at the end of the treatment.[111] Pairwise comparison of topical antifungals showed that butenafine, naftifine, and terbinafine were significantly more efficacious in sustaining the cured outcome.[111] A 2014 Cochrane review suggests that individual treatments with terbinafine and naftifine are effective and have few mild adverse events.[112] Topical antifungal agents are generally well tolerated. Side effects are uncommon, except for rare instances of contact dermatitis. Common causes of treatment failure include poor compliance, drug resistance, reinfection from close contact and auto-inoculation, and misdiagnosis.[113] Some authors suggest the addition of a topical corticosteroid to the topical antifungal agent, especially in individuals with inflammatory dermatomycosis.[114,115]

Systemic antifungal treatment is indicated if the lesion is extensive, deep (e.g. Majocchi granuloma), recurrent, chronic, or unresponsive to topical antifungal treatment; if the patient is immunodeficient; or if there are multiple site lesions.[24,52,84] Randomized control trials support the efficacy of systemic treatment with oral antifungal agents.[116,117] Oral antifungal agents used for the treatment of tinea corporis include itraconazole (children: 3–5 mg/kg/day [maximum 200 mg/day]; adults: 200 mg/day), fluconazole (children: 6 mg/kg once weekly [maximum: 200 mg once weekly]; adults: 200 mg once weekly), terbinafine granules (children: <25 kg, 125 mg/day; 25–35 kg, 187.5 mg/day; >35 kg, 250 mg/day), and terbinafine tablets (children: 10–20 kg, 62.5 mg/day; 21–40 kg, 125 mg/day; >40 mg, 250 mg/day; adults: 250 mg/day).[2,24,56,118] The duration of treatment varies, depending on the response. The usual duration of treatment is 2–4 weeks but may take longer for recalcitrant cases.[84] Oral ketoconazole should be avoided because of the risk of hepatotoxicity, adrenal insufficiency, and drug interactions.[24] Oral griseofulvin (not available in many countries, including Canada) is less effective, has more adverse events, and requires longer duration of therapy.[5,50] As such, oral griseofulvin is not the medication of choice in the treatment of tinea corporis. Combined therapy with oral and topical antifungal agents may increase the cure rate.[119]

In recent years, the incidence of tinea corporis refractory to terbinafine treatment has been on the rise.[116,120–124] Terbinafine acts by inhibiting the enzyme squalene epoxidase, which is responsible for synthesis of ergosterol – an essential component of fungal cell wall.[122] Resistance to terbinafine has largely been attributed to point mutations in the squalene epoxidase target gene (*SQLE*).[123] Identification of the point mutation can be achieved by DNA sequencing of the *SQLE* gene of the fungal isolate. Subtherapeutic dosage, non-compliance to treatment, and abuse of over-the-counter topical preparations that combine antifungals with corticosteroids may also be contributory.[116]

Prevention

Close contact or sharing of fomites and clothing with an infected individual should be avoided.

Prognosis

The prognosis for localized tinea corporis is excellent with appropriate treatment and patient compliance. Recurrence may occur if therapy is stopped too soon without complete eradication of the fungi. Reinfection may occur if a reservoir (tinea pedis, tinea capitis, onychomycosis) of infection is present.[5,50,125]

Conclusion

A well-demarcated, sharply circumscribed, mildly erythematous, annular, scaly plaque with a raised leading edge, and scaling and central clearing on the body is characteristic of tinea corporis. At times, the diagnosis can be difficult due to the prior use of medications, such as calcineurin inhibitors or corticosteroids. Furthermore, diseases that present with annular lesions may mimic tinea corporis. Tinea corporis is a common fungal infection and the differential diagnosis is broad and, at times, difficult. Physicians must be familiar with this condition so that an accurate diagnosis can be made and appropriate treatment initiated.

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