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Diagnosis and Management of Tinea Infections

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Abstract

Tinea infections (often called ringworm) are caused by dermatophyte fungi and classified by the body site involved. Tinea corporis and tinea capitis are most common in prepubertal children, and tinea cruris, tinea pedis, and tinea unguium (most common type of onychomycosis) are more likely in adolescents and adults. Clinical diagnosis without testing may be unreliable because other conditions can resemble tinea infections (eg, tinea corporis can be confused with eczema, and onychomycosis with dystrophic toenails from repeated low-level trauma or psoriasis). Tinea corporis, tinea cruris, and tinea pedis generally respond to inexpensive topical antifungal agents, but oral antifungal agents may be indicated for patients with extensive disease, lack of response to topical treatment, immunocompromise, or hair follicle involvement (eg, tinea capitis). Oral terbinafine is considered first-line therapy for tinea capitis and onychomycosis because it is well tolerated, effective, and inexpensive. Emerging tinea infections may be more severe than classic tinea infections and generally do not improve with first-line topical or oral antifungals. These infections may require prolonged oral antifungal therapy and specialized diagnostic testing. Antifungal stewardship, including avoiding the use of combination antifungal-corticosteroids, should be emphasized to optimize outcomes and help prevent resistance.

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This article updates previous articles on this topic by Ely, et al.¹; Weinstein and Berman⁶²; and Noble, et al.⁶³

Data Sources: A PubMed search was completed using the MeSH term Tinea [majr] and included meta-analyses, guidelines, randomized controlled trials, and reviews. Essential Evidence Plus, the Cochrane Database of Systematic Reviews, and UpToDate were also searched. We performed multiple targeted searches in PubMed and reference lists of previously retrieved studies to fill in the remaining information gaps, such as the performance characteristics of laboratory tests used to diagnose fungal infections. Search dates: November 1 to December 1, 2024, and July 7, 2025.

The findings and conclusions in this report are those of the authors and do not necessarily represent the official position of the Centers for Disease Control and Prevention (CDC). Use of trade names and commercial sources is for identification only and does not imply endorsement by the CDC.

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Tinea infections (often called ringworm) are common superficial fungal infections caused by dermatophyte molds^{1,2} (Table 1¹). The word tinea is usually followed by a Latin term to indicate the body site involved, such as tinea corporis or tinea pedis. Onychomycosis is a general term for fungal nail infections, which are most often caused by dermatophytes (tinea unguium) but may be caused by yeasts or non-dermatophyte molds.^{3,4} The most common dermatophytes that cause human infections are from the genera *Trichophyton*, *Microsporum*, and *Epidermophyton*.^{3–7}

Tinea corporis and tinea capitis are the most common tinea infections in prepubertal children, whereas tinea cruris, tinea pedis, and tinea unguium are more likely in adolescents and adults.^{2,6,8} Correct diagnosis and treatment can be challenging because other conditions can mimic tinea infection.^{2,9} The differential diagnosis of tinea infections is outlined in Table 2.¹ Table 3 lists pitfalls in the management of tinea infections.¹ Table 4 summarizes common antifungals used for treatment.

Although considered mild conditions, tinea infections have received renewed attention due to the recent emergence of dermatophyte strains that cause more severe or difficult-to-treat infections than in the past.^{10–18} Antifungal stewardship, including avoiding the use of combination antifungal-corticosteroids, should be emphasized to optimize outcomes and help prevent resistance.¹⁹

TINEA CORPORIS, TINEA CRURIS, TINEA PEDIS

Clinical Presentation

Tinea corporis classically presents as a red, annular, scaly, pruritic plaque with central clearing and an active border (Figure 1¹). Lesions may be single or multiple and range in size from 1 to 5 cm. Larger lesions and lesion confluence can also occur.^{2,20} Tinea corporis may be mistaken for other skin conditions, particularly eczema, psoriasis, and seborrheic dermatitis.^{9,21} In a study of 148 patients presenting to primary care with erythematous squamous skin lesions, the findings that best ruled in dermatomycosis included maceration (positive likelihood ratio [LR+] = 1.7), overall clinical diagnosis (LR+ = 1.5), concentric rings (LR+ = 1.4), and central clearing (LR+ = 1.2).²²

Tinea cruris (also called jock itch) most often affects adolescent and young adult males.⁶ It usually involves the skin on the upper thigh opposite the scrotum²⁰ (Figure 2¹). The scrotum typically is spared, and scrotal involvement may help differentiate tinea cruris from candidiasis.²

Tinea pedis (also called athlete's foot) typically affects interdigital skin but can spread to the sole, sides, and dorsum of the foot² (Figure 3¹). Patients with acute tinea pedis typically have interdigital erythema and maceration, which may be accompanied by painful vesicles. This type of infection should be differentiated from gram-negative toe web infections, which can occur concomitantly with tinea pedis but may be associated with more pain, greater likelihood of extension to the distal sole, and in the case of the most common pathogen (*Pseudomonas*), a foul-smelling, greenish exudate.²³ Chronic tinea pedis, the more common form, usually involves interdigital scaling, peeling, and erythema, but other areas of the foot

may be affected. The term “moccasin pattern” describes tinea pedis that involves erythema and hyperkeratosis on the plantar and lateral aspects of the foot.^{2,20}

Diagnostic Testing

Tinea corporis, tinea cruris, and tinea pedis may be strongly suspected based on patient history and visual inspection.^{2,20} In-office microscopy is not commonly used for the diagnosis of tinea infections in primary care settings.²⁴ However, this testing can help differentiate tinea infection from other similar-appearing conditions. When feasible, in-office diagnostic testing (eg, potassium hydroxide [KOH] preparation with direct microscopy) can help establish a tinea infection diagnosis as demonstrated in the video at https://www.youtube.com/watch?v=LUwNQI_0BWU.¹⁸ Physicians may treat for tinea infection despite a negative result on KOH preparation if there is a strong clinical suspicion for fungal infection based on visual inspection. Figure 4 shows an example of KOH preparation.¹

If in-office microscopy is not an option, skin scrapings can be transferred to the laboratory for testing by placing them between two clean glass slides or in a sterile container. Cultures usually are not necessary for diagnosis but may be useful if there is concern for antifungal drug resistance or an unusual causative dermatophyte because isolates can be saved for testing at specialized laboratories.¹⁰ Rarely, a skin biopsy with periodic acid–Schiff stain may be used for the diagnosis of atypical or persistent lesions.²

Treatment

Tinea corporis, tinea cruris, and tinea pedis usually respond to topical creams, such as butenafine, ketoconazole, or terbinafine. Oral antifungal agents may be used for patients with extensive disease, lack of response to topical treatment, immunocompromise, or hair follicle involvement.² For tinea infections that are caused by certain emerging dermatophyte species, prolonged oral antifungal therapy may be needed.^{10,25,26}

When the cause of a rash is uncertain, physicians may choose combination antifungal-corticosteroid products in an attempt to cover fungal and nonfungal etiologies. However, this practice is generally discouraged because the corticosteroid component is often high-potency (clotrimazole-betamethasone) or medium-potency (nystatin-triamcinolone), which can result in skin atrophy or systemic adverse effects.^{27–30} The use of combination antifungal-corticosteroid creams may also contribute to the emergence and spread of antifungal-resistant dermatophyte strains.^{2,27–30}

When diagnostic testing or dermatology referral is impractical, a 2-week trial of topical antifungal therapy (without a corticosteroid component), with follow-up to document improvement, is a reasonable alternative.^{18,27} If a nonfungal lesion is treated with an antifungal cream, the lesion likely will not improve or will worsen. Conversely, worsening of a skin condition after empiric treatment with a topical corticosteroid suggests an alternative diagnosis, including a dermatophyte infection.²

TINEA CAPITIS

Clinical Presentation

In the United States, tinea capitis is most common among Black children aged 3 to 9 years.^{7,8,31} There are three main types of tinea capitis: gray patch, black dot, and favus. Black dot is most common in the United States² (Figure 5¹). The classic presentation is one or more scaly patches of alopecia with broken hairs at the skin line (black dots) and crusting; however, early disease may be limited to pruritus and scaling. Untreated tinea capitis may progress to kerion, a condition characterized by boggy, tender plaques and pustules.²

Diagnostic Testing

Tinea capitis is often treated empirically without diagnostic testing when the clinical presentation is typical.^{31,32} Seborrheic dermatitis and alopecia areata are the most common mimics of tinea capitis. In atypical cases, a KOH preparation can be used to look for fungal spores in scrapings of the black dots (broken hairs).²⁰

Culture is generally more sensitive (51% to 97%) than KOH preparation (59% to 73%) and is performed by moistening a cotton applicator or toothbrush with tap water and rubbing it over the scalp lesion.¹ The sample can then be applied to the Sabouraud liquid medium or dermatophyte test medium. Cultures have a high false-negative rate in children with kerion.²

Treatment

Systemic antifungals should be used to treat tinea capitis because topical agents do not penetrate hair follicles.^{2,31,33} Concomitant treatment with selenium sulfide 1% or 2.5% shampoo, ketoconazole 2% shampoo, or ciclopirox 1.5% shampoo for the first 2 weeks may reduce transmission.^{1,34,35} Although 6 to 8 weeks of griseofulvin therapy has been considered first-line treatment for tinea capitis, randomized clinical trials have shown that shorter treatment courses (4–6 weeks) of terbinafine are equally effective and safe.^{2,33} Terbinafine may be superior to griseofulvin for *Trichophyton* species (typically associated with black dot tinea), whereas griseofulvin may be superior to terbinafine for the less common *Microsporum* species (typically associated with gray patch tinea).²

Cultures can take 2 to 6 weeks to yield results, but terbinafine is a reasonable first choice in patients without recent travel outside of the United States because more than 89% of US tinea capitis cases are caused by *Trichophyton*.^{2,7} However, if cultures show that the pathogen is *Microsporum*, patients may require treatment with griseofulvin. *Microsporum* infections are more common outside of the United States, particularly in South America, Southern and Central Europe, Africa, and the Middle East.³³

Prompt treatment of kerion is critical to prevent scarring and permanent hair loss.^{2,20} In patients starting terbinafine, obtaining baseline liver function tests and blood cell counts is reasonable; however, further monitoring is generally unnecessary unless the patient has an underlying liver or hematologic condition or is 65 years or older.^{36,37} Terbinafine is usually well tolerated; the most common adverse effects include headache, gastrointestinal symptoms, and rash. Terbinafine-related liver damage and taste disturbances are rare.³⁸

ONYCHOMYCOSIS

Clinical Presentation

Onychomycosis is a common cause of dystrophic toenails in adolescents and adults.⁴ The distal, subungual form of onychomycosis is characterized by thickened, brittle, discolored nails (Figure 6¹). Onychomycosis may also present with an uncommon proximal, subungual form, which should raise suspicion for immunocompromise, and a white superficial form, which more often occurs in children^{2,4} (Figure 7¹). In a study of 169 patients with clinically suspected onychomycosis, the findings that best ruled in onychomycosis included tinea pedis in the past year (LR+ = 2.6), white crumbly areas on the nail surface (LR+ = 2.3), and dry, scaly skin on the soles or palms (LR+ = 1.7).³⁹

Onychomycosis may be confused with chronic trauma or psoriasis.⁴ Adolescents and young adults can develop dystrophic toenails from playing sports that involve repeated low-level trauma.² Exclusive involvement of the little toe suggests trauma rather than fungal infection.⁴

Diagnostic Testing

Diagnostic testing should be used to confirm suspected onychomycosis because the treatment course is long and nonfungal mimics are common.^{4,40,41} KOH preparation with staining and direct microscopy is inexpensive and can provide rapid results, but test reliability depends on the expertise of the physician. Fungal culture has poor sensitivity (59%) and carries a risk of contamination by nondermatophyte molds. However, it can provide information on the causative organism, which can help guide treatment.^{3,42}

Histopathology with periodic acid–Schiff stain is performed by placing toenail clippings or curette scrapings in 10% formalin solution and transporting them to the pathology laboratory. Use of polymerase chain reaction testing is increasing in the diagnosis of onychomycosis. Although it provides rapid results, access is limited.^{4,5,43}

Treatment

Treatment courses for onychomycosis are long (4–48 weeks), failure rates are high, and recurrence rates are 20% to 25%.^{20,38} Treatment is optional depending on patient preference, but many patients request treatment for cosmetic reasons or discomfort from shoes.⁴⁴ Oral antifungal therapy is generally more effective than topical therapy^{38,45,46}; however, some patients prefer to avoid systemic treatment. Ciclopirox nail lacquer or tavaborole may be offered with education about their low cure rates (29%–36% and 31%–36%, respectively).^{38,47–49}

For most patients, oral terbinafine, 250 mg once daily for 6 to 12 weeks, is preferred over oral itraconazole, other systemic azole antifungals, and griseofulvin because it is well tolerated, inexpensive, and potentially more effective. A large meta-analysis found moderate-quality evidence that azoles were less effective than terbinafine for achieving mycological cure (risk ratio = 0.77; 95% CI, 0.68–0.88).⁵⁰ Assessment of cure takes 9 to 12 months because toenails grow slowly. Oral itraconazole is a second-line agent for

onychomycosis and is typically used in patients who do not respond to terbinafine; it can be dosed daily or as pulse therapy.⁵¹ Efinaconazole (Jublia), a topical antifungal recently approved by the US Food and Drug Administration, is an effective option for distal and lateral subungual onychomycosis. It has a mycological cure rate of 61.6% but is expensive.⁵²

EMERGING DERMATOPHYTE INFECTIONS

In the past decade, several strains and species of dermatophytes have emerged that cause severe or difficult-to-treat tinea infections (Table 5^{10,14,15,17,25,53–57}). The American Academy of Dermatology has provided updated information on recognizing, diagnosing, and treating these infections.⁵⁸

Trichophyton indotineae

T indotineae (previously called *Trichophyton mentagrophytes* genotype VIII) is a novel pathogen that causes extensive, difficult-to-treat, often reoccurring infections in patients without immunocompromising conditions.^{10,12,25} *T indotineae* most often spreads person-to-person by direct skin contact or fomites, and rare cases of sexual transmission have been reported.⁵⁹ It is uncommon in the United States, but cases have been documented in multiple states, most frequently in patients with travel to or contact with someone who has traveled to South Asia.¹¹

Multiple morphologies have been reported, including erythematous, scaly concentric plaques (ie, ring-on-ring appearance), papulosquamous, pustular, and corticosteroid-modified tinea.^{10,25} Identifying *T indotineae* requires fungal culture and DNA sequencing available only at select laboratories. Based primarily on studies from India, the preferred first-line drug for *T indotineae* infection is itraconazole (up to 3 months) because this pathogen is frequently resistant to terbinafine.^{25,58} Images of *T indotineae* are available at https://www.cdc.gov/mmwr/volumes/72/wr/mm7219a4.htm#F1_down.

Trichophyton mentagrophytes Genotype VII

T mentagrophytes genotype VII is an emerging dermatophyte strain with a predilection for areas of sexual contact, including anogenital skin and the face (images available at https://www.cdc.gov/mmwr/volumes/73/wr/mm7343a5.htm#F1_down).⁵⁴ These infections have been reported in men who have sex with men in France since March 2021 and in the United States since 2024.^{13,14,54} Identification of *T mentagrophytes* genotype VII requires a fungal culture, DNA sequencing, and specialized testing that is available only at select laboratories, but clinicians can initiate empiric therapy based on epidemiologic and clinical features. Based on observational data, oral terbinafine is the recommended therapy, with treatment often required for up to 3 months.^{14,53}

Terbinafine-resistant *Trichophyton rubrum*

Worldwide and in the United States, cases of terbinafine-resistant *T rubrum* infections are increasing, including terbinafine-resistant onychomycosis.^{15,55–57,60,61} Few US laboratories can perform antifungal susceptibility testing for dermatophytes. The American Academy of

Dermatology provides a list of laboratories where testing for terbinafine-resistant *T rubrum* and other emerging dermatophyte species is available.⁵⁸

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SORT: KEY RECOMMENDATIONS FOR PRACTICE

Clinical recommendation	Evidence rating	Comments
When feasible, in-office diagnostic testing (eg, potassium hydroxide preparation with direct microscopy) should be used for the diagnosis of tinea infection. ¹⁸	C	Expert opinion in the absence of clinical trials
Combination antifungal-corticosteroid products, such as clotrimazole-betamethasone, generally should be avoided. ²⁷	C	Expert opinion in the absence of clinical trials
When diagnostic testing or dermatology referral is impractical for suspected tinea corporis, a 2-week trial of topical antifungal therapy (without a corticosteroid component), with follow-up to document improvement, should be considered. ^{18,27}	C	Expert opinion in the absence of clinical trials
Terbinafine is preferred for the treatment of tinea capitis, with shorter treatment courses than griseofulvin. ³³	A	Cochrane review of randomized controlled trials
Before initiating treatment, suspected onychomycosis should be confirmed with diagnostic testing, such as potassium hydroxide preparation, culture, periodic acid-Schiff stain, or polymerase chain reaction. ⁴¹	C	Expert opinion in the absence of clinical trials
Oral terbinafine is preferred for onychomycosis because it is well-tolerated, inexpensive, and more effective than other agents. ⁵⁰	A	Cochrane review of randomized controlled trials

A = consistent, good-quality patient-oriented evidence; B = inconsistent or limited-quality patient-oriented evidence; C = consensus, disease-oriented evidence, usual practice, expert opinion, or case series. For information about the SORT evidence rating system, go to <https://www.aafp.org/afpsort>.

WHAT'S NEW ON THIS TOPIC	
Tinea Infections	
A large meta-analysis found moderate-quality evidence that azoles were less effective than terbinafine for achieving mycological cure in onychomycosis (risk ratio = 0.77; 95% CI, 0.68–0.88).	
<i>Trichophyton mentagrophytes</i> genotype VII is an emerging dermatophyte strain with a predilection for areas of sexual contact, including anogenital skin and the face. These infections have been reported in men who have sex with men in France since March 2021 and in the United States since 2024.	
Worldwide and in the United States, terbinafine-resistant <i>Trichophyton rubrum</i> infections are increasing, including terbinafine-resistant onychomycosis.	

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FIGURE 1.

Tinea corporis.

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FIGURE 2.

Tinea cruris.

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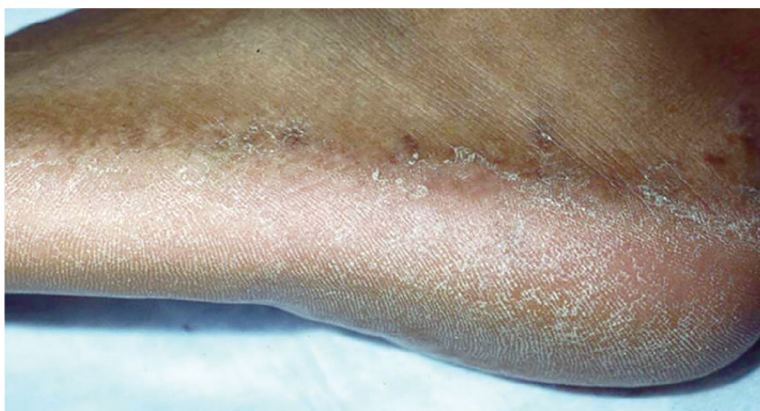


FIGURE 3.

Tinea pedis.

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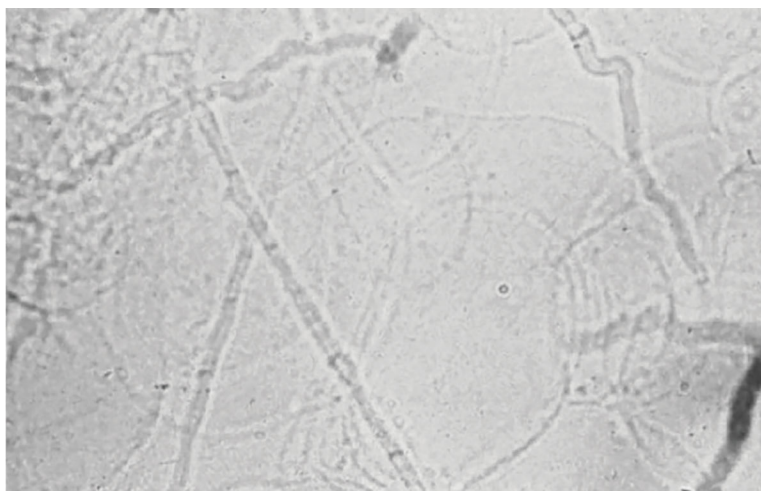


FIGURE 4.
Potassium hydroxide preparation of tinea infection.
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FIGURE 5.

Tinea capitis.

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FIGURE 6.

Onychomycosis.

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FIGURE 7.

White superficial onychomycosis.

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TABLE 1

Superficial Tinea Infections (Ringworm)

Infection	Location	Comments
Tinea capitis	Scalp	Figure 5
Tinea corporis	Arms, legs, trunk	Figure 1; also includes tinea gladiatorum and tinea faciei
Tinea cruris (jock itch)	Upper thighs	Figure 2
Tinea pedis (athlete's foot)	Interdigital skin; may spread to sole, sides, and dorsum of the foot	Figure 3
Tinea unguium (onychomycosis)	Toenails	Figure 6; onychomycosis is a general term for fungal infections of the nail; dermatophyte infections of the nail are called tinea unguium and are the most common type of onychomycosis; onychomycosis may also be caused by yeast or nondermatophyte molds

Adapted with permission from Ely JW, Rosenfeld S, Seabury Stone M. Diagnosis and management of tinea infections. *Am Fam Physician.* 2014;90(10):703.

TABLE 2

Differential Diagnosis of Tinea Infections

Diagnosis	Distinguishing features
Tinea corporis (red, annular, scaly, pruritic plaque with central clearing and an active border)	
Annular psoriasis	Gray or silver scale; nail pitting; 70% of affected children have a family history of psoriasis
Atopic dermatitis	Personal or family history of atopy; less likely to have active border with central clearing; lesions may be lichenified
Granuloma annulare	No scale, vesicles, or pustules; nonpruritic; smooth; commonly on dorsum of hands or feet
Nummular eczema	More confluent scale; less likely to have central clearing
Pityriasis rosea herald patch	Typically occurs in adolescents with a single lesion on neck, trunk, or proximal extremity; pruritus of herald patch is less common; progression to generalized rash in 1–3 weeks
Seborrheic dermatitis	Greasy scale on erythematous base with typical distribution involving nasolabial folds, hairline, eyebrows, postauricular folds, and chest; annular lesions are less common
Subacute cutaneous lupus erythematosus	Located on sun-exposed areas; multiple annular lesions; female to male ratio is 3:1
Tinea cruris (commonly occurs in adolescent and young adult males; spares scrotum and penis)	
Candidal intertrigo	Involves scrotum; satellite lesions; uniformly red without central clearing
Erythrasma	Red-brown lesions; no active border; coral red fluorescence on Wood lamp examination
Inverse psoriasis	Red and sharply demarcated lesions; may include other signs of psoriasis, such as nail pitting
Seborrheic dermatitis	Greasy scale on erythematous base with typical distribution involving nasolabial folds, hairline, eyebrows, postauricular folds, or chest; annular lesions less common
Tinea capitis (one or more patches of alopecia, scale, erythema, pustules, tenderness, or pruritus, with cervical and suboccipital lymphadenopathy; most common in Black children 3–9 years of age)	
Alopecia areata	Discrete patches of hair loss with no epidermal changes (ie, no scale); total loss of hair or fine miniature hair growth; exclamation point hairs; no crusting; no inflammation; possible nail pitting
Atopic dermatitis	Personal or family history of atopy; annular lesions, lymphadenopathy, and alopecia are less common
Bacterial scalp abscess	Alopecia less likely; hair pluck is painful
Psoriasis	Gray or silver scale; nail pitting; 70% of affected children have family history of psoriasis; involvement of other sites
Seborrheic dermatitis	Alopecia and lymphadenopathy uncommon; greasy scale; typical distribution involving nasolabial folds, hairline, eyebrows, postauricular folds, or chest
Trichotillomania	No scale; commonly involves eyelashes and eyebrows; hairs of varying lengths
Onychomycosis (discolored [ie, white, yellow, or brown], thickened nail with subungual keratinous debris and possible nail detachment; often in the great toe but can involve any nail)	
Other nail dystrophies; commonly associated with repeated low-grade trauma, psoriasis, or lichen planus	Appearance can be indistinguishable from onychomycosis; may have other manifestations of alternate diagnosis; exclusive involvement of the little toe suggests trauma rather than fungal infection

Adapted with permission from Ely JW, Rosenfeld S, Seabury Stone M. Diagnosis and management of tinea infections. *Am Fam Physician*. 2014;90(10):704.

TABLE 3

Pitfalls in the Management of Tinea Infections

Do not use oral ketoconazole for the treatment of tinea infection because of hepatotoxicity and the availability of safer agents.
Do not use griseofulvin for the treatment of onychomycosis because terbinafine is usually a better option based on its tolerability, high cure rate, and low cost.
Do not use combination products such as clotrimazole-betamethasone because they can result in skin atrophy or systemic adverse effects.
Do not use topical clotrimazole or miconazole to treat tinea infections because topical butenafine and terbinafine have better effectiveness and similar cost.
In general, do not treat tinea capitis or onychomycosis without first confirming the diagnosis with a potassium hydroxide preparation, culture, polymerase chain reaction testing, or periodic acid-Schiff stain. However, kerion should be treated aggressively while waiting for test results. It may be reasonable to treat a child with typical lesions of tinea capitis that involve pruritus, scale, alopecia, and cervical and suboccipital lymphadenopathy without confirmatory testing. If lymphadenopathy is absent, a confirmatory test is recommended.
Do not treat tinea capitis with topical agents alone, but it is reasonable to combine oral therapy with sporidical shampoos, such as selenium sulfide or ketoconazole.
Do not perform potassium hydroxide preparations or cultures for asymptomatic household members of children with tinea capitis, but consider empiric treatment with a sporidical shampoo.

Adapted with permission from Ely JW, Rosenfeld S, Seabury Stone M. Diagnosis and management of tinea infections. *Am Fam Physician.* 2014;90(10):705.

TABLE 4

Common Antifungals for the Treatment of Tinea Infections

Medication	Formulation	Uses*	Dosage and duration*	Cost†
Terbinafine	Oral	Label: onychomycosis of the toenail or fingernail caused by dermatophytes Off-label: dermatophyte folliculitis (eg, tinea barbae, Majocchi granuloma), tinea capitis, tinea corporis, tinea cruris, tinea manuum, tinea pedis	250 mg once daily 6–12 weeks for onychomycosis; 4–6 weeks for tinea capitis	\$20 for 30 tablets
	Topical	Label: tinea corporis, tinea cruris, tinea pedis Off-label: tinea manuum	1% once daily 1–2 weeks	\$15 for a 30-g tube at Walgreens
Itraconazole	Oral	Label: onychomycosis of the toenail, with or without fingernail involvement, and onychomycosis of the fingernail caused by dermatophytes (tinea unguium) in nonimmunocompromised patients Off-label: fungal folliculitis, tinea capitis, tinea corporis, tinea cruris, tinea manuum, tinea pedis	200 mg twice daily 6–12 weeks for onychomycosis; 2–12 weeks for tinea capitis; 1–2 weeks for tinea infections besides onychomycosis (some patients, such as those with <i>Trichophyton indotineae</i> infection, may require longer treatment)	\$40 for 30 capsules
Fluconazole	Oral	Off-label: onychomycosis, tinea capitis, tinea corporis, tinea cruris, tinea pedis	150–450 mg once weekly 6–12 weeks for onychomycosis; 2–6 weeks for other types of tinea infection	\$30 for 2 tablets (150 mg)
Clotrimazole	Topical	Label: tinea corporis, tinea cruris, tinea pedis Off-label: tinea manuum	1% twice daily 1–4 weeks	\$15 for a 30-g tube
Ketoconazole	Topical	Label: tinea corporis, tinea cruris, tinea pedis Off-label: tinea manuum	2% once daily 2–4 weeks	\$30 for a 60-g tube
Griseofulvin	Oral	Label: tinea barbae, tinea capitis, tinea corporis, tinea cruris, tinea pedis, tinea unguium Off-label: tinea manuum	500–1,000 mg once daily 6–12 weeks for tinea capitis	\$105 for 30 tablets
Butenafine	Topical	Label: tinea corporis, tinea cruris, tinea pedis Off-label: tinea manuum	1% once daily 1–4 weeks	\$10 for a 30-g tube
Efinaconazole (Jublia)	Topical	Label: mild to moderate onychomycosis of the toenail	10% once daily 48 weeks	\$800 for a 4-mL bottle of Jublia (not available as generic)
Ciclopirox	Topical	Label: tinea corporis (cream or suspension), tinea cruris, tinea pedis; topical treatment in immunocompetent patients with mild to moderate onychomycosis (lacquer) Off-label: tinea manuum	8% once daily (lacquer), 0.77% twice daily (cream) Up to 48 weeks for onychomycosis; 1–4 weeks for other tinea infections	\$20 for a 30-g tube of cream \$35 for a 30-mL bottle of suspension \$25 for 1 bottle of lacquer
Tavaborole	Topical	Label: onychomycosis of the toenails	5% once daily Up to 48 weeks	\$100 for a 10-mL bottle

* —Indication by US Food and Drug Administration label status. Information obtained from <https://UpToDate.com>. Accessed March 13, 2025.

† —Estimated lowest GoodRx price unless otherwise noted. Actual cost will vary with insurance and by region. Generic price listed unless otherwise noted. Information obtained at <https://www.goodrx.com> (accessed June 9, 2025; zip code: 66211).

TABLE 5

Summary of Emerging Dermatophytes

Dermatophyte	Epidemiology	Clinical presentation	Testing
<i>Trichophyton indotineae</i> (previously called <i>Trichophyton mentagrophytes</i> genotype VIII)	Mainly affects patients from South Asia; possible transmission reported in the United States ¹⁰	Large, recurrent tinea infection with variable appearance (eg, erythematous plaques, papulosquamous, pustular forms); often worsened by corticosteroids; previous courses of terbinafine have been ineffective ^{10,25}	Terbinafine is usually ineffective; itraconazole is first-line treatment ²⁵
<i>T mentagrophytes</i> genotype VII	Primarily spread through sexual contact in men who have sex with men in the United States and Europe ^{4,53} ; cases in Europe have been reported in people who traveled to Southeast Asia for sex tourism ¹⁷	Pruritic, annular, scaly lesions on the trunk, groin, genitals, or face; may lead to scarring or secondary bacterial infection ⁵⁴	Oral terbinafine is first-line treatment but may require extended courses; itraconazole has shown success in case reports ¹⁴
Terbinafine-resistant <i>Trichophyton rubrum</i>	Limited data, cases of tinea corporis reported in the southern United States ^{5,55} ; onychomycosis noted in the United States and Canada ^{56,57}	Infection resistant to standard doses of terbinafine; persistent lesions possible ⁵⁵	Itraconazole and posaconazole (Noxafil) have shown success in case reports ^{55,56}

Note: Clinicians who suspect *T mentagrophytes* genotype VII infection can contact their state and local health departments (<https://www.cdc.gov/public-health-gateway/php/index.html>) for assistance. They can request consultation for the evaluation and management of challenging cases of sexually transmitted infection from the National Network of STD Clinical Prevention Training Centers STD Clinical Consultation Network, which is supported by the Centers for Disease Control and Prevention's Division of STD Prevention (<https://www.stdccn.org/render/Public>). Detailed information on epidemiology, clinical features, treatment, and laboratories that perform testing for emerging dermatophyte infections is available from the American Academy of Dermatology Emerging Diseases Task Force at <https://www.aad.org/member/clinical-quality/clinical-care/emerging-diseases/dermatophytes/recognizing-trichophyton-indotineae>. Information from references 10, 14, 15, 17, 25, and 53–57.