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Skin Infestations: Fungal and Scabies

Most of us will hardly flinch if we hear "Cardiac arrest coming in," but will recoil in terror at "How are you at rashes?" I am not a dermatologist — and don't want to be. But there is hardly a day that goes by that I am not asked by someone (a patient, a colleague, a friend, or a family member) to look at a rash or lesion on the skin. While some patients present for their rash primarily, many will "add it on" to the visit. In many emergency departments (EDs), a dermatology, or even primary care, follow-up visit is not an option. Therefore, emergency providers need a working understanding of common rashes, and also should recognize those rashes that need referral.

This article provides an in-depth look at common rashes caused by fungi and scabies. We have included some information about potassium hydroxide (KOH) staining, which long has been the standard for making the diagnosis of tinea. I recognize that many no longer have a microscope available in the ED, and many ED providers may not even know how to use one. That information is included for the few of you who can use it.

Although cutaneous fungal infections rarely are life-threatening, they are common, and they are irritating. This review should help you diagnose and treat these infections.

— Sandra M. Schneider, MD, Editor

Introduction

Tinea refers to a superficial fungal infection of the skin, hair, and nails caused by dermatophytes. Dermatophytes are filamentous fungi; the three genera that cause disease are *Microsporum*, *Trichophyton*, and *Epidermophyton*.[1] *Trichophyton rubrum* is the most common cause of dermatophyte infection, accounting for nearly 70% of infections worldwide.[1] These infections are extremely common, but are more prominent in warm, tropical climates. Tinea is seen more commonly in black and Asian patients, has a predilection for young adults, and is three to four times more common in males.[1] Dermatophyte infections are alike in their physiology, morphology, and pathogenicity and are referred to clinically as "tinea."

Transmission may be anthropophilic (human to human), zoophilic (animal to human), or geophilic (soil to human/animal).[1] Unlike other fungi, dermatophytes metabolize keratin as an energy source. They are not particularly virulent and typically invade only the cornified, outer layers of the epidermis. Mannans in the dermatophyte cell wall contribute to invasion of the skin by decreasing epidermal proliferation and by exhibiting immune-inhibitory effects.[1] Defects in the skin barrier (diseases such as Darier disease, Hailey-Hailey disease, and ichthyosis) or maceration tend to encourage dermatophyte invasion.[1] Protective host factors that limit invasion to the keratinized tissue include protease inhibitors, sebum, serum factors, and the host immune

EXECUTIVE SUMMARY

- Tinea is a common skin disorder seen in primary care practices, but it often is misdiagnosed and can be mistaken for mimickers.
- Unlike other fungi, dermatophytes metabolize keratin as an energy source and typically are not particularly virulent.
- Tinea is more common in black and Asian patients, has a predilection for young adults, and is three to four times more common in males.
- The clinical dermatophyte infections tend to be named for the involved body area.
- First-line treatment involves topical agents such as azoles, allylamines, ciclopirox, butenafine, and tolnaftate, but the treatment of choice for tinea capitis and tinea barbae is griseofulvin.
- Scabies is caused by the mite *Sarcoptes scabiei* and must be considered in differential diagnosis, but the distribution and appearance of lesions typically are distinctive.
- Scabies treatment involves two topical applications of permethrin 5% cream given seven days apart.

system.[1] Immunodeficient hosts (chronic mucocutaneous candidiasis, common variable immunodeficiency, and HIV) tend to have more severe, chronic, or recurrent infections than immunocompetent hosts.

Dermatophyte infections commonly are misdiagnosed in clinical practice. The clinical appearance of these infections can vary, and many other dermatologic diseases may have a similar appearance. This review will help you diagnose and treat your patients and provide the most current treatment regimens.

Subtypes of Tinea Infections

Clinical dermatophyte infections tend to be named for the involved body area. The clinical subtypes include tinea corporis (body), tinea capitis (scalp), tinea cruris (groin), tinea pedis (feet), tinea manuum (hands), tinea unguium (nails), tinea barbae (beard), and tinea faciei (face). See Figure 1 for a clinical presentation of tinea manuum. See Table 1 for the clinical features of tinea and its mimickers.

Figure 1. Tinea Manuum

Small collarettes of scale are typical of superficial fungal infections located on the palms and soles.

Image courtesy of Jessica Perkins, DO.

Tinea Corporis

Tinea corporis is an infection of the body surfaces other than palms/soles, groin, face, scalp, hair, and nails. It usually occurs on exposed skin of the trunk and extremities and generally is confined to the epidermis. It is usually acquired by direct contact or secondarily spread from another infected body area.[1] The most common pathogen is *T. rubrum*, followed by *T. mentagrophytes*.[1] Infection with *T. tonsurans* may occur in adults from contact with a child with tinea capitis.[2] Direct contact with an infected cat or dog may lead to infection with *Microsporum canis*.[2] This infection is found globally; however, it is more common in tropical regions. The classic presentation is referred to as "ringworm" and is characterized by a pruritic, erythematous, scaly patch with a raised border and central clearing.[3]

Clinical Variants of Tinea Corporis

Majocchi's granuloma is a rare, deep infection of the hair follicle that invades the dermis or subcutaneous tissue.[2] Inciting events include shaving legs, trauma to skin, or immunosuppression. It is characterized by perifollicular papulopustules or granulomatous nodules.[1] Tinea profunda results from excessive inflammation in response to a dermatophyte infection. It may have a granulomatous or verrucous appearance and can be mistaken for cutaneous tuberculosis or squamous cell carcinoma.[1]

Tinea imbricata (Tokelau ringworm) is a chronic infection caused by *T. concentricum* and is characterized by concentric annular, scaly, erythematous

Table 1. Typical Clinical Features of Tinea and Mimickers	
Tinea Corporis	Annular plaques with a central clearing and a leading edge of scale
Nummular Eczema	Nummular or "coin-shaped" erythematous, scaly plaques in typical distributions (antecubital and popliteal fossa, posterior neck, lower extremities), atopic diathesis
Erythema Annulare Centrifugum	Annular erythema with a trailing scale; may be a response to a tinea infection; perform full skin exam
Scabies	Erythematous papules to pustules with excoriations and possible burrows, flexor wrists, interdigital webspaces, umbilicus, genitals
Lichen Planus	Purple, polygonal, flat-topped papules ± lacy reticulated scale; flexor wrists, ankles, trunk ± oral lesions
Psoriasis Vulgaris	Elevated, well-demarcated, erythematous plaques with overlying silvery scale
Seborrheic Dermatitis	Waxy, yellow scale on an erythematous base; scalp, central face, eyebrows, beard, central chest
Pustular Psoriasis	Deep, yellow papules ± brown collarettes of scale on the plantar/palmar surfaces
Subacute Cutaneous Lupus Erythematosus	Annular, erythematous plaques with central clearing in a photodistribution (V-neck and upper back ± upper arms)
Pityriasis Rosea	Single patch (herald patch) followed by eruption of flesh to erythematous colored scaly patches in a "Christmas tree" distribution on the trunk
Source: Author created.	

plaques.[1] It occurs in the equatorial regions of the world.

Mimickers of Tinea Corporis

The following conditions can mimic tinea corporis: dermatitis (including nummular eczema, stasis, atopic, contact, and seborrheic dermatitis), pityriasis versicolor, pityriasis rosea, parapsoriasis, erythema annulare centrifugum, elastosis perforans serpiginosa, annular psoriasis, granuloma annulare, subacute lupus erythematosus, mycosis fungoides, and impetigo.[1]

Tinea Capitis

Tinea capitis is a dermatophyte infection of the scalp hair that primarily affects children. The peak age is 3 to 7 years, and it is more common in boys.[3] *T. tonsurans* accounts for more than 90% of tinea capitis in the United States, with *M. canis* following as the second most frequent cause.[1] Other etiologies include *T. violaceum* (endemic in Africa) and *M. audouinii* (Europe).[1] Tinea capitis can have a range of clinical presentations that depend on the causative organism as well as the host's immune response. Alopecia with or without scale is the most common presentation; however, it can vary from mild scaling to a severe pustular eruption with alopecia called a kerion.[1]

The three patterns of invasion for dermatophytes that infect the hair include endothrix, ectothrix, and favus. Endothrix patterns result from anthropophilic *Trichophyton* infection within the hair shaft, and common causes include *T. tonsurans* and *T. violaceum*.[1] Ectothrix patterns occur when the infection is outside the hair shaft and results in destruction of the cuticle. The presentation varies from non-inflamed, scaly, patchy alopecia to kerion formation. Microsporum infections may fluoresce under Wood's lamp. The favus pattern is caused by *T. schoenleinii* and is the most severe. It presents as thick, yellow crusts and has a bluish fluorescence under a Wood's lamp.[1] Scarring alopecia may develop if the infection is chronic. It is important to note that many scalp/hair conditions can cause scaling or alopecia, and it is very important to consider and rule out a fungal infection.

Mimickers of Tinea Capitis

Seborrheic dermatitis, alopecia areata, psoriasis, and trichotillomania can mimic tinea capitis. If pustules are present, pyoderma gangrenosum and folliculitis can resemble tinea capitis.[1] If scarring is present, lichen planus, discoid lupus erythematosus, and central centrifugal cicatricial alopecia should be part of the differential diagnosis.[1]

Tinea Cruris

Commonly known as jock itch, tinea cruris is a dermatophyte infection of the inguinal area. Usually it is seen on the inner aspect of the upper thighs and crural folds, but may be seen in the gluteal cleft and buttocks as well. The three most common pathogens are *T. rubrum*, *E. floccosum*, and *T. mentagrophytes*.[1] It is more common in men because the scrotum provides an ideal environment and because more often men have concomitant tinea pedis, which can be transferred to the groin by pulling on undergarments over the feet.[1] Characteristically, lesions are sharply demarcated with a raised, erythematous, scaly advancing border. Lesions may have vesicles, and may be

Figure 2. Tinea Pedis

Shows the classic appearance of tinea pedis with small collarettes of scale extending past the mid-lateral foot with a minor erythema noted on the base. Tinea unguium also is noted, making this a straightforward clinical case.

Image courtesy of Jessica Perkins, DO.

unilateral or bilateral. In males, the scrotum usually is spared. Providers should consider cutaneous candidiasis if the scrotum is involved.[1]

Mimickers of Tinea Cruris

The following conditions can mimic tinea cruris: inverse psoriasis, erythrasma, seborrheic dermatitis, candidal intertrigo, contact dermatitis, lichen simplex chronicus, parapsoriasis, Hailey-Hailey disease, and Langerhans cell histiocytosis.[1]

Tinea Pedis

Also known as athlete's foot, tinea pedis is the most common location for dermatophyte infection. Infection typically occurs on the soles of the feet and, most commonly, in the interdigital area. The major types of tinea pedis are interdigital, hyperkeratotic (moccasin-type), vesiculobullous (inflammatory), and ulcerative. The characteristic finding in the interdigital type is pruritic, erythematous scales and/or erosions that usually present in between the third and fourth toes. Moccasin-type tinea pedis presents as a diffuse, erythematous, hyperkeratotic lesion that covers the soles plus the medial and lateral edges of the feet. (*See Figure 2.*) The vesiculobullous type presents as an erythematous vesicular or bullous eruption that may be pruritic or painful, and it is most common on the medial aspect of the foot. Similar to tinea cruris, the most common pathogens responsible are *T. rubrum*, *E. floccosum*, and *T. mentagrophytes*.[1] Patients also may have a secondary bacterial infection if they present with malodorous erosions or ulcerations.

Tinea manuum has a characteristic presentation of unilateral hyperkeratotic lesions on the palms and interdigital spaces.[1,4] The most common pathogens are the same as for tinea pedis and tinea cruris, and these infections often occur concomitantly.

Mimickers of Tinea Pedis

Mimickers of tinea pedis include dermatitis (dishydrotic and contact), psoriasis vulgaris or pustular psoriasis, secondary syphilis, pitted keratolysis, hereditary keratosis palmoplantaris, and juvenile plantar dermatosis.[1,3] Erythrasma or bacterial infection may resemble the interdigital type of tinea pedis.[1]

Tinea Unguium

Tinea unguium is a dermatophyte infection of the nail unit, most commonly caused by *T. rubrum*, *T. mentagrophytes*, and *E. floccosum*.[1] Often, this infection is referred to as onychomycosis, an umbrella term that encompasses all fungal infections of the nails including non-dermatophyte causes. However, dermatophytes account for approximately 90% of onychomycosis cases. Tinea unguium is more common in men, is often associated with chronic tinea pedis, and occurs on the toenails more than the fingernails. It may be unilateral or bilateral and may affect only one nail or multiple nails. There are three common patterns that are based on the point of entry for the infection. The distal/lateral subungual type is the most common and presents with onycholysis, yellowing, and thickening of the nails.[1] (*See Figure 3.*) The superficial white type is confined to the dorsal surface of the nail, and presents as white patches (*T. mentagrophytes*) or transverse striate bands (*T. rubrum*).[1] The proximal subungual type invades underneath the proximal nail fold and usually is found in immunocompromised patients.[1]

Figure 3. Tinea Unguium

Distal yellow debris on the middle fingernail can serve as a clue to tinea unguium. It is prudent to check the feet in any nail infection, as tinea manuum and pedis are commonly present.

Image courtesy of Jessica Perkins, DO.

Mimickers of Tinea Unguium

Numerous pathogens (dermatophyte and non-dermatophyte) cause onychomycosis, and many factors may cause dystrophy of the nails, making accurate diagnosis a challenge. Diagnoses that can mimic tinea unguium are *Candida* infections, the nail manifestations of psoriasis, lichen planus, dermatitis, hyperthyroidism, external trauma, pachyonychia, and Darier disease.[1] Tinea unguium can be difficult to treat because of the required prolonged treatment times, medication side effects, and recurrences.

Tinea Faciei

A dermatophyte infection on the skin of the face, tinea faciei usually is caused by the same organisms as tinea corporis.[4] Infections with *T. rubrum* can be especially difficult to diagnose, as the margins of the lesion are often indistinguishable. Tinea faciei is seen more frequently in AIDS patients.

Mimickers of Tinea Faciei

Seborrheic, perioral, or contact dermatitis can mimic tinea faciei. Other mimickers include rosacea, lupus erythematosus, acne, and annular psoriasis in children.[1]

Tinea Barbae

Tinea barbae occurs exclusively in the beard hair distribution of the face and neck of men. It is commonly transmitted by animals, and the typical causative pathogens are *T. mentagrophytes* and *T. verrucosum*.[1] The clinical presentation may be severe in the zoophilic pathogens, and often it presents with intense inflammation, pustules, and abscesses with bacterial superinfection.[4] *T. rubrum* causes a mild, superficial variant similar to tinea corporis.

Mimickers of Tinea Barbae

Bacterial folliculitis, herpes simplex/zoster, acne vulgaris, and cervicofacial actinomycosis can mimic tinea barbae.[1]

Diagnosis

The most important factor in the diagnosis of a dermatophyte infection is a thorough physical exam. It is important to remember that there may be more than one area of the body infected at the same time, which warrants a complete cutaneous exam. For example, tinea pedis often occurs with tinea unguium or tinea cruris.[2]

Clinical findings suspicious for a dermatophyte infection should be followed by confirmation testing. Diagnosis usually is confirmed with a potassium hydroxide (KOH) examination via microscopy. Most EDs have removed the microscopes and reagents. For those who still have access to these useful tests, a KOH prep can be very helpful. KOH examinations may be enhanced with chlorazol black E stain. The disadvantage of KOH testing is that it often yields false-negative results.[5] A skin scraping for a KOH examination should be taken from the active edge of the lesion with a No. 15 blade.[6] The provider should apply alcohol to the lesion before scraping to enhance the scale adhering to the blade.

All dermatophytes appear the same in KOH examinations. If there is a need to identify a particular species, a culture is required. Cultures are slow growing and need many weeks of incubation time.[7] If KOH examination and fungal cultures are negative and a dermatophyte infection is still suspected, a skin biopsy may be performed. Although, in general, fungal cultures and skin biopsies are well outside of the emergency physician's normal practice, many insurance companies require a positive culture before they will cover the treatment. As the cost of some of the drugs may be in the hundreds of dollars, patients may prefer referral to a dermatologist if an expensive or long-term treatment is anticipated (onychomycosis). Skin biopsies will show fungal hyphae within the stratum corneum. Periodic acid-Schiff (PAS) stains and silver stains often are used to enhance fungal elements in biopsy specimens. Techniques using polymerase chain reaction (PCR) and mass spectroscopy also can be used to identify dermatophyte strains.[8] PCR is a fast, simple procedure that is highly specific for diagnosis of dermatophyte infections.[9] There are also promising studies for immunochromatographic diagnosis of dermatophytes.[10] The most reliable way to diagnose onychomycosis is by histologic examination of a formalin-fixed, PAS-stained nail plate.[1] The practitioner places a nail clipping into formalin and sends it to the local pathology lab for review.

Treatment

First-line treatment for uncomplicated, superficial, and localized tinea infections (corporis, cruris, and pedis) is topical antifungals. Topical agents include azoles (clotrimazole, econazole, ketoconazole, efinaconazole, luliconazole, miconazole, oxiconazole, sertaconazole, sulconazole), allylamines (naftifine, terbinafine), ciclopirox, butenafine, and tolnaftate.[2] Several of these topical preparations are available over the counter and often are adequate treatment for mild infections. Azoles tend to be more cost effective, although they are not quite as effective as allylamines.[6] Topical antifungals should be applied once or twice a day for two to four weeks and should be continued for one week after clinical clearance.[6] It is important to note that although topical nystatin is effective in treating *Candida*, it is not effective in dermatophyte infections.[2] Photodynamic therapy is also a treatment option if other methods have failed.[6]

Infections including tinea manuum, tinea capitis, tinea unguium, and Majocchi's granuloma; infections covering extensive areas; and infections

that fail topical treatment usually require systemic antifungal medication. Oral agents include terbinafine, fluconazole, itraconazole, and griseofulvin. Prescribing guidelines for extensive infections, failure of topical treatment, and relapsing infections suggest terbinafine 250 mg daily for 14 days, fluconazole 50 mg daily for two to four weeks (six weeks for tinea pedis) or 150-200 mg once weekly for two to four weeks, itraconazole 100 mg daily for 15 days or 200 mg daily for seven days (may require longer treatment for tinea pedis and manuum), or griseofulvin microsize 500-1,000 mg daily for two to four weeks.[2,6]

The treatment of choice for tinea capitis and tinea barbae is griseofulvin. Terbinafine, itraconazole, and fluconazole are not FDA-approved for the treatment of tinea capitis or barbae.[1] Adjunct treatment for tinea capitis includes antifungal shampoo (ketoconazole 2% or selenium sulfide 2.5% every other day), disinfecting grooming devices, and treatment of close contacts.[1] In addition to antifungal treatment, products containing urea, glycolic acid, and lactic acid can be used in treating tinea pedis or tinea manuum.[1,11] Tinea unguium can be extremely difficult to treat and often needs long-term systemic treatment.

Tinea incognito is a condition that occurs if a dermatophyte infection is misdiagnosed and treated with a topical corticosteroid. It can alter the clinical appearance of the infection and make diagnosis difficult. Treatment with corticosteroids also may lead to exacerbation of the infection and cause Majocchi's granuloma.[2] It is not recommended to use topical corticosteroids in conjunction with antifungals even though it may lead to more rapid resolution of inflammation and disease.[2]

Figure 4. Lichen Planus

Polygonal, purple, flat-topped papules on the flexor wrist is a classic presentation of lichen planus.

Image courtesy of George Gibbons, MD.

Scabies

The scabies mite, *Sarcoptes scabiei var. hominis*, causes human scabies.[12] The entire life cycle of the mite takes place in the epidermis, with the female mite laying three eggs daily. These eggs mature over the course of 10 days. The mites typically live on the human host for less than three days, except in the case of crusted scabies, in which they may survive up to seven days.[12] Crusted scabies is a type of scabies found in individuals with compromised immune systems allowing the mite to survive with minimal symptoms and a large number of mites.[12]

The cutaneous lesions of scabies have a typical distribution. They often involve the web spaces of the fingers, flexural wrists, posterior neck, and umbilicus. Men often have lesions on the penis and scrotum, while women tend to have vulvar and areolar lesions. The burrow is the classic and distinct clinical finding in scabies and represents the track of the female mite to lay her eggs. Clinically, the burrow presents as a small white to grey winding plaque.[12] The burrow may not always be present.[12] Other cutaneous findings can include erythematous papules, pustules, or vesicles with excoriations often prominent. The diagnosis can be confirmed via skin scraping, curetting, or microscopic examination of transparent tape from an infested area. A skin biopsy can confirm the diagnosis.[12]

Symptoms of scabies can present two to six weeks after first exposure or within one to two days on second exposure. The primary symptoms are pruritus and skin lesions. Scabies commonly is known as one of the most pruritic rashes. The pruritus can be exacerbated by a hot shower or can cause insomnia, as it worsens at night. Household contacts often are plagued by the pruritus as well.

Standard treatment of scabies can be completed with two topical treatments of permethrin 5% cream given seven days apart (day 1 and day 8 applications).[12] For adults, the cream is applied from the neck down on all skin surfaces at night and washed off in the morning. For elderly patients and infants (2 months of age and older), the cream also should be applied to the face and scalp. Patients with a sensitivity or allergy to formaldehyde or chrysanthemums should avoid using permethrin cream. Other topical medications that may be considered in scabies if resistant or allergic to permethrins include lindane 1% lotion/cream, sulfur ointment (5-10%),

and crotamiton 10% lotion/cream. Ivermectin (200 to 400 mcg/kg) orally is also an excellent choice for resistant scabies, treating on day 1 and day 8 or 14.[12] Ivermectin may cause central nervous system (CNS) toxicity in infants and young children. Lindane also has potential CNS toxicity in patients weighing less than 50 kg.[12] Therefore, these medications are used only when absolutely needed.

Mimickers of Scabies

Lesions of lichen planus (*see Figure 4*) have a typical presentation on the flexor wrists, but are flat-topped, purple papules, often with a white reticulated veil, and will lack the characteristic burrow of scabies. Other mimickers could include viral exanthems, guttate psoriasis, secondary syphilis, drug reactions, or other arthropod assaults.

Conclusion

Dermatophyte and scabetic infestations are extremely common worldwide and often are misdiagnosed. Patients without access to primary care may seek treatment for these conditions in the emergency department. More often this may be a secondary complaint. Numerous other dermatologic skin conditions may mimic these infections. A thorough cutaneous examination often can reveal clues to the diagnosis. Other lab modalities, such as skin scraping, biopsy, or culture, can further aid in making the correct diagnosis. Patients should be cautioned to seek further care if the rash does not respond to the treatment prescribed. If therapies discussed above fail to treat the skin rash, it is always prudent for physicians to refer to dermatology for further evaluation and treatment.

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CME/CE Questions

1. Shaving one's legs while infected with tinea corporis could lead to which inflammatory dermatophyte infection of the hair follicles?
   a. Majocchi's granuloma
   b. Tinea imbricata
   c. Tinea profunda
   d. Tinea concentricum
2. Dermatophyte infections can be treated with all but which of the following?
   a. Ciclopirox
   b. Fluconazole
   c. Terbinafine
   d. Nystatin
3. What is the clinical entity resulting from a dermatophyte infection treated with topical steroids called?
   a. Tinea concentricum
   b. Tinea incognito
   c. Tinea imbricata
   d. Bullous tinea
4. What is the most common cause of dermatophyte infection worldwide?
   a. *Microsporum canis*
   b. *Candida glabrata*
   c. *Trichophyton tonsurans*
   d. *Trichophyton rubrum*
5. Permethrin cream may cross-react with which of the following?
   a. Lindane
   b. Sulfa products
   c. Poison ivy
   d. Chrysanthemums
   e. Roses
6. Which of the following is a common complaint of scabies?
   a. Pruritus
   b. Lesions on the flexor wrists and webspaces
   c. Worsening after hot shower
   d. Lesions or pruritus in household contacts
   e. All of the above
7. Permethrin cream should be applied at night to the neck down on all body surfaces on:
   a. days 1 and 3.
   b. day 1.
   c. day 1 and day 8.
   d. day 1 and day 5.
8. A 70-year-old patient presents with intense itching and raised scaled lesions over most of his body. This rash has been present for more than three months. He has been in the emergency department multiple times and has been treated with topical and systemic steroids without improvement. Now, several of the lesions appear to be infected. Which of the following is the likely cause?
   a. Tinea corporis
   b. Tinea versicolor
   c. Tinea pedis
   d. Scabies

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EMERGENCY MEDICINE REPORTS

Skin Infestations: Fungal and Scabies

Table 1. Typical Clinical Features of Tinea and Mimickers

Tinea Corporis	Annular plaques with a central clearing and a leading edge of scale
Nummular Eczema	Nummular or "coin-shaped" erythematous, scaly plaques in typical distributions (antecubital and popliteal fossa, posterior neck, lower extremities), atopic diathesis
Erythema Annulare Centrifugum	Annular erythema with a trailing scale; may be a response to a tinea infection; perform full skin exam
Scabies	Erythematous papules to pustules with excoriations and possible burrows, flexor wrists, interdigital webspaces, umbilicus, genitals
Lichen Planus	Purple, polygonal, flat-topped papules ± lacy reticulated scale; flexor wrists, ankles, trunk ± oral lesions
Psoriasis Vulgaris	Elevated, well-demarcated, erythematous plaques with overlying silvery scale
Seborrheic Dermatitis	Waxy, yellow scale on an erythematous base; scalp, central face, eyebrows, beard, central chest
Pustular Psoriasis	Deep, yellow papules ± brown collarettes of scale on the plantar/palmar surfaces
Subacute Cutaneous Lupus Erythematosus	Annular, erythematous plaques with central clearing in a photodistribution (V-neck and upper back ± upper arms)
Pityriasis Rosea	Single patch (herald patch) followed by eruption of flesh to erythematous colored scaly patches in a "Christmas tree" distribution on the trunk
Source: Author created.	

Supplement to *Emergency Medicine Reports*, April 15, 2018: "Skin Infestations: Fungal and Scabies." Authors: Jessica Perkins, DO, PGY-3, Dermatology Resident, Nova Southeastern University College of Osteopathic Medicine/Largo Medical Center, Largo, FL; Molly Buckland, DO, PGY-1, Dermatology Resident, Nova Southeastern University College of Osteopathic Medicine/Largo Medical Center, Largo, FL; Richard A. Miller, DO, Program Director, Dermatology Residency Program, Nova Southeastern University College of Osteopathic Medicine/Largo Medical Center, Largo, FL.
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