Cutaneous Fungal Infections in Older Adults

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KEYWORDS

• Older adults • Dermatophytosis • Candidal cutaneous infection
• *Pityrosporum* infection

KEY POINTS

- The skin of older adults may be more prone to cutaneous fungal infections due to a history of long-term environmental exposures, as well as due to intrinsic metabolic and degenerative skin changes that occur over time.
- Dermatophytes, a type of mold, can invade and multiply within keratinized tissue to cause tinea pedis, tinea corporis, tinea unguium, and tinea capitis.
- *Candida albicans*, a yeast, can cause clinical infection of the skin, mucous membranes, and nails when the normal commensal balance of the skin is disturbed.
- Conditions such as seborrheic dermatitis, pityrosporum folliculitis, and tinea versicolor are caused by another yeast, *Pityrosporum ovale.*

INTRODUCTION

In 1900, the population of adults aged older than 65 years consisted only 4% of the population.[1] By 2050, that number is expected to increase to 22%.[2] The prevalence of dermatoses, specifically, cutaneous fungal infections, will increase accordingly. The skin of older adults is more susceptible to infection due to long-term exposure to ultraviolet light, smoking, and other environmental factors. Intrinsic degenerative and metabolic changes also occur, leading to thinning of the epidermis and damage with mild friction, creating a port of entry for microorganisms.[3]

Common cutaneous fungal infections can be divided into 3 broad causes.

1. The dermatophytes (a group of molds): tinea pedis, tinea corporis, tinea cruris, tinea unguium, and tinea capitis

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2. *Candida* (a yeast species): candidiasis, perleche, intertrigo, erosio interdigitalis blastomycetica, vaginitis, balanitis, and chronic paronychia
3. *Pityrosporum* (another yeast species): seborrheic dermatitis, tinea versicolor, and pityrosporum folliculitis

These pathogens cause superficial fungal infections, meaning they are limited to the stratum corneum, hair, and nails in immunocompetent patients.

DERMATOPHYTOSIS

Dermatophytosis refers to superficial skin, hair, and nail infections caused by dermatophytes. Dermatophytes can invade and multiply within keratinized tissue. There are 3 main genera: *Microsporum*, *Trichophyton*, and *Epidermophyton*. *Trichophyton rubrum* is the most common cause of dermatophytosis worldwide.[4]

Tinea Pedis

Tinea pedis, or athlete's foot, is a dermatophyte infection that localizes to the interdigital spaces and sides of the feet. *T rubrum*, *Trichophyton mentagrophytes*, and *Epidermophyton floccosum* are the most common pathogens involved. They flourish in warm, moist environments such as within occlusive footwear, pools, saunas, and gyms.[4] Older adults may dismiss dry, scaly skin as part of the normal aging process, and therefore, may experience a delay in diagnosis.

There are 3 clinical variants of tinea pedis: (1) moccasin, (2) interdigital, and (3) inflammatory (vesiculobullous).

The moccasin type presents as pruritic scaliness and mild erythema of the entire plantar surface and sides of the foot, creating a moccasin outline (**Fig. 1**). The most common causative agent is *T rubrum*. Patients may also present with unilateral hand involvement (tinea manuum), known as two feet–1 hand syndrome.[5]

The interdigital type, caused by *T rubrum*, is the most common presentation of tinea pedis. It begins with scaling, erythema, and erosion of the interdigital skin of the feet. Coinfection can lead to a malodorous and pruritic overlying bacterial infection.[6]

The inflammatory (vesiculobullous) variant is most commonly caused by *T mentagrophytes* and presents as vesicles or bullae on the foot.[7]

The differential diagnosis for tinea pedis includes contact dermatitis, dyshidrotic eczema, pustular psoriasis, and rarely, an autoimmune bullous dermatosis. Tinea manuum, tinea unguium, and tinea cruris are often seen in conjunction as a result of autoinoculation. Thus, the hands, nails, and groin should also be examined.

For diagnosis, a no.15 blade is used to scrape dry superficial scales onto a glass slide, which can then be examined under a microscope with 20% potassium

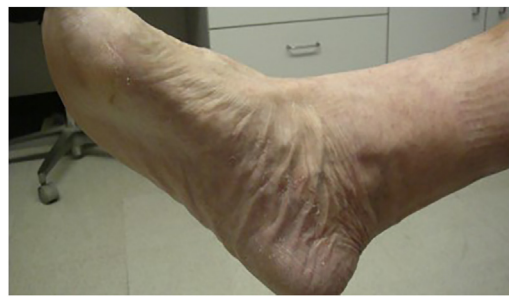

Fig. 1. Moccasin subtype of tinea pedis with scaliness on the plantar surface and sides of the foot.

hydroxide (KOH) or chlorazol black solution. Positive examination reveals long branching septate hyphae.

Treatment usually involves topical antifungals such as imidazoles (ie, ketoconazole, econazole) and allylamines (ie, terbinafine, butenafine) twice daily for 2 to 4 weeks.[3] Studies have shown no difference in efficacy between the 2 options.[8,9] If hyperkeratosis is present, keratolytics such as salicylic acid and lactic acid should be used to improve penetration of the antifungal agents. Oral therapy should be given to patients who have difficulty applying topicals to their feet. Pulse doses of 150 mg fluconazole once weekly for 2 to 6 weeks, 400 mg itraconazole daily for 7 days, and 250 mg terbinafine daily for 2 weeks have been shown to be effective.[10] Failure to treat the infection can lead to secondary bacterial infection such as cellulitis.[11,12] Patients should be taught preventative measures including wearing shower shoes in common bathrooms, completely drying feet after showering, and using a prophylactic antifungal powder or cream intermittently.

Tinea Corporis

Tinea corporis presents as a pruritic, scaly, annular, erythematous plaque on the trunk or extremities (**Fig. 2**). Spread through contact with contaminated shed skin cells, causative fungi thrive in warm and moist environments.[13] Thus, occlusive clothing that traps heat can predispose to infection.[14] The differential diagnosis is vast and includes nummular dermatitis, psoriasis, contact dermatitis, and pityriasis rosea.[5] Diagnosis is made through microscopic examination of scraped scale prepared with KOH.

Treatment is limited to topical antifungal agents unless the area of involvement is extensive or difficult to reach. Topical azoles and allylamines can be used for 2 to 4 weeks. The best oral option is terbinafine 250 mg daily for 1 to 2 weeks because it has the fewest interactions with other drugs.[15] Fluconazole (150–200 mg once weekly for 2–4 weeks) or itraconazole (200 mg daily for 7 days) can also be used for treatment.[16]

Tinea Cruris

Tinea cruris, or jock itch, presents as a red, scaly, annular plaque on the inguinal folds, perineum, and/or buttocks. It is often spread through clothes contaminated by other infected body parts while dressing. Sources of autoinoculation include tinea pedis, tinea manuum, and tinea unguium. Therefore, the hands, nails, and feet should be examined. Patients with these conditions should be encouraged to put on gloves or socks before dressing to prevent spread.[17] Notably, tinea cruris is more common in

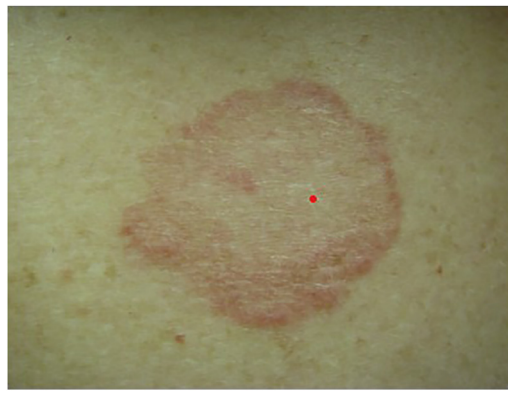

Fig. 2. Classic tinea corporis presents as an erythematous, scaly plaque with central clearing.

men than women because the external genital male anatomy predisposes to warmth and moisture. The differential diagnosis includes candida intertrigo, erythrasma, and inverse psoriasis. Tinea cruris can be distinguished from candidal infection because it spares the penis and scrotum. Treatment is similar to that of tinea corporis.[18]

Tinea Unguium

Tinea unguium is a type of onychomycosis in which there is a dermatophyte infection of the nail bed and plate.[19,20]

Generally, onychomycosis can be divided into 3 clinical types: (1) distal subungual, (2) proximal subungual, and (3) white superficial. The first 2 are most often caused by dermatophytes such as *T rubrum*, *T mentagrophytes*, *Trichophytonrichophyton tonsurans*, and *E floccosum*. The third, most commonly caused by *T mentagrophytes*, can also be caused by nondermatophyte molds such as *Aspergillus terreus*, *Fusarium oxysporum*, and *Acremonium potonii*.

Distal subungual onychomycosis is the most common form and leads to yellowing, thickening, subungual hyperkeratosis, and onycholysis of the toenails (**Fig. 3**). Secondary bacterial infection can occur, leading to additional green or black discoloration.[20]

Proximal subungual infection seems as a white nail with proximal subungual hyperkeratosis and onycholysis. This finding may be an indicator of an underlying immunosuppressed state.[20]

The nail in the white superficial type becomes crumbly and soft with a chalky white discoloration and rough dorsal surface.[20]

For diagnosis, a nail clipping can be submitted for fungal culture and/or microscopic examination. A separate clipping sample is submitted for each test. Submitting a nail clipping for histologic examination, when possible, tends to have a higher sensitivity (85%) compared with culture (32%).

There is a broad differential for onychodystrophy, including psoriasis, trauma, chronic eczematous dermatitis, and lichen planus.[4]

Treatment of onychomycosis is the same regardless of whether a dermatophyte or nondermatophyte is cultured. Terbinafine 250 mg daily for 12 weeks for toenails and the same dose for 6 weeks for fingernails is the treatment of choice. Terbinafine pulse therapy (500 mg daily for 1 week per month, 2 pulses for fingernails and 3 pulses for toenails), itraconazole pulse therapy (200 mg twice daily for 1 week per month, for 3 months), griseofulvin, and fluconazole can also be effective but have lower cure rates and more drug interactions.[21,22]

A yellow or white streak in the nail indicates a dermatophytoma, which is a walled off mass of fungus. Dermatophytomas should be removed surgically.[23] Superficial white

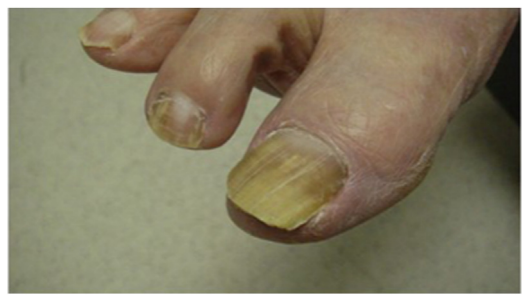

Fig. 3. Distal subungual onychomycosis characteristically presents with yellow, thick nails and onycholysis.

onychomycosis is localized to the dorsal nail surface and can be treated with topical azoles and ciclopirox 8% lacquer with occlusion.[24]

Due to the slow growth of toenails, resolution of onychomycosis can take 12 to 18 months, depending on how much of the nail is involved. Recurrence is common, with a 42% recurrence rate during a 1-year period, despite treatment with oral antifungals.[25]

Old shoes are a common source of reinfection. Therefore, patients should be advised to treat them with over-the-counter antifungal sprays or discard them altogether.

Tinea Capitis

Tinea capitis is a dermatophyte infection of the hair shaft, most commonly occurring in children. However, adults can also be affected, presenting with scalp scale and associated alopecia, pruritus, and/or posterior cervical lymphadenopathy. These symptoms may be mistaken for persistent dandruff or psoriasis, so precautionary testing in this setting is important.

Tinea capitis can be classified as endothrix or ectothrix depending on how the pathogen invades. In endothrix infections, it invades the hair shaft itself, often causing hairless patches with black dots (broken hair shafts). In ectothrix infections, the microorganisms cover the outside of the hair, resulting in scaly patches of alopecia known as gray patch tinea capitis (**Fig. 4**). The differential includes seborrheic dermatitis, eczematous dermatitis, or psoriasis. Severe inflammation can lead to kerion formation, which is a pustular eruption with scarring alopecia.

To prevent spread, anyone in close contact with a symptomatic patient should be treated with ketoconazole 2% shampoo. Fomites such as combs, hats, and hair accessories should also be disinfected or discarded. Some also advocate for the evaluation of home pets, who often serve as a persistent source of reinfection.

Diagnosis can be made with KOH examination, culture, or Wood lamp examination. *T tonsurans* is the most common cause of tinea capitis in the United States and does not fluoresce under a Wood lamp. However, the second most common cause, *Microsporum canis*, does fluoresce a characteristic yellow-green color.[4] Fungal cultures should be taken with a cotton swab swept over the scaly region. Broken hairs should also be included within the culture tube.

A dosage of 20 to 25 mg/kg daily of micronized griseofulvin for 6 to 8 weeks has historically been the gold standard treatment of tinea capitis caused by *Trichophyton* species.[5] However, multiple clinical trials have shown terbinafine to be just as effective with a shorter treatment duration (4 weeks).[26] Resolution can be achieved with either medication (**Table 1**).

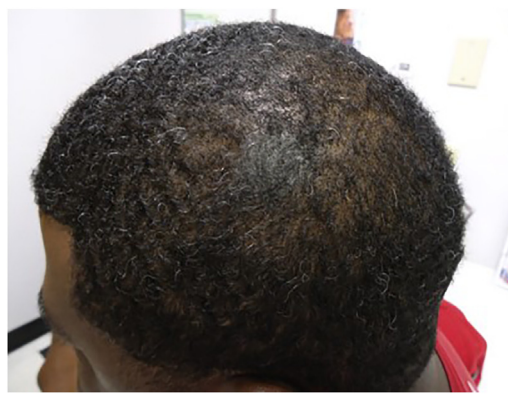

Fig. 4. Ectothrix tinea capitis classically causes scaly gray patches of alopecia.

Table 1
Treatment options for dermatophyte infections

Disease	Systemic Treatment	Amount
Tinea pedis	Fluconazole	150 mg/wk × 2–6 wk
	Itraconazole	400 mg/d × 7 d
	Terbinafine	250 md/d × 2 wk
Tinea corporis/cruris	Terbinafine	250 mg/d × 1–2 wk
	Fluconazole	150–200 mg/wk × 2–4 wk
	Itraconazole	200 mg/d × 7 d
Tinea unguium	Terbinafine	250 mg/d × 12 wk (toenails)
		250 mg/d × 12 wk (fingernails)
Tinea capitis	Griseofulvin	20–25 mg/kg/d × 6–8 wk
	Terbinafine	250 mg/d × 4 wk

KEY POINTS

- Dermatophytosis refers to superficial skin, hair, and nail infections caused by dermatophytes.
- These molds flourish in warm, moist environments and have the ability to invade keratinized tissue.
- Tinea pedis localizes to the feet and interdigital spaces. The infection is spread by walking barefoot on contaminated surfaces.
- Tinea corporis, or ringworm, presents as a pruritic, scaly, annular, erythematous plaque on the trunk or extremities.
- Tinea cruris, or jock itch, involves the inguinal folds, perineum, and/or buttocks. It presents as a red, scaly, annular plaque.
- Tinea unguium is a dermatophyte infection of the nail bed and plate. Resolution can often take 12 to 18 months.
- Tinea capitis is a dermatophyte infection of the hair shaft most commonly found in children. It can also be found in older adults but is often overlooked.

CANDIDAL CUTANEOUS INFECTIONS

Cutaneous candidiasis is most commonly caused by *Candida albicans*, a yeast that is part of the normal flora balance of the skin, mucous membranes, and nails of healthy humans. A disturbance of this balance can lead to infection of these sites, typically in either the very young or elderly. Note that chronic and recurrent episodes of candidiasis in older adults may indicate underlying conditions such as diabetes, malignancy, vitamin deficiency, or malnutrition. Appropriate workup and investigation should be performed.

Oral Candidiasis or Candidiasis (Thrush)

The oral cavity is the most common location for overgrowth of *Candida* species. Older adults are predisposed to this infection due to the frequent occurrence of dry mouth (xerostomia), along with the use of systemic, inhaled, or topical corticosteroids, broad-spectrum antibiotics, immunosuppressive drugs, and dentures.[27]

Gray-white plaques loosely adherent to the surface of the tongue, buccal mucosa, palate, and pharynx are clinically suspicious. These are easily removed with a tongue depressor to reveal a red, beefy, moist base.[28] Oral hairy leukoplakia and lichen planus

may seem similarly, however, the plaques will be fixed. Acute oral candidiasis can sometimes present with a glossy, red tongue with atrophic papillae.[27] Diagnosis can be confirmed by performing a fungal culture on a swab of the mucosal surface.

An important first step in treatment is to address any predisposing factors, such as denture cleanliness and potential medication causes. Pharmacologic treatment of patients with mild disease includes clotrimazole troches (1–2 tablets 4–5 times daily up to 14 days) or nystatin solution. Patients requiring systemic treatment can be given fluconazole (200 mg on day 1, then 100–200 mg daily continued for 7–14 days).[29]

Denture Stomatitis

Denture stomatitis presents as a localized, sharply demarcated, erythematous, and edematous lesion in denture-occluded areas. Candida infection is the most common cause for denture stomatitis. Predisposing factors include poor denture care and wearing dentures for longer than 24 hours.[30] Prevention involves removing dentures before sleeping, confirming a proper fit to prevent any trauma to the region, and cleaning them properly.[31]

Perleche (Angular Cheilitis)

Angular cheilitis, often associated with denture stomatitis, presents with maceration, fissuring, and crusting at the angles of the mouth. It is often caused by candida infection although infection with *Staphylococcus aureus* may also be causative. Patients with this condition are often edentulous and collect saliva in the affected area. Treatment involves a combination of topical anticandidal creams and low-to-mid potency topical corticosteroids (eg, 2.5% hydrocortisone). As a preventative measure, vaseline can be applied to the corners of the mouth at bedtime.

Candidal Intertrigo

Candidal intertrigo refers to inflammation (rash) in various intertriginous areas of the body. Patients present with sharply demarcated, pruritic to painful, erythematous, macerated patches, which are often studded with satellite pustules or a thin peripheral collarette of scale. Predisposing factors include free-hanging, overlapping skin, obesity, diabetes, chronic bed rest, inadequate personal hygiene, and use of broad-spectrum antibiotics.[28]

Diagnosis can be confirmed by a fungal swab culture. Erythrasma, a *Corynebacterium* bacterial infection, can present similarly but is distinguished by a characteristic coral red fluorescence on Wood lamp examination.

Treatment of candidal intertrigo involves the use of topical antifungals such as ketoconazole cream or nystatin powder twice a day for 2 to 4 weeks.[4] Low-potency topical steroids such as 2.5% hydrocortisone cream can be added to decrease inflammation but should not be used as monotherapy. Towels or washcloths moistened with a dilute vinegar solution can be used once or twice daily for 10 to 15 minutes to enhance medication penetration and provide additional antimicrobial effect. In severe or extensive cases, a course of oral antifungals at the same dosage as for oral candidiasis is appropriate. To keep the area dry and prevent recurrences, daily use of antifungal powders such as nystatin or miconazole after treatment of active infection should be encouraged.

Interdigital Candida (Erosio Interdigitalis Blastomycetica)

Another variant of intertrigo is interdigital Candida, also known as erosio interdigitalis blastomycetica. It presents as an area of macerated white skin on an erythematous, painful base between the middle and ring fingers of the hand and/or the fourth interspace of the feet. Predisposing factors include diabetes and repetitive immersion of

the hands in water. In the feet, this can be difficult to distinguish from tinea pedis without culture. Additionally, erythrasma should be considered and can be distinguished by Wood lamp examination. Treatment is similar to candidal intertrigo with the use of topical azoles and keeping the area clean and dry.[28]

Candidal Vulvovaginitis and Balanitis

The common term "yeast infection" refers to candidal vulvovaginitis, which occurs from an overgrowth of *Candida* in the vagina. Although easily treatable, older women may experience a delay in diagnosis due to less frequent gynecologic visits. Affected patients complain of severe pruritus, burning, and sometimes dyspareunia. Examination shows labial erythema and a white cottage cheese-like discharge.[32]

Predisposing factors include diabetes, use of broad-spectrum antibiotics, and long-term use of tamoxifen.[28] Tamoxifen has an antiestrogen effect on malignant breast cells but an estradiol agonist effect in the genital tract of postmenopausal women, facilitating *Candida* colonization.[33]

Uncircumcised older men who have intercourse with an affected woman are at risk for candidal balanitis, an inflammation of the glans. Symptoms include mild burning and pruritus. Examination shows glassy erythema, and occasionally small satellite pustules, on the inner aspect of the foreskin. Although the most common source of transmission is through intercourse with an infected partner, infectivity is measured at only 10%.[34]

Visualization of yeast in a smear of vaginal discharge treated with KOH, or a fungal culture, confirms the diagnosis in a female. In a male, the surface of a pustule can be prepared with KOH or cultured. The treatment of choice is a single dose of fluconazole 150 mg. Other options for women include the use of topical or suppository antifungals such as miconazole and clotrimazole.[35,36]

Candidal Paronychia

Paronychia is inflammation of the skin around the nail, known as the nail fold. Although *S aureus* is the most common cause of acute paronychia, *Candida albicans* is found in 40% to 90% of chronic (>6 weeks) or recurrent cases and is thought to represent secondary colonization.[37] Similar to interdigital candidiasis, candidal paronychia is more common in diabetics and patients who do wet work.

Paronychia presents as painful erythema and edema of the nail fold, leading to separation from the nail plate and eventual loss of the cuticle. Chronic inflammation can lead to nail changes such as onycholysis and yellow-green discoloration.

Addressing predisposing factors is essential in the treatment of candidal paronychia. Patients should be advised to minimize wet work and seek treatment of underlying diabetes if present. Pharmacologically speaking, topical steroids have been found to be more effective than systemic antifungals as have topical calcineurin inhibitors such as tacrolimus.[38–40] This may indicate that chronic paronychia is more an inflammatory disorder of the proximal nail than a fungal infection. Still, a topical antifungal (ciclopirox) could be considered as adjunctive therapy in refractory cases but may or may not provide additional efficacy.

KEY POINTS

- Cutaneous candidiasis is most commonly caused by *C albicans*, a yeast that is part of the normal flora balance of the skin, mucous membranes, and nails of healthy humans. When this balance is disturbed, infection of these sites can occur.

- Oral candidiasis seems as grayish-white plaques that are loosely adherent to the surface of the tongue, buccal mucosa, palate, and pharynx.
- Denture stomatitis presents as a localized, sharply demarcated, erythematous, edematous lesion in an area that is occluded by the dentures and is most often caused by candidal infection.
- Angular cheilitis can be associated with denture stomatitis and affects the oral commissures, presenting with maceration, fissuring, and crusting at the angles of the mouth. *Candida* is the most common causative agent.
- Candidal intertrigo refers to candidal infection in various intertriginous areas of the body.
- Candidal vulvovaginitis occurs when there is an increased growth of *Candida* in the vagina.
- Paronychia is inflammation of the nail fold that often occurs after repetitive wet work.

PITYROSPORUM INFECTIONS

The yeast *Pityrosporum ovale* (also known as *Malassezia furfur*), a lipophilic fungus, causes seborrheic dermatitis, pityrosporum folliculitis, and tinea (or pityriasis) versicolor. A normal skin commensal, disease manifests when its numbers increase past a certain threshold.

Seborrheic Dermatitis

Patients with seborrheic dermatitis often complain of a dry, scaly scalp (Fig. 5). Additionally, pink patches with greasy scale can occur in other areas rich in sebaceous glands, such as the eyebrows, external ear canals, paranasal folds, and forehead (Fig. 6).

Sebocyte turnover rate decreases with age, leading to an increased incidence of seborrheic dermatitis in older adults.[41] Diagnosis is made clinically based on the classic distribution and appearance of the lesions. Patients with scalp involvement can be treated with antifungal (ketoconazole 2% and ciclopirox 1%) and cytostatic (zinc pyrithione and selenium sulfide) shampoos.[42] If severe, addition of topical steroid solutions or gels can help with inflammation and pruritus. Mild topical steroids (hydrocortisone 2.5% cream) and topical antifungals (ketoconazole 2% cream) can be used for the involvement of other areas such as the ears, neck, face, and chest.[43] Topical steroids should be discontinued on resolution. However, continued intermittent use of topical antifungals can prevent recurrence.

Fig. 5. Seborrheic dermatitis can present with a dry scalp with white flakes.

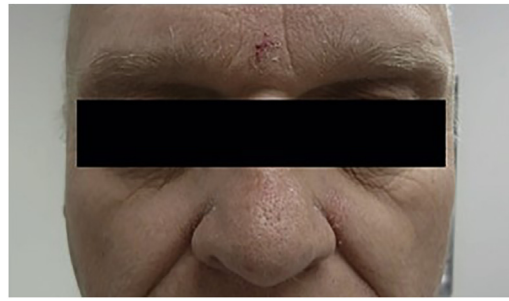

Fig. 6. Seborrheic dermatitis, seen as greasy scale on the eyebrows.

Pityrosporum Folliculitis

Pityrosporum folliculitis presents clinically with pruritic follicular papules and pustules, usually on the trunk. Although uncommon in older adults, it should be considered when patients complain of itching on the back. Predisposing factors include diabetes and use of corticosteroids or broad-spectrum antibiotics.[44] The diagnosis can be made clinically or by visualizing the *Pityrosporum* yeast on skin biopsy; it cannot be cultured with normal fungal culture medium. Response to therapy can also be used for diagnosis. Systemic treatment with itraconazole (200 mg daily for 1–3 weeks) or fluconazole (100–200 mg daily for 1–4 weeks or 300 mg weekly for 4–8 weeks) can be considered.[45] Topical therapy with selenium sulfide 2.5% or ketoconazole 2% shampoo can be used as adjunctive therapy. This condition is prone to relapse, so prophylactic treatment with selenium sulfide, econazole cream, or ketoconazole shampoo a few times weekly is beneficial even after treatment.[28]

Tinea Versicolor

Tinea, or pityriasis, versicolor is a common skin disorder that causes well-defined, slightly scaly, hypopigmented (most commonly) or hyperpigmented macules and patches (**Fig. 7**). Areas of change are more apparent in tanned individuals. The condition is more common in the summer months and in oily areas of the skin. The classic distribution involves the chest, back, neck, and face.

Diagnosis is confirmed by skin scraping and direct microscopy using KOH or chlorazol black. The classic "spaghetti and meatballs" appearance of the yeast with short hyphae and oval yeast forms confirms the diagnosis.

Tinea versicolor can be treated with selenium sulfide 2.5%, clotrimazole 1%, or ketoconazole 2% shampoo applied once daily for 2 to 4 weeks. Oral therapy with fluconazole 300 mg weekly for 2 weeks or itraconazole 200 mg daily for 5 to 7 days can be given, especially to older adults who may not be able to apply topicals.[46–48] The condition often recurs, therefore weekly use of an antiyeast shampoo is recommended long-term. Patients should be informed that this condition is neither contagious nor related to hygiene and that the color change will resolve within a few weeks to months. See **Table 2** for treatment options for canididal and pityrosporum infections.

KEY POINTS

- The yeast *P ovale* (also known as *M furfur*) causes seborrheic dermatitis, pityrosporum folliculitis, and tinea (or pityriasis) versicolor.

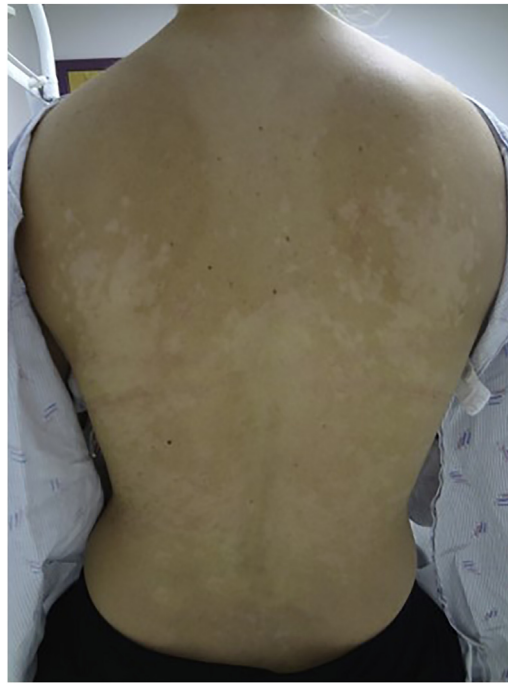

Fig. 7. Tinea versicolor presents as hypopigmented macules and patches on the back of this patient.

- Patients with seborrheic dermatitis have patches of greasy scale on the scalp, eyebrows, external ear canals, paranasal folds, and/or forehead. It can be treated with antifungal and cytostatic shampoos.
- Pityrosporum folliculitis presents clinically with pruritic follicular papules and pustules that usually involve the trunk. It can be treated systemically with the oral azole antifungals and topical antifungal shampoos.
- Tinea versicolor causes pigmentary changes in the skin that seem as hypopigmented or hyperpigmented patches in most individuals.

SPECIAL CONSIDERATIONS REGARDING SYSTEMIC ANTIFUNGAL TREATMENT

Systemic antifungal medications should be prescribed and used with caution due to interactions with other medications and potential side effects.

- All systemic azole antifungals can be hepatotoxic, so liver function tests should be checked before prescribing. Specifically, oral ketoconazole is discouraged from use because it can cause severe liver damage, adrenal gland issues, and harmful interactions with other medications.[49,50]
- Itraconazole can cause negative inotropic effects and should not be used in those with a history of congestive heart failure.[51]
- Itraconazole inhibits the cytochrome P3A4 enzyme, responsible for the metabolism of medications like H1 receptor antagonists, warfarin, and others, increasing the risk for medication-related side effects.[49,52]

Table 2
Treatment options for cutaneous candida and pityrosporum infections

Disease	Topical treatment	Systemic treatment
Oral candidiasis	Clotrimazole troches; 1–2 tablets 4–5/d × 14 d Nystatin solution; 4–6 mL 4/d × 7–14 d	Fluconazole 200 mg on day 1; 100–200 mg/d × 7–14 d
Candidal intertrigo	Ketoconazole cream or nystatin powder; twice daily × 2–4 wk	Fluconazole 200 mg on day 1; 100–200 mg/d × 7–14 d
Candidal vulvovaginitis and balanitis	Clotrimazole 1% cream; 5 g/d × 7–14 d Miconazole 2% cream; 5 g/d × 7 d	Fluconazole; 150 mg
Candidal paronychia	Medium-High potency topical steroid or topical calcineurin inhibitor ± topical ciclopirox solution	
Seborrheic dermatitis	Ketoconazole 2% cream and shampoo Zinc pyrithione or selenium sulfide shampoo Ciclopirox 1% shampoo	
Pityrosporum folliculitis	Selenium sulfide 2.5% or ketoconazole 2% shampoo (adjunctive therapy)	Itraconazole 200 mg/d × 1–3 wk Fluconazole; 100–200 mg/d × 1–4 wk or 300 mg/wk × 4–8 wk
Tinea versicolor	Selenium sulfide 2.5%, clotrimazole1%, or ketoconazole 2% shampoo once daily × 2–4 wk	Fluconazole; 300 mg/wk × 2 wk Itraconazole 200 mg/d × 5–7 d

- Fluconazole has fewer medication interactions because it is metabolized through the CYP 2C9 and 2C19 pathways, which are not commonly used for the metabolism of medications.
- Although griseofulvin is the mainstay therapy for tinea capitis, its liquid form has a poor compliance rate due to its bitter taste. Its side effect profile includes cytochrome P450 induction, photosensitivity, headache, and gastrointestinal upset.[28] Additionally, it must be taken with high-fat foods for optimal absorption.
- Terbinafine inhibits the CYP 2D6 system and can affect the metabolism of beta-blockers and tricyclic antidepressants.[5]

SUMMARY

As the patient population aged older than 65 years increases, it becomes more important to recognize and treat skin conditions seen in this age group. When determining treatment choice, it is important to consider different physiologic characteristics faced by older adults, their often lengthy medication lists (warfarin, metformin, hydrochlorothiazide, clopidogrel, and so forth), and their social living conditions. These patients should be prescribed simple regimens with a low risk of drug interactions. Treating skin conditions in older adults can lead to a significant increase in quality of life. Additionally, some fungal infections may become a port of entry for more serious infections, such as bacterial cellulitis, which can have significant morbidity and mortality in this patient population.

CLINICS CARE POINT

- Fungal infections tend to affect the hair, skin, and nails of older adults.
- Proper diagnosis requires an understanding of the clinical features unique to the various diseases discussed in this article. Once diagnosed, treatment of these infections is generally similar (topical ± oral antifungal medications).

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CONFLICT OF INTEREST OR DISCLOSURE

The authors have no conflict of interest to declare.

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